2007 AOA/ACOP




PEDIATRIC TRACK

OCTOBER 1 - 3, 2007 • SAN DIEGO CONVENTION CENTER

American College of Osteopathic Pediatricians
2209 Dickens Road • Richmond, VA 23230-2005 • (877) 231-ACOP
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AMERICAN COLLEGE OF OSTEOPATHIC PEDIATRICIANS

CARING FOR AMERICA'S CHILDREN

October 16, 2006

Dear Attendees:

2209 DICKENS ROAD, RICHMOND, VA 23230-2005

PHONE 877.231.ACOP • FAX 804.282.0090

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American College of Osteopathic Pediatricians

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NOTES

AMERICAN COLLEGE OF OSTEOPATHIC PEDIATRICIANS

Osteopathic Pledge of Commitment

As members of the osteopathic medical profession, in an effort to instill loyalty and strengthen the profession, we recall the tenets on which this profession is founded — the dynamic interaction of mind, body and spirit; the body's ability to heal itself; the primary role of the musculo-skeletal system; and preventive medicine as the key to maintain health. We recognize the work our predecessors have accomplished in building the profession, and we commit ourselves to continuing that work.

I pledge to:

Provide compassionate, quality care to my patients;

Partner with them to promote health;

Display integrity and professionalism throughout my career;

Advance the philosophy, practice and science of osteopathic medicine;

Continue life-long learning;

Support my profession with loyalty in action, word and deed; and

Live by each day as an example of what an osteopathic physician should be.

NOTES

2007 AOA/ACOP Pediatric Track

Faculty

Andrea Amalfitano, DO, PhD
Michigan State University
East Lansing, MI

Luis Felipe Amunategui, PhD
University Hospitals
Medical Center
Cleveland, OH

Michelle Bez, DO, FACOP
Phoenix Children's Hospital
Phoenix, AZ

Shawn K. Centers, DO, FACOP
Osteopathic Center for Children
San Diego, CA

Kevin Z. Craig, DO
Children's Hospital
Central California
Fresno, CA

Scott R. Elkin, DO
University of Texas at Arlington
Austin, TX

Michelle Fox, MS, CGC
UCLA Medical Center
Los Angeles, CA

Michael Kayser, DO
Warren Clinics &
St. Francis Hospital
Tulsa, OK

Mary Jean Sage, CMA-AC
The Sage Associates
Pismo Beach, CA

Daniel W. Saylak, DO
University of Texas Health
Science Center
Fort Worth, TX

Malcolm S. Schwartz, DO, FACOP
Monmouth Medical Center
Long Branch, NY

Don Self
Don Self & Associates, Inc
Whitehouse, TX

Paul Gregory Smith, DO
University Hospitals of Cleveland
Cleveland, OH

Ava C. Stanczak, DO, FAAP
Virginia College of
Osteopathic Medicine
Blacksburg, VA

Lizabeth H. Sumner, RN, BSN
The Elizabeth Hospice
Escondido, CA

Carey Walker, PhD
Mead Johnson Nutritionals
Evansville, IN

Faculty Disclosures

1 - No Relationshiip with Commercial Supporters
2 - Employment
3 - Management Position
4 - Independent Contractor (Including Contracted Research)
5 - Consulting
6 - Speaking and Teaching
7 - Membership on Advisory Committees or Review Panels
8 - Board Membership
9 - Other Activities
10 - Author did not provide disclosure information prior to printing. Disclosure will occur prior to the presentation.

Prior to the start of the meeting, all identifed conficts of interest will be resolved as per the ACOP CME Resolution of Confict of Interest (COI) Policy dated December 30, 2005.

Prior to the start of their presentations, speakers who did not provide disclosure information prior to the printing of this syllabus, will make a disclosure prior to their presentation.

Andrea Amalfitano, DO, PhD
6 – Genzyme Corporation
5 – Etubics Corporation
9 – Etubics Corporation (Stock Owner)

Luis Felipe Amunategui, PhD – 10

Michelle Bez, DO, FACOP – 10

Shawn K. Centers, DO, FACOP – 1

Kevin Z. Craig, DO – 10

Scott R. Elkin, DO
6 – Forest Pharmaceuticals
Michelle A. Fox, MS, BA, CGC
4 – Shire Human Genetics Therapies Center for Extramural Clinical Research and Education Program (CECRE)
5 – Shire Human Genetics Therapies Center for Extramural Clinical Research and Education Program (CECRE)

Michael Kayser, DO – 1

Mary Jean Sage, CMA-AC – 1

Daniel W. Saylak, DO, FACOFP – 1

Malcolm S. Schwartz, DO, FACOP
6 – Eli Lily, Genentech, Tercica

Don Self – 1

Paul Gregory Smith, DO – 1

Ava C. Stanczak, DO, FAAP – 1

Lizabeth Sumner, RN, BSN – 1

Carey Walker, PhD
2 – Mead Johnson Nutritionals

2007 AOA/ACOP Pediatric Track

Scientific Program

MONDAY, OCTOBER 1, 2007

6:30 am - 6:00 pm	Registration	*San Diego Convention Center - 4 Foyer*
7:00 am - 7:50 am	**ACOP LECTURE** ***Computers and PDAs in Pediatric Medical Practice*** Daniel W. Saylak, DO, FACOFP	*San Diego Convention Center - 4*
9:30 am - 10:00 am	Exhibit Break	
10:00 am - 11:00 am	**ACOP/AOA Practice Management Lecture Series** ***Audits*** Mary Jean Sage, CMA-AC	
11:00 am - 12:00 n	**ACOP/AOA Practice Management Lecture Series** ***Pediatric Billing*** Don Self	
12:00 n - 1:00 pm	**ACOP/AOA Practice Management Lecture Series** ***How to Optimize Income by Proper Documentation and Billing*** Mary Jean Sage, CMA-AC	
1:15 pm - 2:30 pm	Alumni Luncheons	
2:30 pm - 3:00 pm	Exhibit Break	
3:00 pm - 4:00 pm	**ACOP Lecture** ***Pediatric Obesity*** Malcom S. Schwartz, DO, FACOP	
4:00 pm - 5:00 pm	**ACOP LECTURE** ***Pediatric Diabetic Type II*** Malcom S. Schwartz, DO, FACOP	
5:15 pm - 5:45 pm	**Pediatric Program Directors Meeting**	
6:00 pm - 8:00 pm	**ACOP Reception**	*San Diego Convention Center - 14B*

TUESDAY, OCTOBER 2, 2007

6:30 am - 5:00 pm	Registration	*San Diego Convention Center - 4 Foyer*
7:00 am - 8:00 am	**ACOP LECTURE** ***Evaluation of Newborn with Dysmophic Features*** Andrea Amalftano, DO, PhD	*San Diego Convention Center - 4*
8:00 am - 9:00 am	**ACOP LECTURE** ***Ambulatory Care of NICU Graduates*** Michelle Bez, DO, FACOP	
9:00 am - 10:00 am	**ACOP LECTURE** ***Death in the NICU: Moral Distress in Health Care Providers*** Liz Sumner, RN, BSN	
10:00 am - 11:00 am	**ACOP LECTURE** ***Early Recognition of Spasticity in Infants*** Kevin Z. Craig, DO	
11:00 am - 12:00 n	**ACOP LECTURE** ***Pre and Probiotics: Are They Wise to Add to Infant Nutrition?*** Carey Walker, PhD	
12:00 n - 12:30 pm	Exhibit Break	
12:30 pm - 2:00 pm	General Membership Luncheon	*San Diego Convention Center - 14B*
2:00 pm - 3:00 pm	**ACOP LECTURE** ***New Expanded Newborn Metabolic Screen*** Michael Kayser, DO	*San Diego Convention Center - 4*
3:00 pm - 4:00 pm	**ACOP LECTURE** ***New Techniques for the Diagnosis of Genetic Diseases*** Michael Kayser, DO	
4:00 pm - 5:00 pm	**ACOP LECTURE** ***Current Concepts in Genetic Counseling*** Michelle A. Fox, MS, BA, CGC	
5:00 pm - 6:00 pm	**CME Committee Meeting**	
7:00 pm - 10:00 pm	**AOA/AAOA Presidential Reception**	

WEDNESDAY, OCTOBER 3, 2007

6:30 am - 5:00 pm	Registration	*San Diego Convention Center - 4 Foyer*
8:00 am - 9:00 am	**ACOP LECTURE** ***School Bullies*** Scott Elkin, DO	*San Diego Convention Center - 4*
9:00 am - 10:00 am	**ACOP LECTURE** ***Pain That Lasts Forever: Recognition, Diagnosis and Treatment of Post-traumatic Stress Disorder in Children and Adolescents*** Ava Stanczak, DO, FAAP	
10:00 am - 11:00 am	**ACOP LECTURE** ***What Happens in the Home after Tragedy in the Community?*** Felipe Amunategui, PhD	
11:00 am - 12:00 n	**ACOP LECTURE** ***Panel Discussion*** Ava C. Stanczak, DO, FAAP; Felipe Amunategui, PhD	
12:00 n - 1:00 pm	Lunch Break	
1:00 pm - 2:00 pm	**ACOP LECTURE** ***Pediatric OMT*** Shawn K. Centers, DO, FACOP	
2:00 pm - 3:00 pm	**ACOP LECTURE** ***Pediatric OMT Lab*** Shawn K. Centers, DO, FACOP	
3:00 pm - 4:00 pm	**ACOP LECTURE** ***Pediatric Palliative Care*** Paul G. Smith, DO	
4:00 pm - 5:00 pm	**ACOP LECTURE** ***Death and Dying Pediatric Issues*** Liz Sumner, RN, BSN	

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American College of Osteopathic Pediatricians

MONDAY, OCTOBER 1, 2007

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MONDAY, OCTOBER 1, 2007

7:00 am - 7:50 am

ACOP LECTURE
Computers and PDAs in Pediatric Medical Practice

Daniel W. Saylak, DO, FACOFP

After this presentation the participant will be able to:

1. Explore the expanding role of information technology is playing in medical offices
2. Identify the role of Personal Digital Assistants (PDA's) in pediatric resource management
3. Identify potential pitfalls in electronic medical record selections
4. Discuss new technologies that can make the incorporation of medical information technology more efficient

NOTES

Computers and PDA's in Pediatric Medical Practice

Daniel W. Saylak, D.O., FACOFP
College Station, Texas

American College of Osteopathic Pediatricians
October 1, 2007

Objectives:

- Explore the expanding role information technology is playing in pediatric offices
- Identify the role of Personal Digital Assistants (PDA's) in pediatric resource management
- Identify potential pitfalls in Electronic Medical Record selections
- Discuss new technologies that can make the incorporation of medical information technology more efficient.

All Electronic Health Records Are Not Created Equal

- Pediatrics presents some unique opportunities and challenges to programmers
- Extensive use of graphing and charts
- Drug dosing
- Immunization scheduling
- "A child is not a little adult"

Why Do I Need An Electronic Medical Record?

- Legibility
- Ease Of Record Retrieval
- Risk Management Issues For The Practicing Physician
- Safety
  - Computer Assisted Physician Ordering Entry (CPOE) Systems
  - Electronically Produced Prescriptions
- Drug Lists For Various Third-party Carriers
- Drug - Drug Interaction Prevention
- Patient Cost Data
- E-prescribing Is Frequently One Of The First Steps Taken By Many Physicians

Improved Efficiency

- Ease of record retrieval
- Rapid Response to Inquiries
  - Pharmacies
  - Consultants
  - Medico-Legal

When did patient safety become part of the electronic medical record initiative?

In 1991, the Institute of Medicine set forth the vision for an electronic patient record system in a report entitled "Computer - Based Patient Records: An Essential Technology for Healthcare". This landmark report began to define a vision for what an electronic patient record system could become. With the advancement in information technology over the subsequent 16 years, the scope of the electronic patient record has grown substantially. Further driving this vision was a patient safety initiative where error prevention rather than cost-containment became the focus for the initiative to replace paper-based recordkeeping systems. The Centers for Medicare and Medicaid Services (CMS) further adopted this through its support of the National Healthcare Information Initiative (NHII) promoted by the Bush administration.

How can a physician ever hope to recover the costs associated with the adopting electronic health record?

Because there is **a lack of universal definitions** associated with what should be included in electronic health record, there are numerous solutions available through software and hardware packages that make the selection of the appropriate vendor and electronic solution extremely difficult. An entire industry of consultants, a driven sales force, and a lack of commonality in the medical community has further driven physicians to a point of frustration as they examine how to incorporate this new technology into their current medical practices.

Many physicians feel that their office practices are "efficient enough" and see no real value other than preparation for the Pay for Performance model. However, it is the experience of many physicians who have adopted electronic health records that they have seen some tangible improvements in their practices as a result of the adoption. The most notable improvement appears to be in charge capture. **Numerous chart audits by external firms have shown that physicians frequently under code their charts and miss numerous billable procedures in their day-to-day office practice. This can sometimes reach 20% of lost charges.** By improvement in charge capture, the physician can expect to recover the initial cost of investment associated with the adoption of electronic health record.

Many electronic health record vendors now include prompting to make sure that the essential elements required for evaluation and management codes are included in the patient's health record. This improvement in documentation creates a legible and accurate depiction of each patient encounter supporting the billing for goods and services provided by the physician. This improvement in documentation also provides a mechanism to recover the cost of investment.

Other intangibles such as recovery of office space lost to storage of paper records are intangible benefits, but should also be considered in cost recovery analysis.

How can an individual physician make good decisions related to the adoption of electronic health record in their practice?

An EHR for Pediatrics

- **Make sure the EHR is designed for Pediatrics and not simply modified from a Family Practice Template**
- **Growth charts should be automated**
- **Immunization schedules should be updatable and automated**
- **Drug Dosing should be Pediatric specific**
- **Drug-Drug Interactions should Pediatric specific and supplied disinterested third parties (e.g. US Pharmacopeia)**

The Personal Digital Assistant (PDA's) and the Pediatrician

Selecting a PDA

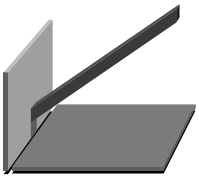
- Cost
- Expandability
- Color
- Internet Connectivity
- Compatibility with software
- Compatibility with hardware

Uses

- A Little Black Book Replacement
  - Appointments
  - Addresses
  - Telephone Numbers
  - To Do Lists (Tasks)
  - References
  - Diagnostic Tools

PDA's- A Growing Sector of Medical Computing

- Portability
- Utility
- Low Cost Software
- Wireless Utility (802.11b or g or n)
- Multiple freeware sites:
  - Tucows.com
  - ACOFP.org



Windows CE

- A familiar interface
- Sound
- Interaction with desktop computers
- Sophisticated interaction with desktop/server hardware and software
- Wireless interface

Palm Operating System

- Simple interface
- Designed for the computer illiterate
- Rapid initial adoption
  - Palm was top company in late 1990's
- Data entry
  - Keyboard (touch)
  - Graffiti ®
- Numerous Hardware options
  - Confusing?

What do you want to do?

For the Novice

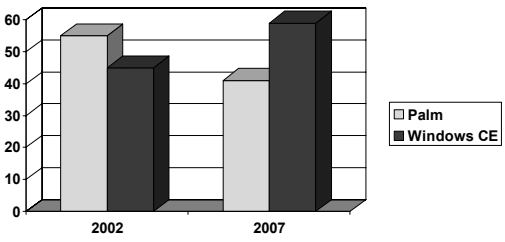
- Palm ® Operating System is the clear choice
- Ease of operation
- Vast software library
- Inexpensive
  - Starts at $99.00
- Options:
  - Palm Pilot
  - Handspring Visor
  - HandEra
  - Sony

Windows CE
For the Integrated User

- Many software manufacturers are now developing:
  - integrated medical office software
  - medical records templates
  - patient interaction documentation
- PDA functions as a data entry tool
- However…..

Operating Systems
Market Share

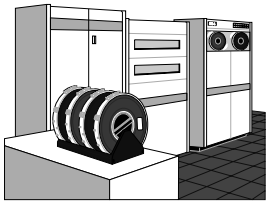
If you are going to use more sophisticated healthcare applications….



Source: Forrester Research, Cambridge, Mass.

Remember…

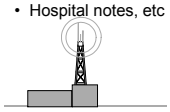
The PDA is not a replacement for the desktop



Are You A Real Geek?

The average doctor is now carrying:

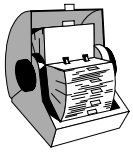
- Cellular telephone – "Smart Phone"
- Pager
- Notes
- Pieces of paper
- Hospital notes, etc



A Single PDA Can Serve All These Functions

- Blackberry ®
- LifeDrive ®
- PalmOne ®
  - More to Come

Increased Functionality = Increased Price



What are your colleagues using PDA's for?

- Reference
- Calculations
- Address book
- Appointments
- Stock Quotes
- E-Mail

Reference

- Huge Library of Titles
- Publishers are creating reference texts in both formats:
  - Skyscape
- Sites for Pediatric-specific Handheld software
  - Pediatrics On Hand
    - http://www.dcchildrens.com/pdas/home.aspx
  - DocMD.com
    - http://docmd.com/pdasoftware/
  - Yale Pediatrics
    - http://info.med.yale.edu/pediat/pedres/palmsoftware.html

Version 4.0

ePOCRATES

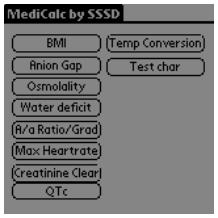
Version 1.0

- Infectious Disease Database
- Pharmaceutical Database
- http://www.epocrates.com/

Free!!!

Calculators

- Medicalc – http://www.angelfire.com/ms/medicalc/
  - Body Mass Index
  - Water Deficit
  - Creatinine Clearance
  - A-a gradients
  - QTc
  - Estimated Fetal age/weight



Handango

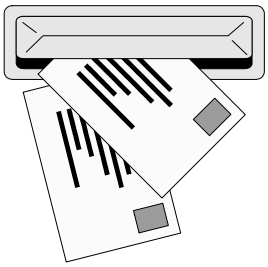
Stock Quotes

RIM Blackberry

- **"Push" technology**
- **Internet enabled stock/sale purchase**

E-Mail

- Windows CE
  - Outlook CE
- Palm OS
  - Date Book Plus + Mail Application
  - Sends during Synchronizing



Charge Capture Programs

The physician inputs what was done for the patient…..

and the software picks the proper codes

The result…

- Physicians report an average of 5% increase in income.
- Example practice showed an increase of $67,000 per doctor per year
  - Improved coding ($55,000)
  - Elimination of lost charges ($12,000)

- *Medical Economics, December 17, 2001*

Patient trackers improve on scraps of paper

- During hospital rounds, many doctors jot down notes on paper scraps that tend to get lost.

A PDA patient and procedure tracker can help you:

- Reduce paperwork
- Improve productivity
- Record and access patient information
- Room number
- Labs

- Example Programs:
  - ePatient2000
  - Grail
  - Hospital Census
  - Patient Database
  - Patient Log
  - PocketMD
  - TestTrakker

E-Prescribing

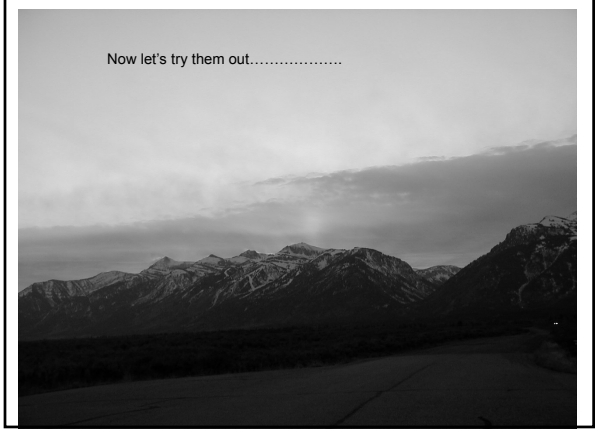
- Legible
- Accurate
- Transmittable
- Secure?

- Time Consuming
- May Be costly
  - iScribe (free)
  - PDA is dedicated to E-scribing
- HIPAA?

Beam Me Over…..

Applications can “beamed” between PDA’s with the same operating system

- Uses an infrared port
  - Caution: “slow”
- Needs to be shareware or freeware
- Check your licensing
- Most common method for printing
  - Printer must be infrared capable



NOTES

American College of Osteopathic Pediatricians

MONDAY, OCTOBER 1, 2007

10:00 am - 11:00 am

ACOP/AOA Practice Management Lecture Series
Audits

Mary Jean Sage, CMA-AC

After this presentation the participant will be able to:

1. Identify those things in the billing department that can realistically be audited
2. Determine who, what and when to audit
3. Select an appropriate party to perform an audit
4. Understand how to interpret the results of an audit

NOTES

American College of Osteopathic Pediatricians

MONDAY, OCTOBER 1, 2007

11:00 am - 12:00 n

ACOP/AOA Practice Management Lecture Series
Pediatric Billing

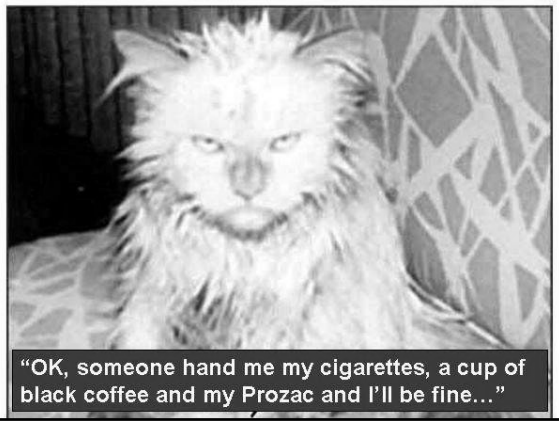
Don Self

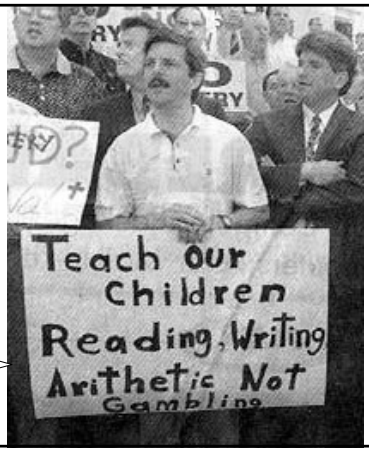
NOTES

Practice Management Lecture Series - Pediatric Billing – AOA/ACOP 2007

DON SELF & ASSOCIATES, INC

WWW.DONSELF.COM





Success of billing dependent upon getting proper billing information BEFORE the services are rendered

- What info do you collect when appt made?
- What do you expect from responsible parties?
- Who is responsible for the charges?
- Who is responsible for office policy adherence?
- Do you educate patients/families on policies?

WHAT ARE YOUR OFFICE/BILLING POLICIES?

What Can You Do?

- **Tighten up your patient collections policies.**
- **Do on line insurance verifications**
- **Collect all co-pay up front**
- **Send patients only 3 statements before putting them in collections**

What Can You Do?

- **Charge a "cost of re-billing" fee for each additional bill sent.**
- **Charge for no-shows/ cancelled appointments.**
- **Let the physician practice medicine – let the staff handle money – "anything you work out with my staff will be fine with me"**

Office Policies

- **Telephone calls**
- **Prescription refills**
- **Cancellations/No Shows**

Financial Policy for Patient Care Services

It is our policy to file for insurance as a courtesy to you if we have accurate and complete insurance information.

1. **The balance due is still your responsibility if we have not received payment from the insurance company within 30 days.**
2. **If you tell us that you or your child does not have insurance or Medicaid fraudulently in order to receive services, you now understand that this can be considered to be "attempted theft of services" by the courts, which is a criminal offense.**
3. **If you have insurance and we file with your carrier, we ask that you follow your policy responsibility according to your plan, ie., any deductible, co-pay , co-insurance amounts.**
4. **Since we are not a party to the agreement between you and your insurance company, we ask that you assist us in contacting them in the event that services are not paid within 30 days. If you fail to pay your co-pay, co-insurance or deductible and we have to turn your account to our collection agency, we are REQUIRED to notify your insurance carrier that you have not followed the terms of your insurance policy. This may very well result in your policy cancellation by your insurance carrier.**

5. If you do not have insurance and are not covered by either Medicare or Medicaid, you will be considered a "SELF PAY" patient. Payment is due in full at the time of service.

6. Patient "no shows" and cancellations are a tremendous loss for a practice. Please help our office reduce those losses by canceling within 24 hours if you cannot keep your appointment. <u>Failure to give notice 24 hours prior to your appointment will result in a $25 fee to be paid by the responsible party for visits. If a surgery is scheduled, the fee will be $100.00.</u>

To help in this policy we ask that you assist us by:

1. Providing us with current and updated information on yourself and your insurance company and to keep all changes up to date.
2. Make payment at the time of service for the entire balance if you are a "SELF PAY" patient, or for the amount of any deductibles or co-pays that may be due.
3. Discuss your account balance only with the check-out or business staff or contact the billing department of the hospital and /or physicians. Please do not discuss the financial aspects of your care with the physician(s). It is important for them to be allowed to practice medicine and provide patient care. Please work with the rest of the office staff on any account questions or problems you may have. If they cannot help you or answer your questions to your satisfaction, then please, do not hesitate to contact the office manager.

Patient Name (printed) _____

_____ _____ _____
Responsible Party Signature Date Staff Signature

CMS The Medicare, Medicaid, and SCHIP Agency

SOAP, 1995 & 1997 DOCUMENTATION RULES

HCFA Health Care Financing Administration

Subjective, Objective, Assessment, Plan

1995 & 1997

If it's not documented - it wasn't done !!!!

E & M DOCUMENTATION

NEW PATIENT

3 OF 3

EST PATIENT

2 OF 3

HISTORY
EXAM

MEDICAL DECISION MAKING

E & M DOCUMENTATION

ALL E&M SERVICES REQUIRE A CHIEF COMPLAINT

A VISIT WITHOUT A CHIEF COMPLAINT IS A PREVENTIVE MEDICINE PHYSICAL or SPORTS PHYSICAL

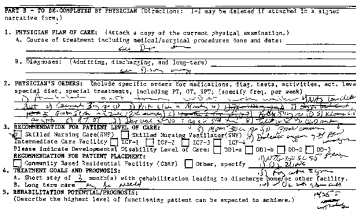
E & M DOCUMENTATION

ALL DOCUMENTATION MUST BE READABLE

Readable by the auditor

Give the doctor a reason to improve

2 Layperson Rule



99215	99214	99213	99212	99211	
5	4	3	2	1	Level
Y	Y	Y	Y	Y	cc
≥4 ≥3	≥4 ≥3	1-3	1-3	1-3	HPI
>10	2-9	1	–	–	ROS
2	1	–	–	–	PFSH
9 **18**	2-6 **12**	≥1 **≥6**	≥1 **1-5**	–	97 exam
≥8 **18**	2-7 **12**	2-7 **≥6**	≥1 **1-5**	–	95 exam
	extended system exam	*limited system exam*			
High	Moderate	Low	Minimal		MDM
40	25	15	10	5	Time

2 3 ESTABLISHED OUTPATIENT

E & M CODING - TIME FACTORS

- **COUNSELING (99201 - 99233)**
- **PROLONGED SERVICE (99354 - 99357)**
- **HOSPITAL DISCHARGE (99238 & 99239)**

COUNSELING & COORDINATION OF CARE - TIME FACTORS

99211	5 Minutes
99212	10 Minutes
99213	15 Minutes
99214	25 Minutes
99215	40 Minutes

Total Minutes

+

"_____ Minutes SPENT COUNSELING ABOUT _____"

The physician may document time spent with the patient in conjunction with the medical decision-making involved and a description of the coordination of care or counseling provided. Documentation must be in sufficient detail to support the claim.

PROLONGED SERVICE - TIME FACTORS

99211	5 Minutes	99354 30 - 74 min - Out-pt
99212	10 Minutes	99355 Ea. Addt'l 30 min. Out-pt
99213	15 Minutes	99356 30 - 74 min - In-pt
99214	25 Minutes	99357 Ea. Addt'l 30 min. In-pt
99215	40 Minutes	

PROLONGED SERVICE IS ABOVE & BEYOND NORMAL TIMES - WITH MEDICAL NECESSITY

HOSPITAL DISCHARGE - TIME FACTORS

- **99238 Hospital Discharge Day Management; 30 minutes or less**
- **99239 Hospital Discharge Day Management; More than 30 minutes.**

Included in Hospital Discharge: Final examination(s), Discussion of hospital stay with patient and/or family, instructions for continuing care to all relevant caregivers, preparation of discharge record, prescriptions, home health, DME, and referrals forms,

CMS now requires a face-to-face encounter on the day of discharge.

REMOVAL IMPACTED CERUMEN

A simple ear wash or irrigation is included in the E&M

69210 = "Impacted", Curette & Otoscope

CONSULTATIONS - INITIAL 99241 - 99255

- 99241 - 99245 - Out-patient (new or estab)
- 99251 - 99255 - In-patient (new or estab)
- Pre-op Clearance

99203 $93.00 **99213 $43.00**

99243 $133.00

Opinion being sought by another health care professional

Requesting Provider Must Document Request for Opinion

THANK YOU!

PLEASE VISIT OUR WEBSITE AT WWW.DONSELF.COM

OR

SEND US AN EMAIL AT DONSELF@DONSELF.COM

AND TELL US HOW WE DID.

American College of Osteopathic Pediatricians

MONDAY, OCTOBER 1, 2007

12:00 n - 1:00 pm

ACOP/AOA Practice Management Lecture Series
How to Optimize Income by Proper Documentation and Billing

Mary Jean Sage, CMA-AC

After this presentation the participant will be able to:

1. Understand and appreciate the relationship of documentation, coding and billing
2. Determine how to ensure everything done in the practice is being billed
3. Set benchmark goals for billing

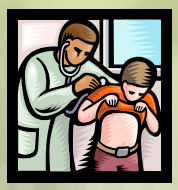
NOTES

How to Optimize Income

By Proper Documentation and Billing

A Seminar for:

American College of Osteopathic Pediatricians



October 2007

San Diego, CA

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About This Manual

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Today's Objectives

- Understand and appreciate the relationship of documentation, coding and billing
  - Everyone on the office has a role
- Determine how to ensure everything done in the practice is billed
- Gauge how the billing department (or agency / service) is performing
- Set benchmark goals for billing

5

REIMBURSEMENT MANAGEMENT

From Appointment Scheduling

↓
↓
↓

To Account Closure

6

The Reimbursement Team

Scheduler
↓
Receptionist
↓
Clinical Staff
↓

7

Reimbursement Team:

Physicians (and Extenders)
↓
Cashier
↓
Insurance Biller
↓
Collector

8

Scheduler

- Do we contract with this patient's insurance?
- Is Pre-authorization required?
- Is the patient eligible for coverage?
- Have you provided financial responsibility information?
- Will you mail a new patient information package?

9

Receptionist

- Verify Patient Information
  - As Needed
  - At Least Annually
- Checkpoint for Other Information
- Collect Co-payment

10

Clinical Staff

- Which ancillary services can you provide?
- Where to patients go for those you do not or can not provide?
- Have ancillary services been recorded?

11

Physician

- Do you code your own services and code correctly?
- Do you know significance of diagnosis and medical necessity?
- Do you know which services might be bundled?

12

Cashier

- Collected the right co-pay or co-insurance?
- Collected for non-covered services?
- Checked the superbill/encounter form for completeness and accuracy?

13

Insurance Biller

- Does this individual know:
  - ALL the plans with which you contract?
  - What is considered a "clean claim"?
  - How to appeal for add'l payment of denied or underpaid claims?

14

Insurance Biller . . . . . .

Do you mail or transmit insurance claims daily or weekly?

How do you handle the day-to-day correspondence from the insurance plans?

15

Collector

16

Collector . . . . . .

- Compliance with the rules and regulations of each contracted plan
  - Know how to read the EOB or RA from each plan
- Expected payment from each contract
- Determine what is billable to patient or another insurance vs. written off

17

Reimbursement Management

- Key Financial Indicators
  - Aging – benchmark percentages in each category – 30, 60, 90, 120 days
  - Days (or months) in accounts receivable
  - Collection ratio - both gross and adjusted

18

Billing Policies & Procedures

- Put them in writing
  - Reduce to paper the steps required to get a claim out the door and paid
  - If you purchase "model" policies, make sure you customize them to YOUR practice
- Annually (more often if needed or there is a change) review and update policies
- Establish Benchmarks for Billing
  - Timely Claim Submission
  - Correspondence Turnaround
  - Account Closure

19

Speed Up Your Payments

20

Speedy Payments Depend On:

- Correct Patient Demographics
  - Name (as on ID card)
  - Date of Birth
  - Health Insurance ID Number
- CMS 1500 or electronic format submitted correctly
  - By Payer
    - Use Your NPI correctly

21

Speedy Payments . . . . . .

- Prompt Claim Submission
  - 1-2 days for office services
  - 7-14 days for out of office
- Immediate reconciliation of claims rejection reports
- Use of correct claim submission addresses
- Prompt Correspondence Turnaround
  - 1-3 days from receipt

22

Compliant Claim Submission

By Payer

23

Assuring Compliant Claim Submission

- Know the requirements of **EACH** payer
- Adhere to Billing Time Limits
- Fill out claim correctly
  - Field 1a (ID)
  - Fields 9 and 9D – other insurance
  - Field 10d
  - Field 11 – insurance info (a-d)

24


- Fields 12 and 13
  - Medicare no longer requires assignment of benefits signature
  - Other payers require it is updated annually
- Fields 17 and 17a
- Field 23
- Fields 24
- Field 31
- Field 32
- Field 33

25

Optimizing Income

Spot
Claims Trends

And

Identify Denials

26

Coding Vs. Reimbursement

Who Rules?

27

Reporting Rules

CPT – developed rules for reporting procedures using codes. CPT is written and maintained by the AMA with significant input from medical specialty societies

28

Payment Rules

- Payers
  - Write their own reimbursement rules
  - Determine what codes will be "bundled"
  - Determine what codes are paid separately, if they pay them at all
  - Determine discount rules

29

Payer Rules

- Payers remain autonomous in their payment rules
  - Indemnity
  - Managed Care
  - Government
  - IPA/Medical Groups

You MUST Know Them All ! ! !

30

COLLECTIONS

Do You Have . . . . .

1. Clear Financial Policy
2. Minimum Balance Write-Off
3. Payment Schedule
4. Set Collection Procedure
5. Prompt Follow-Up Policy
6. Effective Outside Collector

32

Do You Have . . . . .

- Procedure for Updating Office Fee Schedule Annually
- Procedure for Updating Office Charge Ticket Annually
- Procedure for Performing Insurance Fee Schedule Analysis

33

Are You Prepared To

- Collect Higher Co-Payments
- Collect Towards Higher Deductibles
- Report Data for Performance

34

? ? YOUR QUESTIONS ? ?

1.
2.
3.
4.
5.

35

American College of Osteopathic Pediatricians

MONDAY, OCTOBER 1, 2007

3:00 pm - 4:00 pm

ACOP Lecture
Pediatric Obesity

Malcom S. Schwartz, DO, FACOP

After this presentation the participant will be able to:

1. Provide an understanding of the relationship of the anatomic and metabolic controls effecting obesity in children
2. Present a plan for the management of obesity using a multiple disciplinary approach
3. Discuss the use of a diet and exercise model for management of patients with insulin resistance and obesity
4. Present a database for the monitoring of criteria associated with diagnosis of insulin resistance disorders (Metabolic Syndrome)

NOTES

Pediatric Obesity: A Growing Problem

Malcolm S. Schwartz, DO, FACOP
Chief, Pediatric Endocrinology and Diabetes
Children's Hospital at Monmouth Medical Center

Childhood obesity is the most prevalent nutritional disorder in the United States. Not only is it increasing in incidence in this country, but it is becoming a major health problem worldwide. Obesity is defined in adults as individuals who have a Body Mass Index (BMI= kg/m^2) > 30. Children who have a BMI >2 standard deviations above the mean for height and gender (NIH graph for BMI) are considered obese. Those who have a BMI in the 90th to 95th percentile (90% to 95%) are overweight. And those who have a BMI between the 85th and 90th percentile should be watched closely for the development of obesity.

1. 15.5% of children 12-19 years old have a BMI > 95 %
2. 15.3% of children 6-11 years old have a BMI > 95 %
3. 10.4% of children 2-5 years old have a BMI > 95 %
4. By 1998 the prevalence of BMI > 85 % reached
   - 35% in Hispanic and African American children
   - >20 % in Caucasian children
5. The economic burden of childhood obesity has increased 3-fold in the past 20 years, reaching $127 million per year
6. 10 to 15% of all children in the United States 6-17 tears of age are obese

Across the world, the rate of children and adolescents who are overweight and obesity is increasing by alarming numbers. The complications of obesity, which had previously been observed in adults, are now being seen with common frequency in children. These include Type 2 Diabetes Mellitus, hypertension, hyperlipidemia, gallbladder disease, steatohepatitis (fatty liver), sleep apnea, and orthopedic complications.

Obesity: The Genetic Component

Many genes are known to have an effect on body weight. The basic premise is that the body seeks to maintain a steady weight. The hypothalamus and surrounding structures produce chemicals and hormones that stimulate appetite and decrease energy expenditure as well as produce different chemicals and hormones that decrease appetite and increase energy expenditure. The stomach, small and large intestines also play similar roles in increasing and decreasing appetite. As of now, these genes and their products control us. We do not control them.

However, we have done major research on and have some affect on insulin. Approximately 30,000 years ago when we were hunter-gatherers and did not know when the next meal was expected, some people were able to develop "thrifty genes." These genes allowed the body to become insulin-resistant by interfering with cell mechanisms

that allow blood glucose to be transported into the cells so that the cells could utilize this compound for energy. Therefore the pancreas had to make more insulin to accomplish this action. This action, which produced excessive insulin, allowed the fat cells to accumulate storage of fats for energy at a time of hunger and famine. This provided a much higher survival rate. Then approximately 5000 years ago we became an agrarian society and these thrifty genes helped us gain weight because food became plentiful and we ate multiple times during the day. As time passed we also did less physical activity. The combination of multiple daily meals, larger portions, less physical work and the thrifty genes has increased our rate of obesity and its complications.

Obesity Complications

Insulin excess due to insulin resistance is responsible for skin changes that increase the roughness of the skin and change its color to dark brown or black (Acanthosis Nigricans). Skin tags are also a direct result of excess insulin. Fatty liver is now seen in children. Atherosclerosis and hypertension begin in childhood and are a direct result of the obesity and hyperinsulinemia. In females Polycystic Ovarian Syndrome is related to the insulin resistance and may present with obesity, infertility and hyperandrogenism (development of secondary sexual characteristics e.g. facial, or abdominal hair, acne). It is present in 5% to 10% of American women. Diabetes mellitus Type 2 is a major complication of obesity and insulin resistance. Cholelithiasis is more common in obese adults. Although gallstones are unusual in childhood, nearly one half of all cases of cholecystitis in adolescents are associated with obesity. Anecdotal evidence suggests that depression and eating disorders are common in children and adolescents referred to obesity clinics. Prejudice and discrimination against individuals with obesity are ubiquitous within US culture; even young children have been found to regard their peers who have obesity in negative ways. Social isolation, peer problems, and lower self-esteem frequently are observed.

Treating Obesity

The increasing prevalence of obesity in childhood and adolescence, accompanied by insulin resistance, appears to explain the increasing incidence of type 2 diabetes in adolescents, particularly in minority populations. Treatment of obesity, hyperinsulinism and insulin resistance is not easy. It necessitates a multidisciplinary approach which includes the physician, nurse educator, dietician, exercise physiologist or personal trainer, psychosocial worker and psychologist. The main points of our therapy at the Children's Hospital at Monmouth Medical Center consist of changing the diet and emphasizing exercise.

Our dietician stresses a lower carbohydrate diet (40% of total daily calories) spread evenly over the entire day. Protein and fat may have to be increased slightly but we have not found an adverse affect on serum lipids. Complex carbohydrates and fruits and vegetables are emphasized in our clinic. Patients are taught carbohydrate counting (grams of carbohydrate) so that they make intelligent choices and substitutions. Foods with monounsaturated fats are stressed.

We believe that a program of intense exercise is necessary for weight loss. Insulin

sensitivity can be improved by exercise. Insulin resistance in the muscle liver and fat cells is lowered by exercise. Our exercise trainers handle the majority of our patients. They develop a series of exercises that include aerobic and weight resistance components. The patients can do the program at home or at a gym near their home if they live too far a distance from our trainers. Children with special needs are referred to a physical rehabilitation centers and a similar program is also prepared for them taking into account their limitations. The children are encouraged to select a program that is fun to do.

There are no FDA approved medications for children to lose weight. However metformin is approved for children 10 and older with Diabetes Mellitus Type 2. It is a biguanide insulin sensitizer. Its actions include: Increased peripheral sensitivity to insulin, decreases hepatic glucose output, weight loss, and improved plasma lipid profile. It has side effects which include: Anorexia, nausea, and diarrhea are most common.

Malabsorption of vitamin B12, anemia is uncommon. Lactic acidosis (has been reported with phenformin (a similar molecule) in renal/hepatic/cardiac dysfunction). It is very rare with metformin occurring only 1 in 50,000 cases. We use metformin in patients for weight loss who are severely insulin resistant with or without glucose intolerance. Our results demonstrate beneficial results.

Sibutramine inhibits norepinephrine, serotonin and dopamine reuptake. The patient recognizes that he/she has eaten. It is not approved for children under 16, but it has been used in patients who are binge eaters. The medication has caused hypertension and therefore it must be monitored closely.

Obesity Prevention

Prevention of obesity is always preferable to treatment. However it may be just as difficult to prevent obesity because our society promotes the economic and social benefits of advertising sales of food which are obesity enhancers.

The following are suggestions to prevent obesity:

1. During pregnancy, normalize the BMI of the mother
2. Prevent smoking
3. Maintain moderate exercise as tolerated
4. Pursue meticulous glucose control in gestational diabetes
5. During the postpartum and infancy period, encourage breast-feeding and postpone the introduction of solid foods
6. Families should eat meals as a family in a fixed place and time. Do not skip meals, especially breakfast. No television should be watched during meals and television should be limited to two hrs per day.

In Schools:

- Eliminate fundraisers with candy and cookie sales.
- Review contents of vending machines for healthier choices.
- Install water fountains.
- Educate teachers, especially physical education and science faculty, about basic nutrition and benefits of physical activity.
- Educate children on appropriate diet and lifestyle.
- Mandate minimum standards for physical education, including 30–45 min of strenuous exercise two to three times weekly.
- Encourage walking to and from school when it is safe.

In Communities:

- Increase family-friendly exercise/play facilities for all age children.
- Discourage the use of elevators and moving walkways.

If there was an infectious disease that affected as many patients as obesity does, our population would rise up and demonstrate to provide funds and opportunities to decrease it incidence. We need to work together to prevent this ongoing epidemic of obesity and decrease it serious complications.

MONDAY, OCTOBER 1, 2007

4:00 pm - 5:00 pm

ACOP LECTURE
Pediatric Diabetic Type II

Malcom S. Schwartz, DO, FACOP

NOTES

Diabetes Mellitus Type 2

Malcolm S. Schwartz, DO, FACOP

Diabetes Mellitus Type 2 is increasing in incidence in the United States. Because the epidemic of obesity and inactivity in children, type 2 diabetes is occurring at younger and younger ages. Although type 2 diabetes typically affects individuals older than 40 years, it has been diagnosed in children as young as 2 years of age. About 90% of patients who develop type 2 diabetes are obese. Previous reports indicated that Diabetes Mellitus Type 2 was present in 2 to 3% of all children with diabetes mellitus. Recent statistics, in one Midwestern diabetes clinic demonstrated that Diabetes Mellitus Type 2 increased from 4% (1982 – 1991) to 16% (1994). And now approximately 33% of all newly diagnosed Diabetes Mellitus Type 2 patients were 10 to 19 year-olds (1994).There is a strong genetic influence on the development of type 2 diabetes, based on the following observations. Diabetes Mellitus Type 2 is 2-6 times more prevalent in African Americans, Native Americans, Pima Indians, and Hispanic Americans, and Asian-Pacific Islanders in the United States than in whites. 39% of patients with Type 2 Diabetes Mellitus have at least one parent with the disease. The lifetime risk for a first-degree relative of a patient with type 2 diabetes is five to ten times higher than that of age and weight matched subjects without a family history of diabetes. The prevalence of impaired glucose tolerance and Type 2 Diabetes Mellitus has increased dramatically in several ethnic groups whose lifestyle has become 'westernized' in the last few decades (e.g. Asians and Northern Europeans). The most striking features in these groups and of most patients who develop type 2 diabetes are increased weight gain and decreased physical activity, each of which increases the risk of diabetes. Some of the genetic markers that have been established in the last several years include: North American Caucasians 20q, Mexican Americans 2q and 11p & 6, Pima Indians 3q, 4p, 9q, 22q, 11q, 1q, 7q and Bosnian Finnish 12q.

The pathophysiology of Diabetes Mellitus Type 2 begins with insulin resistance and obesity. It ends with inflammation and destruction of the beta cells of the pancreas by various mediators. Approximately 30,000 years ago when we were hunter-gatherers and did not know when next meals were expected, some people were able to develop “thrifty genes”. These genes include uncoupling proteins, PPAR-$_{y}$ and PPAR-$_{a}$, CALPAIN 10, and adrenergic receptor polymorphisms. They allowed the body to become insulin resistant by interfering with cell mechanisms that allowed blood glucose to be transported into the cells and be phosphorylated so that the cells could utilize this compound for energy. Therefore the pancreas had to make more insulin to accomplish this action. This action which produced excessive insulin allowed the fat cells to accumulate storage of fats for energy at a time of relative hunger and famine. This provided a much higher survival rate. Then approximately 5000 years ago we became an agrarian society and these thrifty genes helped us gain weight because food became plentiful and we ate multiple times during the day. As time passed we also did less physical activity. The combination of multiple daily meals, less physical work and the thrifty genes has increased our rate of obesity and its complications. Diabetes Mellitus Type 2 is a major

complication. Insulin resistance with the combined inability of the pancreas to maintain adequate compensatory hyperinsulinemia leads to chronic hyperglycemia. Along with decreased muscle activity, the combination forms a vicious cycle in which. Type 2 Diabetes Mellitus follows. Tissue necrosis factors (TNF), free triglycerides and glucosamine, have been implicated in the inflammation and destruction of the beta cells proceeding to insulinopenia and the more difficult to treat of Type 2 Diabetes Mellitus. Sometimes it is difficult to distinguish between Type 1 and Type 2 Diabetes Mellitus. Suffice it to say that if the diagnosis is unclear, insulin therapy should be instituted until one can decide on the correct entity.

The mean age at diagnosis is 12 to 14 years of age. Female-to-male ratio is approximately equal. The prevalence of Type 2 Diabetes Mellitus varies by population groups. Caucasian adolescent incidence has been determined at 0.11 per 1000 but is increasing. African-American adolescents' incidence has been determined at 0.31 per 1000 but is also increasing. Pima Indians range by age groups. From 5-9 years of age (yoa) it is <0.5%, from10-14 (yoa) it is 1.5-3%, from 15-19(yoa) it is 4-5%, and over 35 (yoa) it is 40 to 50%. In Native Americans (Manitoba) from 7-14 (yoa) it is 0.53 per 1000.

The differential diagnosis of Type 2 Diabetes Mellitus includes some of the following. Prediabetes often precedes overt Type 2 Diabetes Mellitus. Prediabetes is defined by a fasting blood glucose level of 100-125 mg/dL. Patients who have prediabetes have an increased risk for macrovascular disease, as well as diabetes. Maturity-onset diabetes of the young (MODY) is a form of type 2 diabetes that affects many generations in the same family with an onset in individuals younger than 25 years. Several types exist. MODY (types 1, 2, and3) have deficient insulin secretion with normal insulin sensitivity. MODY in African-Americans demonstrate low insulin concentration, they are not associated with obesity, and have mitochondrial defects. Gestational diabetes mellitus (GDM) is defined as any degree of glucose intolerance with the onset or first recognition during pregnancy. The complications of diabetes include hypoglycemia and hyperglycemia, increased risk of infections, microvascular complications (ie, retinopathy, nephropathy), neuropathic complications, and macrovascular disease such as hypertension, and coronary artery (CAD). Diabetes is the major cause of blindness in adults aged 20-74 years. Diabetes is the leading cause of nontraumatic lower-extremity amputation and end stage renal disease (ESRD). Any patient showing the signs of hypertension or renal disease should be placed on an angiotensin-converting enzyme (ACE) inhibitors or angiotensin II–receptor blockers (ARBs). These medications may also reduce CAD risk independent of its effects on blood pressure. Statins may be used for hyperlipidemia. These complications must be searched for and given the appropriate treatment even in children. Chronic hyperglycemia is associated with an increased risk of microvascular complications, as shown in the Diabetes Control and Complications Trial (DCCT) of Type 1 Diabetes Mellitus and the United Kingdom Prospective Diabetes Study (UKPDS) of Type 2 Diabetes Mellitus. It is clear that complications in either type of diabetes can be either micro or macro vascular. In the DCCT, intensive therapy to maintain normal blood glucose levels greatly reduced the development and progression of retinopathy, microalbuminuria, proteinuria, and neuropathy over 7 years. Intensive therapy was not

associated with increased mortality or incidence of major macrovascular events and did not decrease the quality of life, though it did increase the likelihood of severe hypoglycemic episodes. In the UKPDS, more than 5000 patients with type 2 diabetes were followed up for up to 15 years. Those in the intensely treated group had a significantly lower rate of progression of microvascular complications than that of those receiving standard care. Rates of macrovascular disease were not altered except in the metformin-monotherapy arm, in which the risk of MI was significantly decreased. Moreover, severe hypoglycemia occurred less often than it did in patients with type 1 diabetes in the DCCT. Immunological, reproductive, and teratogenic complications are common in Diabetes Mellitus Type 2.

The following laboratory studies are helpful in diagnosing Type 2 Diabetes Mellitus or differentiating it from Type 1 Diabetes Mellitus. All laboratory studies should be individualized to the clinical situation. A finger stick glucose test is appropriate for virtually all patients with diabetes. In patients who present with symptoms of uncontrolled diabetes (e.g., polyuria, polydypsia, nocturia, fatigue, and weight loss) with a confirmatory random plasma glucose level of >200 mg/dL, diabetes can be diagnosed. In asymptomatic patients whose random serum glucose level suggests diabetes, a fasting plasma glucose (FPG) concentration should be measured. The oral glucose tolerance test no longer is recommended for the routine diagnosis of diabetes. An FPG level of $\geq$126 mg/dL on 2 separate occasions is diagnostic for diabetes. An FPG level of 110-125 mg/dL is considered impaired fasting glucose (IFG). An FPG level above >90 mg/dL may be associated with an increased risk for the metabolic syndrome if other features are present. A fasting C-peptide level >1 ng/dL in a patient who has had diabetes for more than 1-2 years is suggestive of type 2 diabetes (ie, residual beta-cell function). Islet-cell autoantibodies are present in early Type 1 but not Type 2 Diabetes Mellitus. Measurements of these autoantibodies within 6 months of diagnosis can help differentiate Type 1 and Type 2 Diabetes Mellitus. However, titers of islet-cell autoantibodies decrease after 6 months. Anti–glutamate decarboxylase (GAD) antibodies can be present at diagnosis of Type 1 Diabetes Mellitus and are persistently positive over time. Therapeutic options can be grouped into 3 categories. (1) Weight control through diet & exercise. (2) Oral hypoglycemic agent. (3) Insulin therapy. Treatment of Diabetes Mellitus Type 2 necessitates a multidisciplinary approach which includes the physician, diabetes nurse educator, dietician, exercise physiologist or personal trainer, psychosocial worker and psychologist. The main points of our therapy consist of changing the diet and emphasizing exercise. Our dietician stresses a lower carbohydrate diet (40% of total daily calories) spread evenly over the entire day. Protein and fat may have to be increased slightly but we have not found an adverse affect on serum lipids. Complex carbohydrates and fruits and vegetables are emphasized in our clinic. Patients are taught carbohydrate counting (grams of carbohydrate) so that they make intelligent choices and substitutions. Foods with monounsaturated fats are stressed. We believe that a program of intense exercise is necessary for weight loss.

Oral hypoglycemic agents decrease blood glucose by increasing insulin secretion, increasing insulin action, decreasing hepatic glucose output, decreasing nutrient absorption, and sensitizing fat liver and muscle cells to insulin. There are now 7 classes

of medications that are used to treat patients with diabetes. Only the biguanide, metformin is FDA approved for children, but some of the others are used in older adolescents and off label as combined therapy in cases that are difficult to control. Oral medications consist of Biguanides, Sulfonylureas, Meglitinides, Alpha-glucosidase inhibitors (AGIs), Thiazolidinediones**,** Incretin mimetics, and Amylin analogues.

Biguanides increase peripheral sensitivity to insulin, decreases hepatic glucose output. They also stimulate weight loss, and improve plasma lipid profile. Their side effects include anorexia, nausea, and diarrhea. Malabsorption of vitamin B12 and anemia are uncommon. Lactic acidosis has been reported with phenformin (a similar molecule in renal/hepatic/cardiac dysfunction). It is very rare with metformin occurring only 1 in 50,000 cases.

Sulfonylureas increase insulin secretion by enhancing beta cell responsiveness to glycemic stimuli. They attach to beta cell surface receptors (ATP-dependent potassium channels), cause depolarization, calcium influx and stimulation of insulin release. Chlorpropamide is a 1st generation drug. Glyburide and Glipizide are 2nd generation drugs. The side effects include hypoglycemia, nausea, abdominal discomfort, skin reactions, abnormal liver function tests, jaundice, hematological complications, hyponatremia and fluid retention (Chlorpropamide).

Meglitinides agents are short-acting insulin secretagogues. They act on the ATP-dependent potassium channels in pancreatic beta cells, allowing opening of calcium channels and increased insulin release. Repaglinide (Prandin) is a meglitinide that stimulates insulin release from pancreatic beta cells. The side effects include hypoglycemia and abnormal liver function tests. **AGIs** (Acarbose) do not increase insulin levels or inhibit lactase; their major effect is to lower postprandial glucose levels (lesser effect on fasting levels). They do not cause weight gain. Alone, AGIs do not cause hypoglycemia. Less than 2% is absorbed as active drug. They are used as monotherapy or combined with sulfonylurea, TZD, metformin, or insulin. Taken with food minimizes the GI effects.

Thiazolidinediones (Rosiglitazone) are agents that increase peripheral insulin sensitivity by increasing transcription of nuclear proteins that help increase uptake of glucose, probably with effects on free fatty acid levels. About 12-16 weeks are needed to achieve maximal effect. They are an insulin sensitizer, and may be used as monotherapy or combined with sulfonylurea, metformin, a meglitinide, or insulin. Its major effect is stimulating glucose uptake in skeletal muscle and adipose tissue. It lowers plasma insulin levels. The side effects of Rosiglitazone are hypersensitivity, active liver disease, congestive heart failure and diabetic ketoacidosis (DKA). They are known to cause an unexplained weight gain. It may increase the risk of hypoglycemia when used in combined therapy.

Incretin mimetics mimic glucose-dependent insulin secretion, suppresses elevated glucagon secretion, and delays gastric emptying. Exenatide (Byetta) stimulates glucose-dependent insulin secretion and several other antihyperglycemic actions of incretins.

They improve glycemic control in patients with Type 2 Diabetes Mellitus by enhancing glucose-dependent insulin secretion by pancreatic beta cells, suppress inappropriately elevated glucagon secretion, and slow gastric emptying.

Amylin analogs are agents have endogenous amylin effects by delaying gastric emptying, decreasing postprandial glucagon release, and modulating appetite. Pramlintide acetate (Symlin) is a synthetic analogue of human amylin, hormone made in beta cells. It slows gastric emptying, suppresses postprandial glucagon secretion, and regulates food intake (centrally mediated appetite modulation). They are indicated to treat type 1 or 2 diabetes, with insulin.

The latter 2 classifications of medications have not been tested in children and we await further studies to determine if they will be a benefit in pediatric Type 2 Diabetes Mellitus. The goal of oral antidiabetic therapy is to lower blood glucose levels to near-normal (preprandial levels of 90-130 mg/dL or 80-140 mg/dL and HbA1C levels <7%) and to maintain them in this range for the patient's lifetime. The goal of these combined daytime oral agents is to lower the fasting glucose level to 100 mg/dL. If this target is not achieved, once daily long acting insulin (Insulin-Glargine) should be instituted. If a regimen combining oral agents and insulin fails to lower glucose levels into the normal range, patients should be switched to a daily multiple-injection schedule with premeal very rapid-acting insulin and a longer-acting basal insulin. The new inhaled insulin (Exubera) is not yet approved for children. But studies in the pediatric age group are ongoing.

There is a new urgency and a new unknown dynamic in diagnosing and treating Type 2 Diabetes Mellitus. We are obligated to protect our younger population with this disease.

NOTES

AMERICAN COLLEGE OF OSTEOPATHIC PEDIATRICIANS

TUESDAY, OCTOBER 2, 2007

6:30 am - 5:00 pm	Registration	*San Diego Convention Center - 4 Foyer*
7:00 am - 8:00 am	**ACOP LECTURE** ***Evaluation of Newborn with Dysmophic Features*** Andrea Amalftano, DO, PhD	*San Diego Convention Center - 4*
8:00 am - 9:00 am	**ACOP LECTURE** ***Ambulatory Care of NICU Graduates*** Michelle Bez, DO, FACOP	
9:00 am - 10:00 am	**ACOP LECTURE** ***Death in the NICU: Moral Distress in Health Care Providers*** Liz Sumner, RN, BSN	
10:00 am - 11:00 am	**ACOP LECTURE** ***Early Recognition of Spasticity in Infants*** Kevin Z. Craig, DO	
11:00 am - 12:00 n	**ACOP LECTURE** ***Pre and Probiotics: Are They Wise to Add to Infant Nutrition?*** Carey Walker, PhD	
12:00 n - 12:30 pm	Exhibit Break	
12:30 pm - 2:00 pm	General Membership Luncheon	*San Diego Convention Center - 14B*
2:00 pm - 3:00 pm	**ACOP LECTURE** ***New Expanded Newborn Metabolic Screen*** Michael Kayser, DO	*San Diego Convention Center - 4*
3:00 pm - 4:00 pm	**ACOP LECTURE** ***New Techniques for the Diagnosis of Genetic Diseases*** Michael Kayser, DO	
4:00 pm - 5:00 pm	**ACOP LECTURE** ***Current Concepts in Genetic Counseling*** Michelle A. Fox, MS, BA, CGC	
5:00 pm - 6:00 pm	**CME Committee Meeting**	
7:00 pm - 10:00 pm	**AOA/AAOA Presidential Reception**	

NOTES

American College of Osteopathic Pediatricians

TUESDAY, OCTOBER 2, 2007

7:00 am - 8:00 am

ACOP LECTURE
Evaluation of Newborn with Dysmophic Features

Andrea Amalfitano, DO, PhD

After this presentation the participant will be able to:

1. Review broadening Scope of Medical Genetics
2. Review frequency of birth defects and their implications for pediatricians
3. Understand basic definitions and terminology
4. Review aspects of history/physical evaluations
5. Review tenets of dysmorphology
6. Review indications of chromosome/genetic analysis
7. Aspects of common syndrome identification

NOTES

Evaluation of the Dysmorphic Child:

By:
Andrea Amalfitano D.O., Ph.D
Professor of Microbiology, Molecular Genetics, and Pediatrics
Michigan State University
East Lansing, MI

Objectives

- Review Broadening Scope of Medical Genetics
- Review Frequency of Birth Defects and Their Implications for Pediatricians
- Basic Definitions and Terminology
- Review Aspects of History/Physical Evaluations
- Review Tenets of Dysmorphology
- Review Indications for Chromosome/Genetic analysis
- Aspects of Common Syndrome Identification

Financial Disclosure:

- Genzyme Corporation:
  - Paid Consultant
- Etubics Corporation:
  - Scientific Advisor
  - Paid Consultant
  - Stock Owner

Medical Genetics in Primary Care:

71% of American adults say they would most likely ask their ***primary care physician*** about a genetic disorder present in their family.

Genetic Testing: A study of consumer attitude AMA March 1998

Medical Genetics and Infant Deaths:

- **3-5%** of all births result in congenital malformations (Robinson A. and Linden MG. 1993. Clinical Genetic Handbook, Boston, Blackwell Scientific Publications)
- **0.5%** of all newborns and **7%** of all stillborns have a chromosome abnormality
- **30-50%** of post-neonatal deaths are due to congenital malformations (Hoekelman RA, Pless IB. 1988. Decline in mortality among young American during the 20th century: Prospects for reaching national mortality reduction goals for 1990. Pediatrics 82:582-95)
- **20-30%** of all infant deaths are due to genetic disorders (Berry RJ, Buehler JW, Strauss LT, et al. 1987. Birth weight-specific infant mortality due to congenital abnormalities, 1960 and 1980. Public Health Report 102:171-81)

Genetics and Hospital Admissions

- **18.5-50%** are children with other congenital malformations (Scriver CR, Neal JL, Saginur R, and Clow A. 1973. The frequency of genetic disease and congenital malformation among patients in a pediatric hospital. Canadian Medical Association Journal 108:1111-15
- **11.1%** of pediatric hospital admissions are for children with genetic disorders and
- **12%** of adult hospital admissions are for genetic causes (Emery AEH, Rimoin DL. 1990. Principles and Practice of Medical Genetics, Second Edition. New York, Churchill Livingstone)

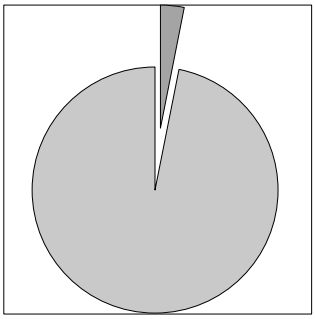
Medical Genetics and Chronic Adult Diseases

- **15%** of all cancers have an inherited susceptibility (Schneider KA. 1994. Counseling about Cancer: Strategies for Genetic Counselors. Dennisport, Massachusetts, Graphic Illusions)
- **10%** of the chronic diseases (heart, diabetes arthritis) which occur in the adult populations have a significant genetic component (Weatherall DJ. 1985. The New Genetics and Clinical Practice, Second Edition. Oxford: Oxford University Press

Medical Genetics and Mental Retardation:

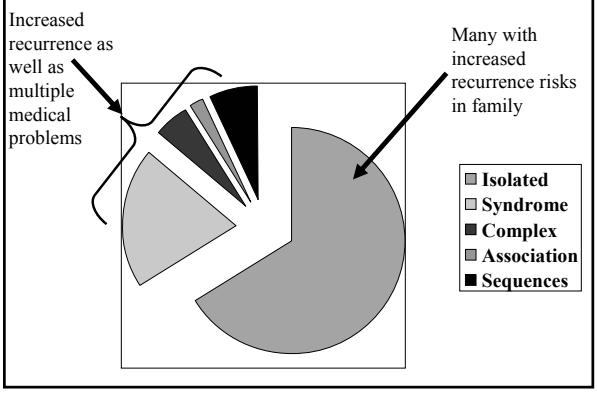
- 50% of mental retardation has a genetic basis (Emery and Rimoin, 1990)

Incidence of Birth Defect Overall:



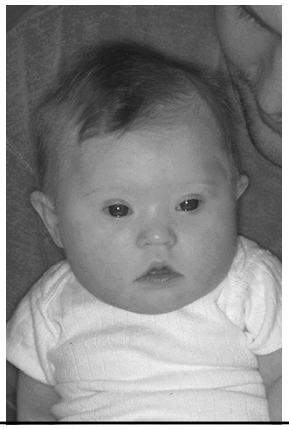
•Major malformations interfere with normal function and require medical attention (3% of all children at birth, 6% of all by age 5)

Etiologies of Birth Defects:

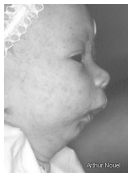


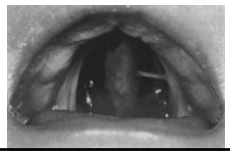
Definitions:

- **Syndrome: A recurring pattern of MCA's representing a specific etiology, (due to either genetic and/or environmental causes)**
  - **Down, Williams, Beckwith-Weideman,**
- **Sequence: single event/anomaly results in cascade effect and multiple malformations:**
  - **i.e: Pierre-Robin sequence :**
    - **Micrognathia-glossoptosis-U-shaped Cleft Palate**
- **Association : A non-random occurrence of MCA's not associated with a specific genetic etiology,,,yet**



Pierre Robin Sequence is characterized by a combination of three features, possibly due to the underdevelopment of the lower jaw. The lower jaw is abnormally small (micrognathia), the tongue is displaced downwards (glossoptosis), and there is an abnormal opening in the roof of the mouth (cleft soft palate).

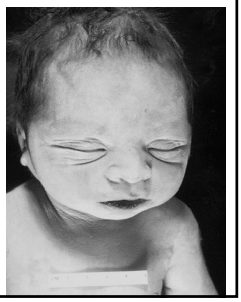





Definitions: Classes of Dysmorphology/Malformations

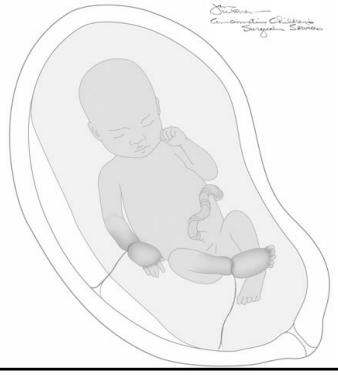
- **Deformation:** primary structure was normal, malformation is secondary to extrinsic forces

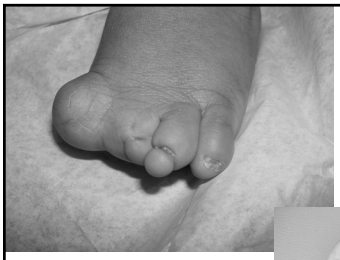
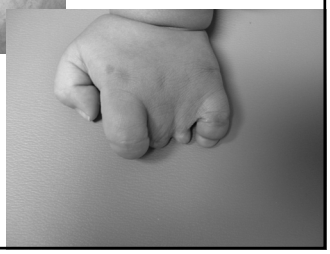
Renal Agenesis/Obstruction ~~d~~decreased amniotic fluid ~~m~~micrognathia, abnormal facies, decreased pulmonary development, ±prune belly



Definitions: Classes of Dysmorphology/Malformations

- Disruption: actual destruction of previously normal tissue.
- Results in Minor or Major Anomalies.
- Causes:
  - Extrinsic forces,
  - Ischemia
  - amniotic bands



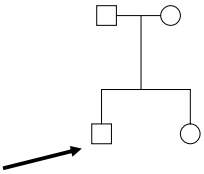
Medical Genetics Evaluation for Syndrome Identification:

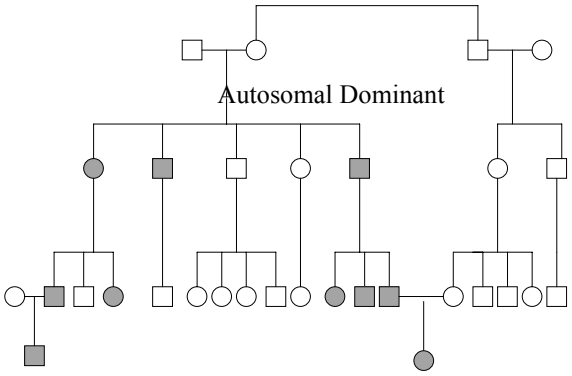
- Complete medical history!
  - Review of records and previous evaluations
- Family history and pedigree
- Physical examination
  - Dysmorphology (Major/minor anomalies)
- Differential Diagnosis to Guide
- Laboratory Investigations
- Referrals for: ophthalmology, audiology, ultrasound, x-ray, etc…
- Diagnosis?
- **Follow-up!**

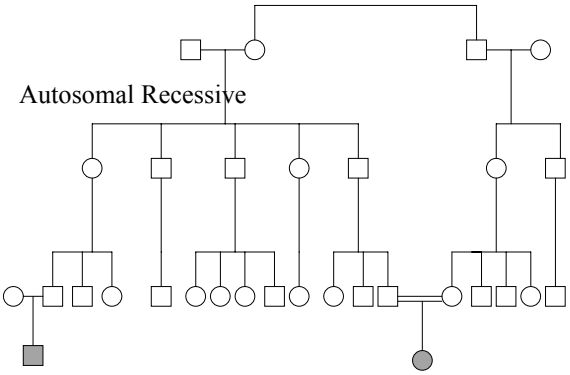
Family/Medical history

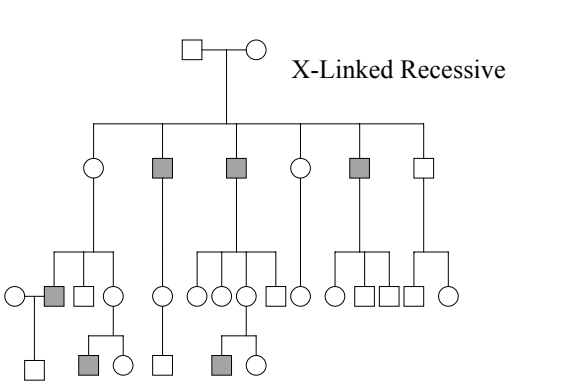
- Accurate <u>prenatal</u>/birth history, medical history, and family history with a three generation pedigree are an essential first step
- Any other individuals with MR, autism, psychiatric disorders, learning problems, congenital abnormalities
- Obtain clinical documentation
- Photographs of patient and family

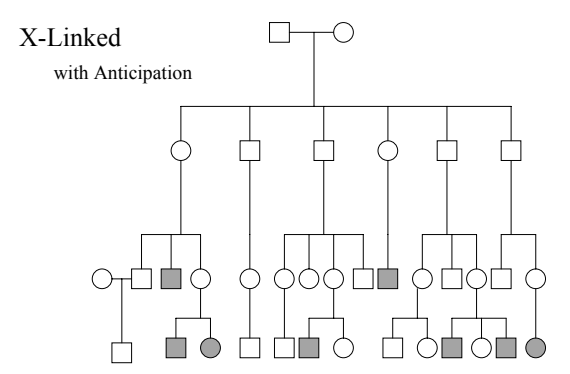
This is NOT a pedigree

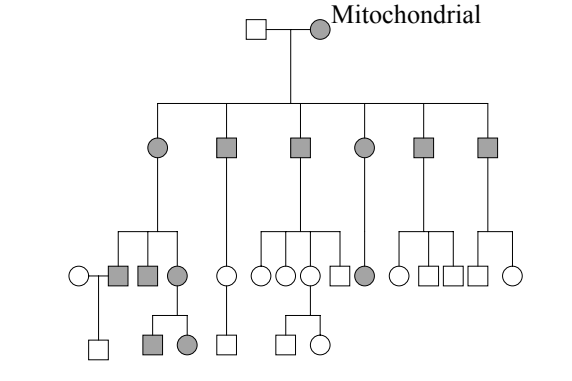












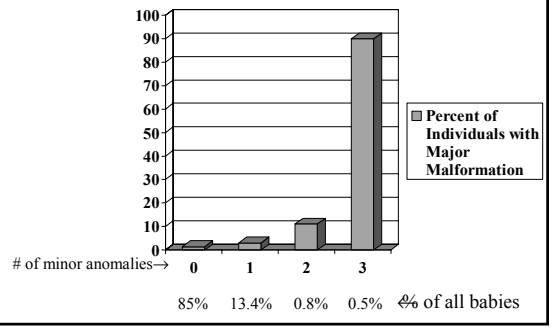
Physical Examination:

- **Anomalies:** malformation due to a primary defect, occurs early in gestation
- **Major Anomaly:**
  - spina bifida, anopthalmia, CL/CP,transposition,renal agenesis,holoprosencephaly
- **Minor Anomaly:**
  - low set ears, hypospadias, bifid uvula
  - ASD, VSD, nail hypoplasia, post-axial polydactyly
  - 4th metacarpal shortening, synophyris, ear tag, clinodactyly
- **Isolated Anomaly:** **single structural defect, not associated with a syndrome**
- **Multiple Congenital Anomalies or Syndrome:** **2 or more defects in 2 or more systems**
  - **Infant of Diabetic Mother**
  - **Down Syndrome**

Physical Exam: Dysmorphology

- The study of abnormal embryonic and fetal development and the resultant change (anomaly) in body form
- **Minor anomaly identification is the KEY to helping identifying specific syndromes!**

As the number of minor anomalies increases, so does the risk of having a major malformation:

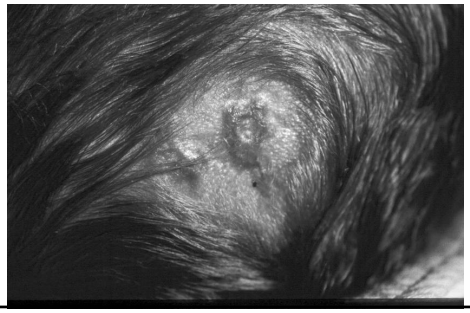


Minor Malformations/Anomalies

- Head: brachycephlay, hair pattern

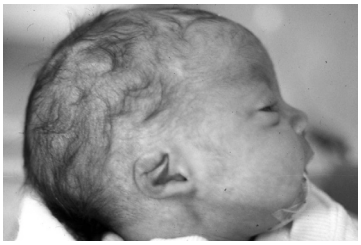
Minor Malformations

- Head:cutis aplasia

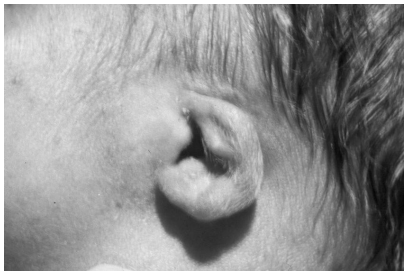


Minor Malformations

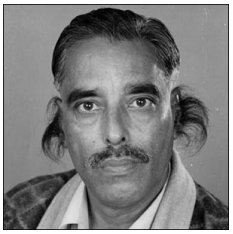
- Ears: low set ears, cupped ears, microtia



Minor Malformations

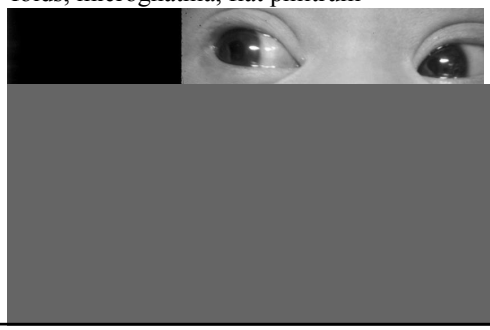


Minor Malformations



Minor Malformations

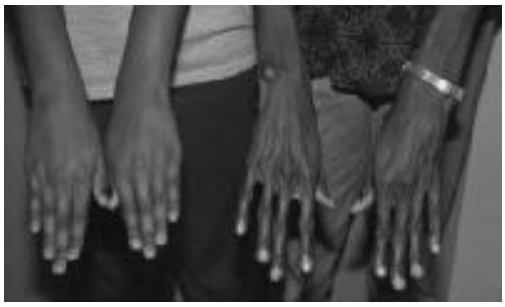
- Face: palpebral fissure slant, epicanthal folds, micrognathia, flat philtrum



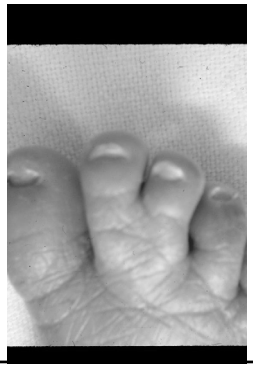
Minor Malformations

- Limbs: simian crease, clinodactyly, 2-3 syndactyly

Arachnodactyly

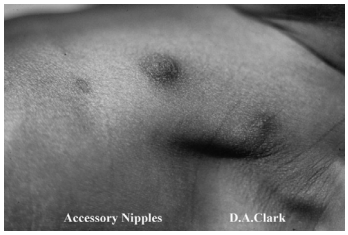


Minor Malformations

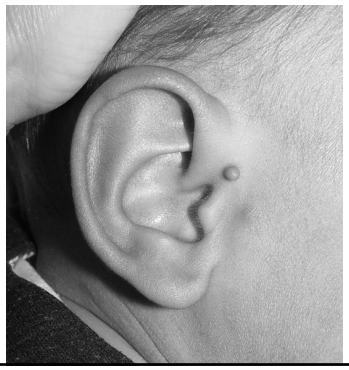


Minor Malformations

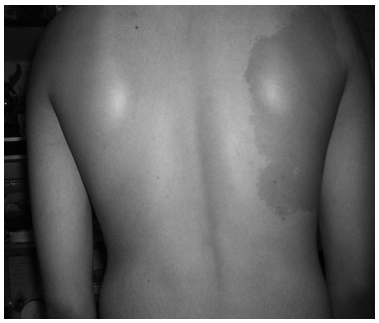
- Skin: tags, sinuses, hemangiomas, café alu lait, accessory nipples



Minor Malformations



Minor Malformations



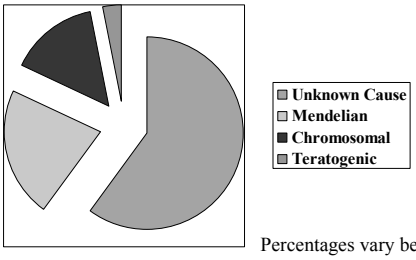
History and Physical guide the ordering of Genetic Tests

As the number of anomalies increases (combined with medical history and pedigree facts) so to does the need for further genetic/medical investigations.

Genetic/Ancillary Tests

- Chromosome analysis
  - **<u>karyotype, standard or high resolution</u>**
    - FISH: for specific gene region/microdeletion
    - subtelomeric FISH panel
    - Genomic Scan (should be considered after karyotype negative, and when considering FISH)
- Fragile X DNA analysis: MR without other anomaly
- Neuroimaging; Metabolic testing
- Specific DNA tests: primarily useful for identification of at risk family members, not diagnostically!

Common Genetic Diseases/Syndromes



Percentages vary between studies:
~15% of all Syndromes
~15-30% severe MR, 5-10% mild MR due to <u>detectable</u> chromosome changes

Indications for Chromosome analysis

- Microcephaly
- Multiple minor anomalies
- Non-isolated Major anomaly
- Stature
- Family history MR
- Family history of fetal loss
- IQ<50
- Skin pigmentary anomalies
- Suspected gene syndrome

Chromosome Disorders

- Abnormal chromosome number : Trisomies
- Unbalanced Structural chromosome abnormalities: translocations, inversions, markers
- Chromosome microdeletions
  - Angelman syndrome - 15q11-q13
  - Prader-Willi syndrome - 15q11-q13
  - DiGeorge syndrome - 22q11
  - Rubinstein-Taybi syndrome - 16p13.3
  - William syndrome - 7q11
- Cryptic subtelomeric deletions (~ 6-7% of idiopathic MR)

Why Make a Genetic Diagnosis?

- Treatment is symptomatic
- Essential for appropriate management
- Early recognition and prevention of complications
  - Allows earlier diagnosis in other family members
  - Prevents unnecessary additional diagnostic evaluations
    ie: CF screening-early dietary intervention =improved cognition
- Anticipatory guidance
  - Health maintenance plans
- Psychological well-being for family
  - May continue to search for diagnosis until one is made
  - Family support groups
- Genetic counseling regarding recurrence risks, at-risk testing, reproductive options
- Newer Therapies becoming available for rarer genetic diseases!

No Diagnosis

- Often no diagnosis can be made after the first evaluations
  - 50-60% of the time no diagnosis
- Follow up is critical
- Serial evaluations are very helpful
- Evolution of a specific recognizable physical or behavioral phenotype may aid diagnosis
- New diagnostic methods are constantly being developed

TUESDAY, OCTOBER 2, 2007

8:00 am - 9:00 am

ACOP LECTURE
Ambulatory Care of NICU Graduates

Michelle Bez, DO, FACOP

NOTES

Life after the NICU

Michelle Bez, DO, FACOP

I. One last test before exit
   A. +/- Altitude Challenge Test
   B. "Nesting"

II. Within the First 1-2 days after DC to Home
   A. Primary care Doc gets to know baby
      i. Review DC summary
      ii. H&P
   B. Weight check and Head Circumference
   C. Home Health Visits
      i. Parent anti-anxiety
      ii. Neo/Ped/FP anti-anxiety

III. Two Weeks after DC to Home
   A. Weight check and Head Circumference
   B. Hct/Retic, alkaline Phosphatase and Ca++, Phos

IV. Regularly Scheduled intervals determined by PPC to address specific issues and Routine Visits

V. Special Circumstances
   a. Nutrition
      i. Weight, length, HC
      ii. How long to fortify breast milk or formula
      iii. Alternative Diets
         1. Vegans
         2.
   b. Anemia of Prematurity
   c. Retinopathy of Prematurity
   d. Immunizations
      i. Hepatitis B
      ii. Prevnar
      iii. Other routine vaccines
   e. Immunoprophylaxis
      i. Who really should get Synagis?
      ii. How long? How many seasons?
   f. Weaning O2
      i. Acclimating to altitude
      ii. Resolution of CLD

g. Gastroesophageal Reflux
    i. Evidence based medicine
    ii. Adjustment of meds.
    iii. Follow up studies
h. Developmental Delays Identifying
    i. Early Intervention
    ii. OT/PT/Speech
    iii. State Resources
i. Hearing
    i. Identification
    ii. Follow up; what test is best
    iii. Hearing-Gene?
j. Trachs, G-tubes
k. Parent support
    i. Support Groups
    ii. Local parents one on one
    iii. Counseling
    iv. Spiritual/Cultural
    v. Post-partum Depression
l. Internet sites for Primary care Providers

VI.

VII.

VIII.

IX.

X.

AMERICAN COLLEGE OF OSTEOPATHIC PEDIATRICIANS

TUESDAY, OCTOBER 2, 2007

9:00 am - 10:00 am

ACOP LECTURE
Death in the NICU: Moral Distress in Health Care Providers

Liz Sumner, RN, BSN

After this presentation the participant will be able to:

1. Describe two clinical examples that trigger moral distress in the health care professional
2. Identify strategies relevant to their setting to respond to moral distress and suffering
3. Write down personal attributes that help to preserve emotional resilience and capacity for uncertainty in the health care professional

NOTES

TUESDAY, OCTOBER 2, 2007

10:00 am - 11:00 am

ACOP LECTURE

Early Recognition of Spasticity in Infants

Kevin Z. Craig, DO

NOTES

Spasticity in Pediatrics

Kevin Z. Craig, DO

1. **Physiology of Spasticity**
2. **Disease States**
3. **Treatment of spasticity**

Pathophysiology of Spasticity:

Normal brain delivers inhibitory neural signals to the spinal cord
Inhibitory signals modulate reflex signals–tone remains normal
Damaged brain fails to generate or sends inadequate inhibitory signals
Lack of neural inhibition leads to spasticity

Definition of Spasticity:

Spastikos (Greek) = to tug or to draw
It is a motor disorder
Increase in muscle stretch reflexes
It is velocity dependent
Associated positive symptoms (flexor response, Babinski response)
Associated negative symptoms (decrease dexterity, paresis/weakness, fatigability)

Quantification of Spasticity

Ashworth Scale

Score	Criteria
1	No increase in tone
2	Slight increase in tone
3	Marked increase in tone, but affected part(s) easily flexed
4	Considerable increase in tone; passive movement difficult
5	Affected part(s) rigid in flexion or extension

Modified Ashworth Scale

Score	Criteria
0	No increase in tone
1	Slight increase in tone (catch and release at end of ROM)
1+	Slight increase in tone, manifested by a catch, followed by minimal resistance throughout remainder (less than half of the ROM)
2	Marked increase in tone through most of ROM, but affected part(s) easily moved
3	Considerable increase in tone; passive movement difficult
4	Affected part(s) rigid in flexion or extension

Tardiu Scale

Possible Advantages of Spasticity

- Maintains muscle tone
- Helps support circulatory function
- May prevent formation of deep vein thrombosis
- May assist in activities of daily living

Consequences of Spasticity

- May interfere with mobility, exercise, joint range of motion
- May interfere with activities of daily living
- May cause pain and sleep disturbance
- Can make patient care more difficult

Factors That May Increase Spasticity

Uncontrollable

- Urinary tract infection
- Kidney stones
- Menses
- Bowel impaction or gas
- Deep vein thrombosis
- Pneumonia
- Wounds or infections
- Progression of disease

Controllable

- Stress
- Ingrown nails
- Restrictive clothing
- Fatigue
- Psychological factors
- Change in temperature or humidity

Spasticity of Spinal Origin

Most Common Causes

- Spinal Cord Injury (SCI)
- Spinal Cord Disease
- Multiple Sclerosis (MS)

The Spinal Cord Injury Process

- Spinal shock
  - first several weeks to three months
- Spasticity develops
  - two weeks to several months
- Spasticity commonly peaks at two years post-injury, then may decrease slightly and stabilize
- Muscle spasms
- Spasticity Associated with Multiple Sclerosis (MS)
  "Many Scars"
  Demyelinating disease
  Affects CNS
  Cause: "Autoimmune Theory"
  - exact cause unknown

Spasticity of Cerebral Origin

Common Diagnoses

- Cerebral palsy
- Brain injury
- Stroke

Spasticity Associated with Cerebral Palsy (CP)

- Disorders affecting
  - movement
  - posture
  - balance
- Injury to the developing brain
- Permanent and non-progressive
- Developmental disability

Classifications of Cerebral Palsy

- Location of brain lesion
  - pyramidal, extrapyramidal, mixed
- Type of movement disorder
  - spastic, dystonic, athetoid, ataxia, mixed
- Extent and location of limb involvement
  - monoplegia, diplegia, hemiplegia, paraplegia, tetraplegia

Spasticity Associated with Brain Injury (BI)

- Acquired brain injury
- Traumatic brain injury
- Brain attack (stroke)

Goals of Spasticity Management

- Decrease spasticity
- Improve functional ability and independence
- Decrease pain associated with spasticity
- Prevent or decrease incidence of contractures
- Improve ambulation
- Facilitate hygiene
- Ease rehabilitation procedures
- Save caregivers' time

Approach to Management of Spasticity

Remove noxious stimuli
Rehabilitation Therapy
Oral medications
Neurolysis
Intrathecal Baclofen Pump
Neurosurgical procedures
Orthopedic procedures

Preventing Nociception

- Identify the "triggering" stimulus
- Eliminate the factors that increase sensory input to the spinal cord

Rehabilitation Therapy

- Stretching and positioning
- Weight bearing
- Cryotherapy
- Inhibitory casting
- Vibration of the antagonist
- Pool therapy
- EMG biofeedback
- Electrical stimulation

Oral Medications

- Baclofen
- Diazepam
- Dantrolene Sodium
- Tizanidine
  Others

Site of Action for Oral Drugs

Drug	Site of action
Baclofen:	GABAb receptors in spinal cord
Diazepam:	Central nervous system
Dantrolene Sodium:	Skeletal muscles beyond the myoneural junction
Tizanidine:	Central acting (spinal and supraspinal) at alpha2 – adrenergic receptor sites

Injection Therapy

- Nerve blocks
  - Diagnostic
    - procaine
    - lidocaine
    - bupivacaine
  - Neurolytic
    - phenol
    - ethanol
- Botulinum toxin

Nerve Blocks

- Anesthetic
  - Diagnostic (procaine, lidocaine, bupivacaine)
- Neurolytic
  - Agent
    - phenol
    - ethanol

Botulinum Toxin

- Can be administered without anesthesia
- No systemic effect
  - diffuses readily into the muscle
- Facilitates treatment goals
- Effects are local and dose-dependent
- Can be used with other therapies
  - including ITB Therapy

Neurosurgeries
Surgical Treatments
Neurodestructive Procedures

- Neurectomy
- Myelotomy
- Rhizotomy
- Cordectomy
- Selective Dorsal Rhizotomy

Selective Dorsal Rhizotomy
Surgical procedure where the dorsal (sensory) nerve roots are severed

- Two primary goals:
  - facilitate patient care
    - sitting, dressing, transfers
  - improve function
    - walking

Orthopedic Surgeries
Soft Tissue Procedures

- Tenotomy
- Tendon lengthening
- Myotomy
- Tendon transfers

Intrathecal Baclofen (ITB™) Therapy
Used to treat individuals with severe spasticity due to:

- Cerebral palsy
- Brain injury
- Brain attack (stroke)
- Spinal cord injury
- Multiple sclerosis

Potential Benefits of ITB™ Therapy

- Decreased spasticity
- Reversible
- Noninvasive programming
- Improved activities of daily living
- Ease of care
- Decreased pain related to spasticity

Potential Risks of ITB™ Therapy

- Drug side effects
  - Most common:
    - hypotonia, somnolence, nausea/vomiting, headaches, dizziness
- System and procedural complications
- Overdose (rare)

Advantages of ITB™ Therapy

- Reversible
- Potentially fewer systemic side effects
- Programmable
  - allows dose titration to give optimal benefit
- Effective in reducing spasticity
  - upper and lower extremities1
  - cerebral and spinal origin

Potential Risks of ITB™ Therapy

- Drug side effects
  - Most common
    - hypotonia, somnolence, nausea/vomiting, headaches, dizziness
- System and procedural complications
- Overdose (rare)

Indications for ITB™ Therapy

- Patients must demonstrate a positive response to the screening test
- Patients with spasticity of spinal origin:
  - unresponsive to oral antispasmodics
  - and/or experience unacceptable side effects at effective doses of oral baclofen
- Patients with spasticity of cerebral origin must be one year post brain injury to be considered for ITB Therapy

Most Common Side Effects of Baclofen Injection

- Hypotonia
- Somnolence
- Nausea/vomiting
- Headache
- Dizziness

Signs of Baclofen Overdose

- Drowsiness
- Lightheadedness
- Dizziness
- Somnolence
- Respiratory depression
- Seizures
- Rostral progression of hypotonia
- Loss of consciousness progressing to coma

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NOTES

American College of Osteopathic Pediatricians

TUESDAY, OCTOBER 2, 2007

11:00 am - 12:00 n

ACOP LECTURE
Pre and Probiotics: Are They Wise to Add to Infant Nutrition?

Carey Walker, PhD

After this presentation the participant will be able to:

1. Recognize potential contributions of gastrointestinal microbiota to infant development and health
2. Define prebiotic carbohydrates and give examples
3. Define probiotic microorganisms and give examples
4. Effectively engage in discussions of the potential relevance of probiotic microorganisms and prebiotic carbohydrates in infant nutrition

NOTES

American College of Osteopathic Pediatricians

TUESDAY, OCTOBER 2, 2007

2:00 pm - 3:00 pm

ACOP LECTURE
New Expanded Newborn Metabolic Screen

Michael Kayser, DO

After this presentation the participant will be able to:

1. Identify the different areas of a practice that can be enhanced to improve efficiency and outcomes
2. Improve the reimbursement efficiency to maintain a thriving practice

NOTES

New Expanded Newborn Metabolic Screen

Michael A. Kayser, D.O.
Medical Director
Warren Clinic Center for Genetics
Center for Genetic Testing at Saint Francis
Tulsa, OK

Objectives

- Brief history of newborn screening
- Recent expansion of newborn screening
- What to do with abnormal results?
- New tests on the horizon

History of Newborn Screening

- Phenylketonuria (PKU)
  - dietary treatment developed in 1950s
  - Treatment prevented neurologic damage, but could not reverse damage once it had occurred
  - Bacterial Inhibition assay using filter paper developed by Dr. Robert Guthrie
  - Screening started in parts of New England in 1961

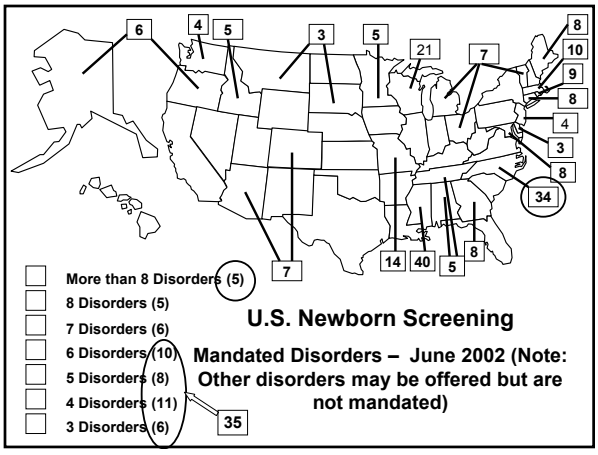
Dr. Guthrie

"Guthrie Card"

History of Newborn Screening

- Soon afterwards hypothyroidism, galactosemia, and sickle cell disease added to newborn screening panels
- Early 1990's Tandem Mass Spectroscopy (MS/MS)- allowed for inclusion of more than 30 disorders
- Newborn screening is at the discretion of the states- some screened for 3 disorders, some for greater than 30

Tandem Mass Spectrometer



U.S. Newborn Screening

Mandated Disorders – June 2002 (Note: Other disorders may be offered but are not mandated)

Maternal and Child Health Bureau

Advisory Committee on Heritable Disorders and Genetic Diseases in Newborns and Children

Health Resources and Services Administration

U.S. Department of Health and Human Services

- Established 02/04/2004 to assist the Secretary, U.S. Department of Health and Human Services
  - 'Specifically, the Committee shall advise and guide the Secretary regarding the most appropriate application of universal newborn screening tests, technologies, policies, guidelines and programs for effectively reducing morbidity and mortality in newborns and children having or at risk for heritable disorders."

HRSA/ACMG Contract

- Contract with the American College of Medical Genetics 06/08/2004
- **Primary Goals**
  - Developing a uniform panel of newborn screening conditions
    - Developing a decision-making tool for use in NBS program **expansion** or **contraction**
    - Criteria for assessing conditions for their appropriateness for newborn screening

HRSA/ACMG Uniform Panel Survey

- What disorders to include in survey?
  - Wilson and Junger 1968 WHO criteria
  - Steering Committee and Expert Group formed
  - Seeking input (invited speakers- International and U.S. participants, public comment opportunities, direct requests of input from other interest groups, literature review)

Uniform Panel Survey

- **Conditions Included in Survey**
  - Endocrine disorders (CAH, CH, IDDM) 3
  - Infectious diseases (HIV, Toxo, CMV) 3
  - Hematologic disorders (Hb-pathies, G6PD) 5
  - Genetic conditions (CF, DMD, FX, Wilson) 12
    - IEM detectable by MS/MS (AA, AC)
    - Amino acid disorders (PKU, MSUD, HCY) 16
    - Fatty acid disorders (MCAD, VLCAD) 14
    - Organic acid disorders (PA, MMA, IVA, GA-I) 15
  - Other IEM (± detectable by MS/MS) **45**
    - Carbohydrate disorders (GALT, CDG) 4
    - Lysosomal disorders (Fabry, Krabbe, Pompe) 5
    - Others (BIOT, ALD, SLO) 6

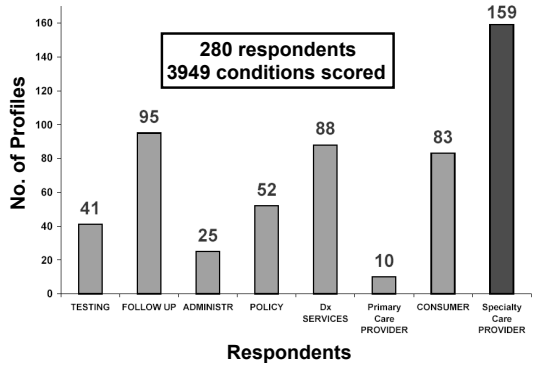
Total **83**

Criteria and Scoring System

- **Incidence of conditions**
- **Identifiable at birth**
- **Burden of disease**
- **Availability of test**
- **Test characteristics**
- **Availability of treatment**
- **Cost of treatment**
- **Efficacy of treatment**
- **Benefits to individual**
- **Benefits to Fam. & Soc.**
- **Mortality prevention**
- **Diagnostic confirmation**
- **Acute management**
- **Simplicity of therapy**

CRITERIA	CATEGORIES	SCORE
Incidence of condition	>1:5,000	100
	>1:25,000	75
	>1:50,000	50
	>1:75,000	25
	<1:100,000	0
Sign & Symptoms clinically identifiable in the first 48 hours	None	100
	<25% of cases	75
	<50% of cases	50
	<75% of cases	25
	Always	0
Burden of disease (Natural Hx if untreated)	Profound	100
	Severe	75
	Moderate	50
	Mild	25
	Minimal	0
Does a sensitive AND specific screening test ALGORITHM currently exist?	YES	200
	NO	0
Test characteristics (Yes = apply score, No = zero)	Double in neonatal blood spots OR by a simple, in-nursery physical method	100
	High throughput (>2000/staff hr)	50
	Overall analytical cost <$ per test per condition	50
	Multiple analytes ordered to one condition are collected to same vial	50
	Other conditions identified by same analyte	50
	Multiple conditions detected by same test (multiplex platform)	200
Availability of treatment	Treatment exists and is widely available in most communities	50
	Treatment exists but accessibility is limited	25
	No treatment available or necessary	0
Cost of treatment	Inexpensive	50
	Expensive (>$50,000/child/year)	0
Potential efficacy of existing treatment	To prevent ALL negative consequences	200
	To prevent MOST negative consequences	100
	To prevent SOME negative consequences	50
	Treatment efficacy not proven	0
Benefits of early intervention (INDIVIDUAL OUTCOME)	Clear scientific evidence that early intervention leading to improved outcome	200
	Some scientific evidence that early intervention leading to improved outcome	100
	No scientific evidence that early intervention leading to improved outcome	0
Benefits of early identification (FAMILY & SOCIETY)	Early identification provides clear benefits to family and society (education, understanding, prevalence and natural history, cost-effectiveness)	100
	Early identification provides some benefits to family and society	50
	No evidence of benefits	0
Early diagnosis and treatment prevent mortality	YES	100
	NO	0
Availability of diagnostic confirmation	Wide	100
	Limited	50
	Poor	0
Acute management	Providers of acute management are widely available	100
	Limited availability of qualified providers of acute management	50
	Acute management is available only in a few centers	0
Simplicity of therapy	Management at the primary care or family level	200
	Requires periodic involvement of a specialist	100
	Requires regular involvement of a specialist	0
	Max score	2100

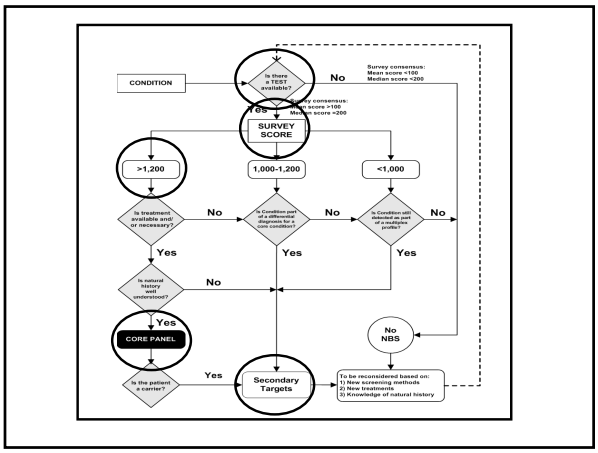
Respondent Profiles

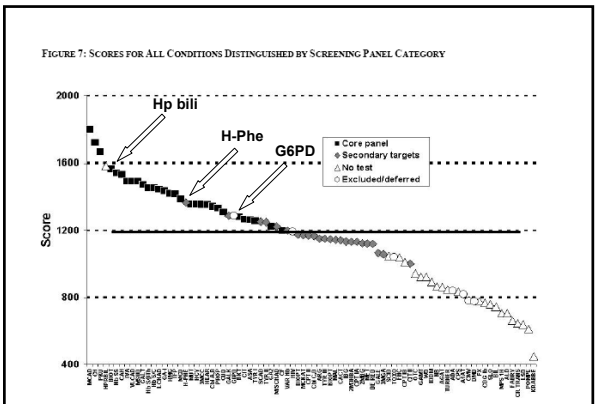


Top 10…

Table 4: Survey Scores of All Conditions (Sorted by Score in Descending Order)

Condition	Code	Responses	Missing data (%)	SCORE of the sum of the Means	Rank (%ile)
Medium-chain acyl-CoA dehydrogenase deficiency	MCAD	90	4	**1799**	1.00
Congenital hypothyroidism	CH	84	3	**1718**	0.99
Phenylketonuria	PKU	120	3	**1663**	0.98
Neonatal hyperbilirubinemia (Kernicterus)	HPRBIL	8	5	**1584**	0.96
Biotinidase deficiency	BIOT	68	2	**1566**	0.95
Sickle cell anemia (Hb SS disease)	Hb SS	55	8	**1542**	0.94
Congenital adrenal hyperplasia	CAH	93	7	**1533**	0.93
Isovaleric acidemia	IVA	53	3	**1493**	0.89
Very long-chain acyl-CoA dehydrogenase deficiency	VLCAD	58	2	**1493**	0.89
Maple syrup disease	MSUD	84	10	**1493**	0.89





Newborn Screening Panel: Core and Secondary Targets

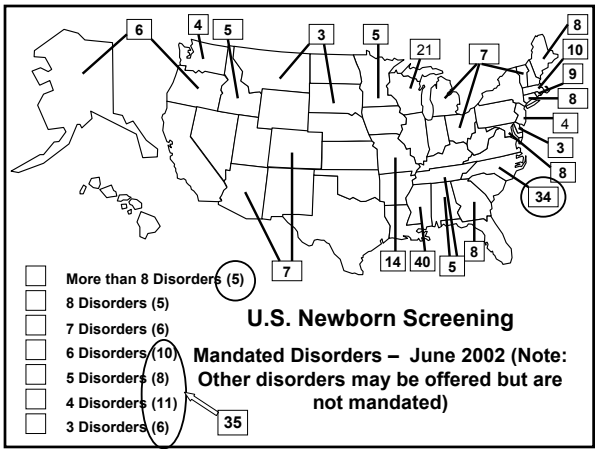
42 MS/MS

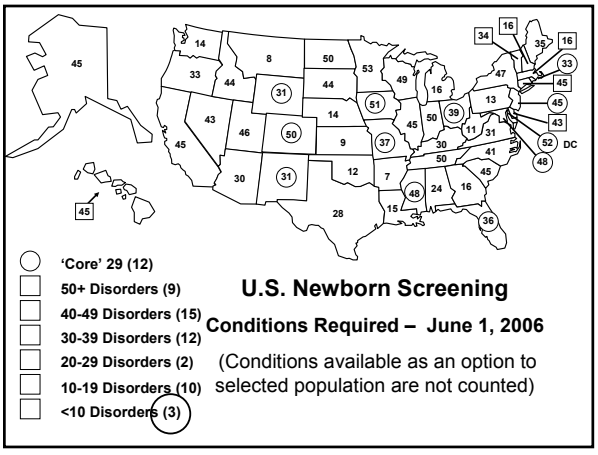
MS/MS				
Acylcarnitines		Amino Acids		
Core Panel				
9OA	**5FAO**	**6AA**	**3Hb Pathies**	**6Others**
IVA GA1 HMG MCD MUT 3MCC Cbl A, B PROP BKT	MCAD VLCAD LCHAD TFP CUD	PKU MSUD HCY CIT ASA TYR I	Hb SS Hb S/Bth Hb S/C	CH BIOT CAH GALT HEAR CF
Secondary Targets				
6OA	**8FAO**	**8AA**	**1Hb Pathies**	**2Others**
Cbl C,D MAL IBG 2M3HBA 2MBG 3MGA	SCAD GA2 M/SCHAD MCKAT CPTII CACT CPT1A DE RED	Hyper Phe TYR II BIOPT (BS) ARG TYR III BIOPT (REG) MET CIT II	Var Hb	GALK GALE

54 Total

Difficulties…

- Unknown natural history
- Low statistical power, poor evidence for improved outcomes based on screening (rare disorders)
- False positives/ false negatives
- Cost effectiveness (Burdon on states)
- “Medical Homes”



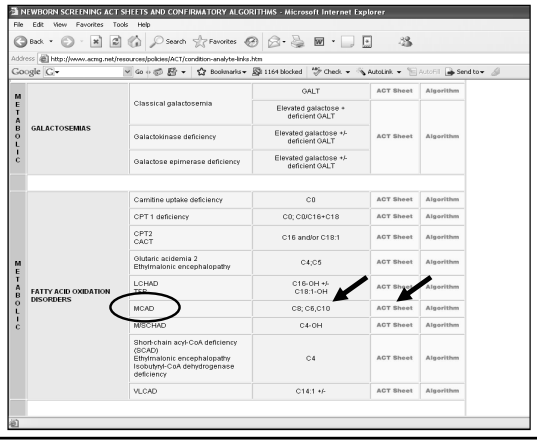


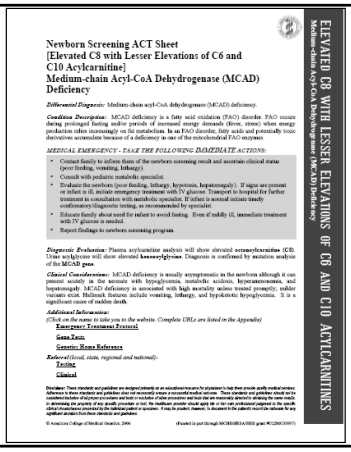
Newborn Screen follow up…

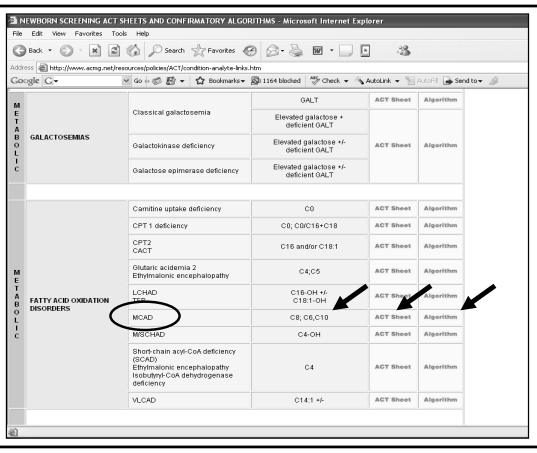
- What do I do with an abnormal result?

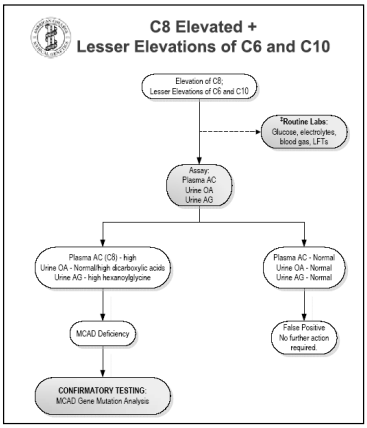
ACT (Action) **Sheets**

- Algorithms were developed by a work group that included experts in the various specialties and conditions involved in newborn screening for endocrine, hematological, genetic and metabolic diseases
  - Approved by the Board of Directors of the American College of Medical Genetics (ACMG)
  - Partial grant funding from Maternal and Child Health Bureau (MCHB) of the Health Resources and Services Administration (HRSA) of the U.S. Department of Health and Human Services
- http://genes-r-us.uthscsa.edu
  - *(Genetics and Newborn Screening Resource Center of the U.S.)*

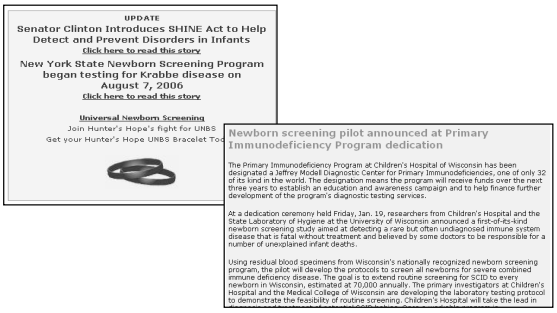








Future of Newborn Screening



UPDATE

Senator Clinton Introduces SHINE Act to Help Detect and Prevent Disorders in Infants

Click here to read this story

New York State Newborn Screening Program began testing for Krabbe disease on August 7, 2006

Click here to read this story

Universal Newborn Screening

Join Hunter's Hope's fight for UNBS

Get your Hunter's Hope UNBS Bracelet Tod

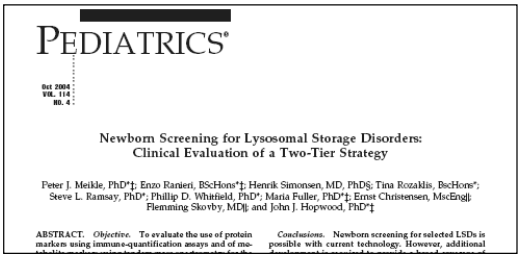
Newborn screening pilot announced at Primary Immunodeficiency Program dedication

The Primary Immunodeficiency Program at Children's Hospital of Wisconsin has been designated a Jeffrey Modell Diagnostic Center for Primary Immunodeficiencies, one of only 32 of its kind in the world. The designation means the program will receive funds over the next three years to establish an education and awareness campaign and to help finance further development of the program's diagnostic testing services.

At a dedication ceremony held Friday, Jan. 19, researchers from Children's Hospital and the State Laboratory of Hygiene at the University of Wisconsin announced a first-of-its-kind newborn screening study aimed at detecting a rare but often undiagnosed immune system disease that is fatal without treatment and believed by some doctors to be responsible for a number of unexplained infant deaths.

Using residual blood specimens from Wisconsin's nationally recognized newborn screening program, the pilot will develop the protocols to screen all newborns for severe combined immune deficiency disease. The goal is to extend routine screening for SCID to every newborn in Wisconsin, estimated at 70,000 annually. The primary investigators at Children's Hospital and the Medical College of Wisconsin are developing the laboratory testing protocol to demonstrate the feasibility of routine screening. Children's Hospital will take the lead in

Future of Newborn Screening



PEDIATRICS®

Oct 2004
VOL. 114
NO. 4

Newborn Screening for Lysosomal Storage Disorders: Clinical Evaluation of a Two-Tier Strategy

Peter J. Meikle, PhD*‡; Enzo Ranieri, BScHons*‡; Henrik Simonsen, MD, PhD§; Tina Rozaklis, BscHons*; Steve L. Ramsay, PhD*; Phillip D. Whitfield, PhD*; Maria Fuller, PhD*‡; Ernst Christensen, MscEng||; Flemming Skovby, MD||; and John J. Hopwood, PhD*‡

ABSTRACT. *Objective.* To evaluate the use of protein markers using immune-quantification assays and of me-

Conclusions. Newborn screening for selected LSDs is possible with current technology. However, additional

…in conclusion

- Newborn screening began in the 1960's
- LCMSMS in 1990's allowed us to screen for greater than 30 disorders
- In 2004 HRSA and ACMG developed a uniform panel of 29 core conditions and 25 report only
- Newborn screening at the discretion of the States
- Need to know what your state screens for and how to handle positive screen results
- New tests for new disorders being piloted (Krabbe, SCID, LSD)
- Multiple difficulties with expanded newborn screening
  - Unknown natural history, Low statistical power, poor evidence for improved outcomes based on screening (rare disorders), False positives/ false negatives, Cost effectiveness (Burdon on states), "Medical Homes"

Why we do it…

www.savebabies.org

http://genes-r-us.uthscsa.edu

(Genetics and Newborn Screening Resource Center of the U.S.)

Includes links and online tools for newborn screening and genetics programs including:

- Program links and testing summaries
- State newborn screening data accumulated annually

NOTES

American College of Osteopathic Pediatricians

TUESDAY, OCTOBER 2, 2007

3:00 pm - 4:00 pm

ACOP LECTURE
New Techniques for the Diagnosis of Genetic Diseases

Michael Kayser, DO

After this presentation the participant will be able to:

1. Identify the different areas of a practice that can be enhanced to improve efficiency and outcomes
2. Improve the reimbursement efficiency to maintain a thriving practice

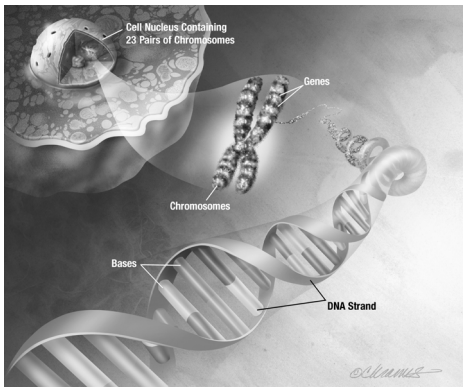
NOTES

New Techniques for the Diagnosis of Genetic Diseases

Michael A. Kayser, D.O.
Medical Director
Warren Clinic Center for Genetics
Center for Genetic Testing at Saint Francis

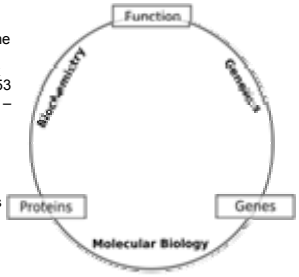
Objectives

- Introduction to three areas of genetic testing (Cytogenetic, Molecular, Biochemical)
- Current clinical applications for genetic testing in the three areas
- New techniques and available testing in the three areas

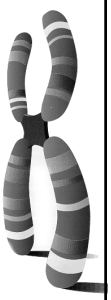


Field of Medical Genetics

- Modern science of genetics, began with the work of Gregor Mendel in the mid-1800s
- James D. Watson and Francis Crick resolved the structure of DNA in 1953
- Human Genome Project completion – April 2003
- Clinical Genetics
  - Prenatal, Adult, Pediatric, Metabolic Disorders
- Genetic Testing
  - 1. **Cytogenetics**- chromosomes
  - 2. **Molecular Genetics** - genes
  - 3. **Biochemical Genetics** – enzymes/metabolites

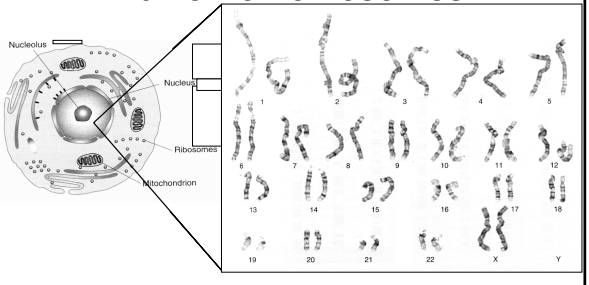


1. Cytogenetics



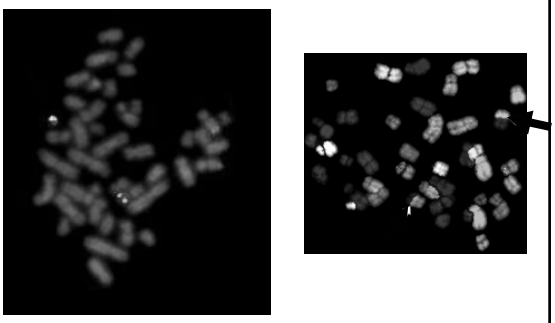
- Cytogenetics specializes in the study of cellular aspects of heredity, particularly chromosomes
- Modern cytogenetics began in 1956 with the discovery that normal human cells contain 46 chromosomes (Tjio and Levan)
- In 1959 Lejeune discovered patients with Down syndrome had an extra copy of chromosome 21
- In the late 1960's Caspersson developed banding techniques
- In the 1980's advances were made in molecular cytogenetics- *fluorescent in situ hybridization* (FISH)
- In the 1990's- *Comparative genomic hybridization* (CGH)- molecular-cytogenetic method for the analysis of copy number changes (gains /losses) in the DNA

Human chromosomes



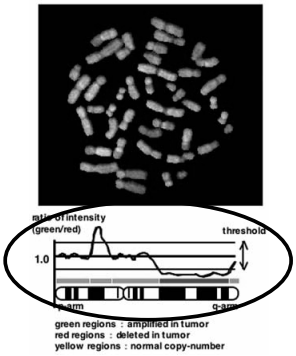
22 pairs of autosomes + 2 sex chromosomes (XX or XY)

FISH and SKY



Array Based Comparative Genomic Hybridization

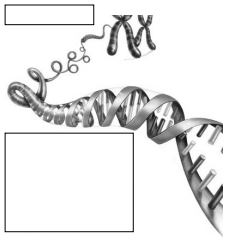
- *Goal:* to detect copy number alterations using a gene chip
- Ideally, the signal intensity is proportional to copy number



Alteration in DNA Copy Number: amplification and deletion

- Abnormal quantity of appearance of a genomic region in the genome.
  - Size: single gene - whole chromosome
  - Abnormality: deletion – amplification
- Some variations among normal individuals
- Can cause defects in human development
- Contributors to cancer
- Can effect function and gene expression

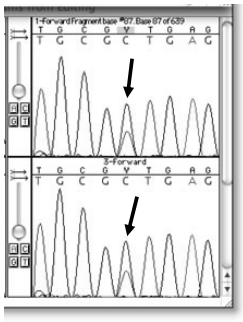
2. Molecular Genetics



- Molecular genetics deals with DNA sequence and gene expression
- The development of chain-termination DNA sequencing in 1977 (Sanger Sequencing)
- PCR method developed by Kary Banks Mullis in 1983
  - Allowed the isolation and amplification of arbitrary segments of DNA

Mutation

- Mutations (sequence variation)- change in the DNA base sequence
  - Offspring rarely get a perfect copy of the DNA from parents
  - mutations are rare: about 1 DNA base change per 10^7-10^9 bases each cell generation
- Some changes are much larger: chromosome rearrangements that include genes torn in half and moved to new locations, sometimes combined with other genes

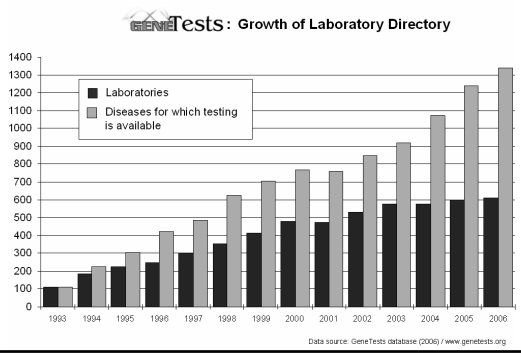


Information gained for Human Genome Project

(complete 2003)

- Only 25-30 thousand genes in human genome
- Gene-dense regions are predominantly composed of the DNA building blocks G and C
- Gene-poor areas are high in the DNA building blocks A and T.
- Chromosome 1 has the most genes (2968), and the Y chromosome has the fewest (231)
- Less than 2% of the genome codes for proteins
- Repeated sequences that do not code for proteins ("junk DNA") make up at least 50% of the human genome
- Identified about 3 million locations where single-base DNA differences (SNPs) occur in humans

Molecular testing lab and available tests



Medicine and the New Genetics

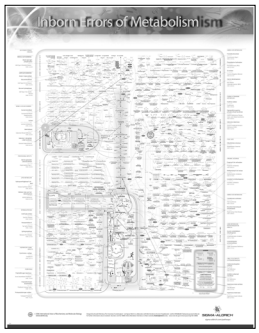
Anticipated Benefits:

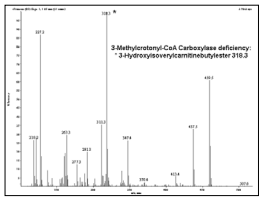
- improved diagnosis of disease
- earlier detection of genetic predispositions to disease
- rational drug design
- gene therapy and control systems for drugs
- personalized, custom drugs

U.S. Department of Energy Genome Programs, Genomics and Its Impact on Science and Society, 2003

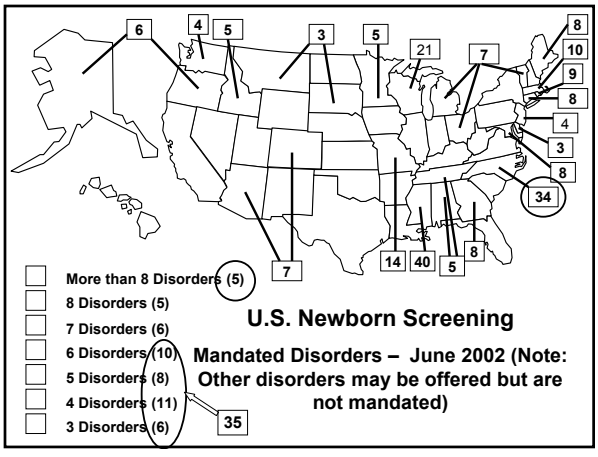
3. Biochemical Genetics

- Biochemical genetics is the study of the relationships between genes, enzymes, and metabolites
- **Inborn errors of metabolism** comprise a large class of genetic diseases involving disorders of metabolism. The majority are due to defects of single genes that code for enzymes that facilitate conversion of various substances (substrates) into others (products)





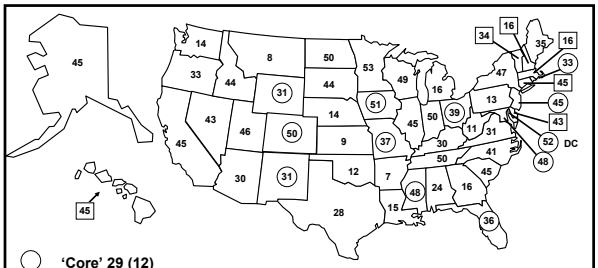
Biochemical genetic testing
Quantitative plasma amino acids, quantitative urine amino acids, urine organic acids by GCMS, newborn screening by LCMSMS



Newborn Screening Panel: Core and Secondary Targets

*Newborn screening (NBS) is a public health program that provides early identification and follow-up for treatment of infants affected by certain genetic, metabolic, hormonal and/or functional conditions
*>4,000,000 babies screened each year- largest use of genetic testing in the world

MS/MS				
Acylcarnitines		Amino Acids		
Core Panel				
9OA	5FAO	6AA	3Hb	6Others
IVA GA1 HMG MCD MUT 3MCC Cbl A, B PROP BKT	MCAD VLCAD LCHAD TFP CUD	PKU MSUD HCY CIT ASA TYR I	Hb SS Hb S/Bth Hb S/C	CH BIOT CAH GALT HEAR CF
Secondary Targets				
6OA	8FAO	8AA	1Hb Pathies	2Others
Cbl C,D MAL IBG 2M3HBA 2MBG 3MGA	SCAD GA2 M/SCHAD MCKAT CPTII CACT CPT1A DE RED	Hyper Phe TYR II BIOPT (BS) ARG TYR III BIOPT (REG) MET CIT II	Var Hb	GALK GALE 54 Total



U.S. Newborn Screening

Conditions Required – June 1, 2006

(Conditions available as an option to selected population are not counted)

ELSI: Ethical, Legal, and Social Issues

- **Privacy and confidentiality of genetic information**.
- **Fairness in the use of genetic information** by insurers, employers, courts, schools, adoption agencies, and the military, among others.
- **Psychological impact, stigmatization, and discrimination** due to an individual's genetic differences.
- **Reproductive issues** including adequate and informed consent and use of genetic information in reproductive decision making.
- **Clinical issues** including the education of doctors and other health-service providers, people identified with genetic conditions, and the general public about capabilities, limitations, and social risks; and implementation of standards and quality-control measures.

U.S. Department of Energy Genome Programs, Genomics and Its Impact on Science and Society, 2003

ELSI Issues (cont.)

- **Uncertainties associated with gene tests for susceptibilities and complex conditions** (e.g., heart disease, diabetes, and Alzheimer's disease).
- **Fairness in access to advanced genomic technologies**.
- **Conceptual and philosophical implications** regarding human responsibility, free will vs genetic determinism, and concepts of health and disease.
- **Health and environmental issues** concerning genetically modified (GM) foods and microbes.
- **Commercialization of products** including property rights (patents, copyrights, and trade secrets) and accessibility of data and materials.

U.S. Department of Energy Genome Programs, Genomics and Its Impact on Science and Society, 2003

Summary

- 1. Cytogenetic, 2. Molecular genetic, and 3. Biochemical genetic testing are important for further understanding of human disease and development of therapies
- Must keep in mind the impact of genetic testing on individuals and families

Questions?

American College of Osteopathic Pediatricians

TUESDAY, OCTOBER 2, 2007

4:00 pm - 5:00 pm

ACOP LECTURE
Current Concepts in Genetic Counseling

Michelle A. Fox, MS, BA, CGC

After this presentation the participant will be:

1. Understand the principles of genetic counseling, including shared decision making
2. Be aware of how genetic counseling and genetic testing impacts the pediatric patient and their family
3. Understand what geneticists and genetic counselors have to offer their patients and families

NOTES

American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

6:30 am - 5:00 pm	Registration	*San Diego Convention Center - 4 Foyer*
8:00 am - 9:00 am	**ACOP LECTURE** ***School Bullies*** Scott Elkin, DO	*San Diego Convention Center - 4*
9:00 am - 10:00 am	**ACOP LECTURE** ***Pain That Lasts Forever: Recognition, Diagnosis and Treatment of Post-traumatic Stress Disorder in Children and Adolescents*** Ava Stanczak, DO, FAAP	
10:00 am - 11:00 am	**ACOP LECTURE** ***What Happens in the Home after Tragedy in the Community?*** Felipe Amunategui, PhD	
11:00 am - 12:00 n	**ACOP LECTURE** ***Panel Discussion*** Ava C. Stanczak, DO, FAAP; Felipe Amunategui, PhD	
12:00 n - 1:00 pm	Lunch Break	
1:00 pm - 2:00 pm	**ACOP LECTURE** ***Pediatric OMT*** Shawn K. Centers, DO, FACOP	
2:00 pm - 3:00 pm	**ACOP LECTURE** ***Pediatric OMT Lab*** Shawn K. Centers, DO, FACOP	
3:00 pm - 4:00 pm	**ACOP LECTURE** ***Pediatric Palliative Care*** Paul G. Smith, DO	
4:00 pm - 5:00 pm	**ACOP LECTURE** ***Death and Dying Pediatric Issues*** Liz Sumner, RN, BSN	

NOTES

American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

8:00 am - 9:00 am

ACOP LECTURE
School Bullies

Scott Elkin, DO

After this presentation the participant will be able to:

1. Understand the nature and definition of bullying
2. Be aware of bullying's diverse expression... physical, verbal, sexual, relational and cyber
3. Understand bullying and the law
4. List the characteristics of bullies and their behaviors
5. Discuss what can be done about bullying as individuals, families, schools and communities

NOTES

The Bully and the Bullied

Scott R. Elkin, DO

I. Stated Objective for Lecture

II. What is it???

Bulling is about human relationships. It is about power and control. It is about those who are trying to establish power and control over others perceived to be weaker than themselves. It is urban, suburban and rural. It is American, European and Asian, a product of the Third World. It is imprinted in the Old Testament and can be found floating in cyberspace.

Cutting in subtlety, it can be overtly cruel in it's potential to harm.
On April 20, 1999, it became a focus of public discourse when 2 teen-age boys that had been relentently teased and bullied walked into Columbine High School outside of Denver and committed both mass murder and suicide.

The Bible references a response to bullying in Leviticus 19:16: Do not stand idly by the blood of your neighbor. Psalms 34:15: Distance yourself from evil, do good, seek peace and pursuit it.

What we are here to do today is to get a better understanding of bullying, it's impact, consequences and how to address it.

II. Bullying's Diverse Expression:
   a. Physical abuse
   b. Verbal abuse
   c. Sexual abuse
   d. Relational and emotional abuse
   e. Cyber abuse

III. Characteristics of Bullys and Their Behaviors:
   a. Who they are
   b. What they do
   c. How they do it and why
   d. What they hope to accomplish

IV: Bullying and the Law

V: Practical Steps to Combat Bullying

VI: Conclusion

NOTES

American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

9:00 am - 10:00 am

ACOP LECTURE

Pain That Lasts Forever: Recognition, Diagnosis and Treatment of Post-traumatic Stress Disorder in Children and Adolescents

Ava Stanczak, DO, FAAP

After this presentation the participant will be able to:

1. Name some of the early signs of post-traumatic stress disorder in young children
2. Explain some of the pathophysiologic changes in young people affected by post-traumatic stress disorder
3. List some of the important clinical findings that are diagnostic of post-traumatic stress disorder
4. Discuss some of the treatment modalities for this disorder in both young children and adolescents

NOTES

PAIN THAT LASTS FOREVER:

Recognition, Diagnosis and Treatment of Posttraumatic Stress Disorder in Children and Adolescents

Ava C. Stanczak, D.O.

GOALS AND OBJECTIVES

1. Name some of the early signs of posttraumatic stress disorder in young children.
2. Explain some of the pathophysiologic changes in young people affected by posttraumatic stress disorder.
3. List some of the important clinical findings that are diagnostic of posttraumatic stress disorder.
4. Discuss some of the treatment modalities for this disorder in both young children and adolescents.

DISCLOSURE

I HAVE NO RELEVANT FINANCIAL OR COMMERCIAL INTERESTS TO DISCLOSE

REFERENCES

"Posttraumatic Stress Disorder in Children" E Medicine, Roy Lubit, M.D., PhD

"Child Abuse and Neglect: Posttraumatic Stress Disorder" E Medicine, Sarah Guzofski, M.D.

Developmental and Behavioral Pediatrics, 2nd edition, Steven Parker, Barry Zuckerman, Marilyn Augustyn pages 268 270

DEFINITION

Posttraumatic stress disorder is a neurologic disorder that has significant impact on brain function.

It occurs after an individual experiences some type of traumatic event that causes or threatens serious harm or injury. The person does NOT have to be the victim to be affected.

This disorder can occur at ANY age, and it's effects may not be experienced immediately.

HISTORY OF THE DISORDER

The first mention of posttraumatic stress disorder (referred to hereafter as PTSD) was in the 1980 DSM III manual. It was described in adult populations, and no mention was made of children being affected.

Early studies in 1956 done on children who experienced horrific effects of a tornado in Mississippi, revealed the seriousness of their behavior after the event. A few sporadic studies were done in other situations later, but no data was collected. In 1987, the first mention of children was made in the DSM III.

PATHOPHYSIOLOGY

Recent limited evidence suggests that there is a genetic predisposition to PTSD, and that its presence may be linked to the temperament of the individual.

Alterations in noradrenergic and dopaminergic neurotransmitters and the stress response of the adrenals, has been documented in animals subjected to severe, frightening stressors.

Changes in brain microarchitecture are also thought to occur.

PATHOPHYSIOLOGY

If the brain is in a very formative stage, (very young children) the changes are thought to be much more severe and permanent. In older adults, subtle changes in architecture can cause much more damage as well.

Some of the research currently being conducted in Alzheimer's Disease, is carefully evaluating the effects of severe stress and the diagnosis of PTSD.

FREQUENCY AND PREDILECTION

Females have a higher incidence than males, but race does not seem to matter. PTSD seems to have a higher incidence in lower socioeconomic groups. Very young and old people are the most vulnerable to this disorder.

Very young children who are not able to process their environment, seem to have the largest amount of damage. These individuals are at a higher risk to develop PTSD if there is family trauma or violence present.

FREQUENCY AND PREDILECTION

Intelligence seems to protect individuals from PTSD, as persons with a higher I.Q. can more easily verbalize their experiences.

Risk factors for PTSD include the following:

- lack of social/parental support
- presence of a comorbid psychological disorder
- chronic , recurring trauma
- abuse by a trusted caretaker

FREQUENCY AND PREDILECTION

In the United States, due to limited data, the frequency is thought to be between 1 and 9%.

About 25% of children and adolescents have experienced a significant traumatic life event by age 16 years old. Of this group, 20% will develop PTSD.

Internationally, there is little or no data, but the disorder certainly exists, in countries such as Bosnia, Afghanistan, or Iraq.

CLINICAL FINDINGS

Early behavioral signs and symptoms that may appear long before diagnosis include:

- sleep difficulties that are new or different
- attention difficulties, new learning disorders
- aggressive or defiant behavior out of character for the child
- new or increased anxiety
- odd phobias
- new social avoidance
- depression with or without agitation

CLINICAL FINDINGS

Physical findings in PTSD are non-specific, and may be quite difficult to see in one office visit. Be aware that some of the findings may be due to a comorbid problem, or some predisposing factor.

Findings that have been noted are as follows:

- persons with PTSD have a smaller hippocampal volume
- basal cortisol levels are lower in these individuals
- glucocorticoid activity and metabolism is altered

CLINICAL FINDINGS

Some other physical findings that have been noted in animals include altered metabolism in the amygdala, and decreased activity in the anterior cingulate gyrus.

The physician may note that a child seems excessively frightened by touch. This is especially true in children that have been previously seen by the same physician, and had no problems.

CLINICAL SUBTYPES

Two subtypes of PTSD exist.

Type I is caused by one, unexpected traumatic incident, and is not ongoing.

Type II is caused by a chronic stressor after a primary event has occurred. This stressor is ongoing, and may go on for months to years.

FORMAL DIAGNOSIS

Symptoms must be present for at least one month, and a child or adolescent must have one from each of four categories. Adult criteria require at least three symptoms be present from each category.

Category A includes:

- the initial response
- horror
- feelings of helplessness
- disorganized behavior

Behavior in this category may be confused with an acute stress reaction.

FORMAL DIAGNOSIS

Category B is a cluster of intrusive recollections which may include some or all of the following:

- recurrent, frightening dreams and nightmares which may or may not be about the specific trauma
- intense distress at cues that symbolize the trauma
- feelings that the event is reoccurring
- flashbacks with no triggers

FORMAL DIAGNOSIS

Category C behavior includes numbing and withdrawal from social events. Some behaviors that occur include:

- efforts to avoid activities, places and persons that cause recollection of the trauma
- inability to recall important aspects of the event
- dissociation (may occur in Category B also)
- decreased interest in activities and skill regression (toileting in young children)
- restricted affect (can't accept or express warm feelings

FORMAL DIAGNOSIS

Category D behaviors are associated with persistent increased arousal. They can include the following:

- difficulty falling or staying asleep
- irritability with or without anger
- hypervigilance, increased startle reflex
- new fears
- new separation anxiety
- sleep issues may involve visual images

FORMAL DIAGNOSIS

Some older adolescents may report visual images during the day, which prevent them from concentrating at school. Occasionally these disturbing visions cause the child to lose interest in previous activities. Older children and adolescents rarely report these phenomena, as they fear they will be labeled as "crazy".

"Hearing voices" can also be associated with PTSD, but to a lesser degree. Children may hear sounds they heard at the time of the traumatic event.

FORMAL DIAGNOSIS

Diagnosing very young children with PTSD can be very challenging as they have difficulty expressing and explaining what they saw.

Some of the following can be good clues to raise suspicion as to the correct diagnosis:

- posttraumatic play that reenacts the significant event
- actually trying to reenact the event
- unexplainable nightmares
- flashbacks
- social withdrawal, complete or partial

PSYCHIATRIC TESTING

In older children (ages 6 and older) there are a few instruments that may be used in addition to symptoms to make the diagnosis of PTSD. In children without underlying comorbid diagnoses, these tests may be able to discern other underlying conditions such as a generalized anxiety disorder.

These tests should be administered by a professional skilled in testing, as they take a bit of time, and need good interpretation.

PSYCHIATRIC TESTING

Some of the more commonly used tests are as follows:

- CPTSDI Children's Posttraumatic Stress Disorder Inventory
- Beck Depression Inventory
- Trauma Symptom Checklist for Children
- Mississippi Scales for Combat-related Posttraumatic Stress Disorder

BEHAVIORAL TREATMENT

The first line of care for young people having PTSD is Cognitive Behavioral Therapy (CBT). Recently, and newer, more specific version of this therapy, Trauma-focused CBT has made a great impact on the care of children having PTSD. This form of therapy is effective in both Type I and Type II PTSD.

Trauma-focused therapy requires 10-18 sessions of one hour each, and focuses on stress management, and education about symptom prevention.

BEHAVIORAL THERAPY

- Trauma focused therapy requires that the child be able to understand instructions, and be able to participate in understanding their feelings and reactions. They also must be able to verbalize their "triggers", and be able to redirect their behavior in a positive way.
- This type of cognitive "reprocessing" can take a lot longer than 18 weeks, if a comorbid condition exists.
- Eye Movement Desensitization and Reprocessing (EMDR) is a newer type of therapy that may be combined with TF CBT.

PHARMACOLOGIC TREATMENT

- There are several medications that are being used to treat PTSD in children and adolescents in concert with any behavioral therapy. What has been noted carefully, is that in most cases, a combination of CBT and medication together, may work better than either alone.
- The following list is not in order of importance, but by drug class. IT IS IMPERATIVE THAT BEFORE MEDICATION IS BEGUN, APPROPRIATE LABS, INCLUDING AN ECG ARE COMPLETED. BE SURE ALL OTHER MEDICATION TAKEN IS DOCUMENTED.

PHARMACOLOGIC TREATMENT

There are several drug classes that are frequently used in treating PTSD. They are as follows:

- SSRIs
- beta adrenergic blockers
- alpha adrenergic agonists
- mood stabilizers
- atypical antipsychotics

PHARMACOLOGIC TREATMENT

Selective serotonin reuptake inhibitors (SSRIs)

- sertraline (Zoloft) approved for use in adult PTSD; not for children under 6 years old, dose for 6 12 years 25mg/day >12years 12.5mg/day should not exceed 200mg/day for any age
- fluoxetine (Prozac) < 8years, not approve, >8 10 mg/day…maximum dose 20mg/day
- paroxetine (Paxil) not approved for use in children, 5 10mg/day
- citalopram (Celexa) not approved for children, 5 10 mg/day

PHARMACOLOGIC THERAPY

Beta adrenergic blockers

- propranolol (Inderal) can use up to 2.5mg/kg/day divided three times a day

Alpha adrenergic blockers

- clonidine (Catapres) not approved for ANY psychiatric use in children. Can cause SUDDEN DEATH in combination with methylphenidate. Limited data suggest if must be used 0.15 03 mg/day divided three times a day
- guanfacine (Tenex) not for use in children <12 years old, no pediatric dosing, but can use 0.5 mg/day initially

PHARMACOLOGIC THERAPY

Mood stabilizers

- carbamazepine (Tegretol) 6 12 years 100mg/day divided two times a day; >12 years maintain plasma levels between 8 12mcg/ml
- valproic acid (Depakote) 10 5 mg/kg/day divided two or three times a day; try to achieve plasma levels of 50 125 mcg/ml MUST MONITOR LIVER FUNCTION, PLATELETS, WATCH FOR RASHES, AND ALTERED MENTAL STATUS

PHARMACOLOGIC THERAPY

Atypical antipsychotics such as quetiapine (Seroquel) and olanzapine (Zyprexia) and others are used after other medications have been given and failed after a reasonable trial. There are no specific doses for these medications, and none are approved for use in children under age 12 16 years.

Many of these medications will be used off label, and some children respond to these quite nicely. Children must be carefully monitored by the prescriber for serious side effects, since these medicines have not been studied in children.

PROGNOSIS

Individual outcome depends on the severity of the illness as well as the chronicity. Children who, due to their environment, must experience life threatening events on a regular basis, have a worse prognosis. Outcome is also based on the person's caregiver and their ability to understand and help with the child's treatment plan.

About 50% of persons recover in three months, but some go on to develop long term problems with a posttraumatic personality, which includes mood swings, substance abuse, rage and dissociation.

PROGNOSIS

In a longitudinal study of teens and young adults, only 52% of 14 24 year olds had a significant remission.

Symptoms may seem to be gone, and then return in the presence of a stressful or life changing event, that is not necessarily traumatic or harmful.

Prognosis is worse if the traumatic event is on going and chronic.

COMPLICATIONS

Some psychiatric conditions may develop as a result of PTSD. Suicidal ideation correlates well with the diagnosis of PTSD, and persons with a PTSD diagnosis have a higher mortality rate from suicide.

Other problems complicating this diagnosis are:

- anxiety and phobia (30%)
- major depression (40%)
- substance abuse (46% ETOH, 25% drugs)
- chronic fatigue, fibromyalgia, irritable bowel
- ADHD

EDUCATION

Children and adolescents should be encouraged to take part in their own therapy planning and treatment.

Young adults may not realize they are having a problem, and may be in denial that a problem ever existed. Part of therapy is to help the young person understand the reasons for therapy, and that they do not need to feel guilty about their feelings.

Parents and caregivers need to be educated that these problems are long term, and can be treated, but not completely cured.

FOLLOW UP

Well controlled data regarding outcomes in PTSD are currently lacking, but are being examined closely so prevention can be accomplished.

After initial diagnosis and therapy have begun, it is important for the patient and the caregivers to understand that regular follow up is important for the long term outcome. If a life- changing event such as a job change or move is planned, the patient should confer with their therapist, or go back into therapy until the event is successfully past.

THE FUTURE

- Research is currently being conducted in several large centers in the U.S., with observation over several years of persons using some of the therapies discussed today.
- Primary care physicians, especially pediatricians, must be aware of some of the subtle signs of PTSD, in order to make an early diagnosis, and have the child obtain treatment.
- Understanding the PTSD diagnosis may change a child's life and lessen the suffering.

NOTES

WEDNESDAY, OCTOBER 3, 2007

10:00 am - 11:00 am

ACOP LECTURE

What Happens in the Home after Tragedy in the Community?

Felipe Amunategui, PhD

NOTES

American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

11:00 am - 12:00 n

ACOP LECTURE
Panel Discussion

Ava C. Stanczak, DO, FAAP
Felipe Amunategui, PhD

NOTES

WEDNESDAY, OCTOBER 3, 2007

1:00 pm - 2:00 pm

2:00 pm - 3:00 pm

ACOP LECTURE
Pediatric OMT

Shawn K. Centers, DO, FACOP

NOTES

American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

3:00 pm - 4:00 pm

ACOP LECTURE
Pediatric Palliative Care

Paul G. Smith, DO

After this presentation the participant will be able to:

1. Appreciate the differences in causes of mortality and sources of suffering between children and adults
2. Understand the needs of children and their families during therapy for life-threatening illness
3. Construct an approach to relieving suffering in children and families during life-threatening illnesses.
4. Discuss national policies that determine resources available to children with chronic or life-threatening diseases.

NOTES

Pediatric Palliative Care - An Obligation

Paul G. Smith, DO

Advances in medical care have saved lives and improved the quality of life for an ever growing number of diseases and conditions. Nowhere is it more apparent than in children that such advances are a two-edged sword as an undesired consequence of medical interventions is suffering. Therapies have side effects, diagnostic tests are painful or unpleasant, and chronic illness is emotionally draining to individuals and their families. Previously non-treatable diseases are now treatable but sometimes the result is prolonging death instead of enhancing life. Many of the diseases are rare so there is uncertainty about disease course and consequences of interventions. Often prognostication is no more than a guess. These issues are magnified in children because life expectancy is greater and children lack the maturity to rationalize the benefits of therapy with the suffering. Because children are precious to us, the tendency is to subject children to more than we ourselves might choose even when any trace of hope is unrealistic. Hope should not be abandoned, but the medical community is obligated to assure that suffering is minimized. Though there is universal recognition that children are at increased risk for suffering with medical advances, there is also recognition that they have been seriously underserved.

Pediatric Palliative Care seeks to fill this obligation. Palliative therapy is defined by the goal of relieving suffering for any source without necessarily having a curative or treatment endpoint. Palliative care is not synonymous with end-of-life care and does not imply abandonment of hope though certainly suffering is greatest in end-of-life care. Palliative care should be a recognized need in whenever suffering can be avoided or treated, not only with life-threatening conditions or at the end of life. This session will stress the need for all Pediatricians to practice palliative care. Sources of suffering unique to children will be discussed as well as the tools with which we face these challenges. Future directions that will be discussed include areas where research is needed and national and global advocacy to improve or establish the financial and community resources necessary to meet children's needs.

After today's discussion the participant will be able to;

1. Appreciate the differences in causes of mortality and sources of suffering between children and adults
2. Understand the needs of children and their families during therapy for life-threatening illness
3. Construct an approach to relieving suffering in children and families during life-threatening illnesses.
4. Discuss national policies that determine resources available to children with chronic or life-threatening diseases.

References:

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American College of Osteopathic Pediatricians

WEDNESDAY, OCTOBER 3, 2007

4:00 pm - 5:00 pm

ACOP LECTURE
Death and Dying Pediatric Issues

Liz Sumner, RN, BSN

After this presentation the participant will be able to:

1. Describe current trends in pediatric palliative care
2. List ways to integrate palliative care principles into the setting they practice in
3. Verbalize examples of models for care in pediatric palliative care

NOTES

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I could put the deadlines here if you'd like. Or something else?

PLAN NOW TO ATTEND!

AMERICAN COLLEGE OF OSTEOPATHIC PEDIA

2008 Annual Spring Conference

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