Question 1: In the NICU, you’ve just examined a newborn with hypotonia and dysmorphic features that included epicanthal folds, upward slanting of the palpebral fissures, flat nasal bridge with anteverted nares and midface hypoplasia. Among the following, the medical complication that is MOST likely to present in the newborn period in this infant is:

1. atlantoaxial instability
2. hypothyroidism
3. leukemia
4. obstructive bowel disease
5. seizures

Question 1: Preferred Response: D

- The baby has features of Down syndrome. He should be observed for:
  - Obstructive GI tract abnormalities (~10%)
  - Duodenal atresia
  - Cardiac anomalies (~40%)
  - ECCD
  - Polycythemia and leukemoid reaction
  - Congenital hypothyroidism (~1%)
  - All patients with DS display cognitive impairment, (most with IQs in the mod MR range). Early intervention to maximize development in early childhood is recommended.
Question 1: Incorrect choices

- Atlantoaxial instability
  - Not in the newborn period
- Congenital hypothyroidism (~1%)
- Leukemia (~1%)
  - Not in the newborn period
- Seizures occur in a small number of patients with DS.

HE SHOULD ALSO HAVE CHROMOSOME TESTING:

Question 2:

You tell the infant’s parents of your concern that their baby has Down syndrome. After the initial shock, the mother asks about her risk of having another child with a chromosomal abnormality. The statement that you are MOST likely to include in your discussion is that her risk:

A. can be estimated by determination of maternal serum alpha-fetoprotein in all future pregnancies
B. cannot be estimated until her infant’s chromosome complement has been determined
C. is increased for Down syndrome, but not for any other chromosomal abnormality
D. is no greater than that of other women her age
E. is not increased until she reaches the age of 35

Karyotype

Trisomy 21 (95%; RR 1% for ANY TRISOMY)
- Unbalanced Robertsonian Translocation (2%)
- Mosaicism (2%)
- De novo (2/3)
- Inherited from balanced parent (1/3)
- Maternal (RR 10-15%)
- Paternal (RR 2-5%)
- 21:21 translocation (RR 100%)
Question 3:
As long as you’re already in the nursery, you're asked to see 3 infants born with cleft lip and/or palate. The first has a left CL+P; exam is otherwise normal. You tell his parents that this is apparently an isolated birth defect. During counseling about their risk for having another similarly affected child, the statement that you are MOST likely to include is that
A. because the defect is isolated, their risk is no greater than that of any other couple
B. Isolated cleft lip and palate is a multifactorial trait that has a 4% risk of recurrence
C. the recurrence risk can be estimated only after a chromosome analysis has been obtained
D. the risk is increased for future females, but not males
E. the risk would be increased only if one of the parents had cleft lip and palate

Question 3: Preferred Response: B
- Multifactorial (MF) traits:
  - interplay of genetic and environmental factors
  - RR for a family in which neither parent is affected and only one child is affected is 2-5%.
- MF inheritance is seen in many common birth defects
- MF inheritance is also the cause of most disease of childhood and adulthood

Question 3: Preferred Response: B
- RR of MF traits increases if more family members affected
  - Eg. 3% if one child affected; 10-15% if one child and one parent affected
- Must first R/O genetic syndrome before quoting RR
- Chromosome analysis should be obtained in pts with two or more major abnormalities (eg, clefting, congenital heart disease) or one major anomaly and two or three minor defects (eg, epicanthal folds, simian line).
Question 4

The next baby you see in the nursery, a boy, has bilateral CL+P. Further exam reveals scalp defect in the parietooccipital region, microphthalmia, cryptorchidism, a cardiac murmur and polydactyly.

Of the following, the MOST likely underlying condition is:
A. holoprosencephaly sequence
B. retinoic acid embryopathy
C. trisomy 13
D. trisomy 18
E. valproate embryopathy

Question 4: Preferred Response: C

- Baby has a multiple malformation syndrome
- Trisomy 13 (47,XX or Y,+13)
  - 1 in 5-10,000 liveborns
  - 5% survive to 6 months
  - Advanced maternal age
- Holoprosencephaly, severe MR; microcephaly; microphthalmia; coloboma; CL+/−P; ear anomalies; distinctive scalp defects in the occipital area; CHD; postaxial polydactyly and cryptorchidism in males
- Peripheral blood karyotype
  - Diagnosis
  - Recurrence risk

Question 4: Incorrect responses

- Fetal valproate syndrome results in CHD, NTDs and unusual facial features (narrow bifrontal diameter, high forehead and epicanthi) as well as limb defects.
- Retinoic acid embryopathy results in facial asymmetry, microtia or anotia, & CHD.
- Trisomy 18 is characterized by growth deficiency, prominent occiput, low-set ears, micrognathia, clenched hand, and structural defects of the heart and kidney. Microphthalmia and clefting are not prominent features.
Question 5:
The next baby you visit in the nursery has a CP and retrognathia, but no other anomalies.
Of the following, the MOST serious complication that may occur in the first 72 hours of this child's life is:
A. acute otitis media requiring antibiotic treatment
B. congestive heart failure
C. difficulties with breastfeeding
D. upper airway obstruction with oxygen desaturation
E. worsening airway obstruction when placed in the prone position

Question 5: Preferred Response: D
The Pierre-Robin malformation sequence (PRMS)
(1) micrognathia
(2) glossoptosis
(3) a U-shaped cleft of the palate

- PRMS may occur in isolation or as part of a syndrome
  - Stickler, 22q11 deletion and CHARGE

- Obstructive apnea with desaturation is by far the most serious complication that may develop within the first days of life. Such obstruction may be severe enough to cause ischemic encephalopathy.

Question 5: Incorrect responses
- Feeding difficulties are common but oral feeding, including breastfeeding, is successful.
- OM is quite common among children who have cleft palate, it typically does not cause problems in early infancy.
- PRMS is a medical emergency in the newborn
  - Prone position
  - Definitive treatment is essential to assure that the airway remains open
    - nasopharyngeal tube, surgical management
Question 6:

As long as you're already in the nursery, the resident asks you to see a newborn who has some unusual features. Born by C/S because of breech presentation, the baby has a deformed cranium, torticollis, facial asymmetry, a dislocated right hip, and bilateral clubfeet. Findings on the remainder of the physical examination are normal.

Of the following, the MOST likely cause of this infant's abnormalities is

A. a chromosomal abnormality
B. a malformation syndrome
C. an underlying CNS defect
D. exposure to a teratogen in utero
E. intrauterine compression

Question 6: Preferred Response: E

- Malformations
  - alterations in the tissue primordia that forms a structure
  - first trimester
  - mild to severe
  - usually require surgical correction
  - ~3% of newborns

- Deformations
  - abnormal external forces acting on normally formed tissue
  - after the first trimester
  - frequently mild
  - usually improve spontaneously
  - 1 to 2% of neonates have one or more deformations.

Question 6: Preferred Response: E

- Deformation sequence due to intrauterine compression
  - restriction of fetal movement
  - 1/3 of all deformations occur in infants who present in the breech position

- A chromosomal abnormality or teratogen exposure associated with the multiple defects described for this infant would be expected to cause malformations of internal organ systems as well as external findings
Question 7: Preferred Response: E

- Neural tube closes by 27th postconceptional day
- Closure proceeds from mid-cervical area caudally and rostrally
- NTD cause increased MS AFP and AF AFP
  - Non-specific; confirm with sonography (most sensitive)
  - Fetal trisomies cause decreased AFP levels
- Isolated or syndromic
  - Isolated NTDs exhibit MF inheritance
- Before 1990, NTDs occurred in 1 in 1,000 births
- Periconceptual folic acid supplementation can decrease the risk of having a child with NTD by 70%

Question 7: Incorrect Responses

- RR does not depend significantly upon the ethnic group
- Location of the defect along the neural axis does not influence RR
- If the NTD is part of a syndrome, the specific RR for the syndrome should be discussed
Question 8: In genetics clinic, you're seeing a 5 y.o. girl who has multiple café au lait spots and axillary freckling. Slitlamp ophthalmic exam reveals the presence of Lisch nodules, confirming the diagnosis of neurofibromatosis.

Of the following, the MOST appropriate statement about the potential for the development of tumors in this child is that:

1. acoustic neuromas are common and annual screening with MRI is indicated
2. all patients with neurofibromatosis eventually develop plexiform neurofibromas
3. annual urinary catecholamine screening for the presence of pheochromocytoma is indicated
4. cutaneous neurofibromas usually do not appear until preadolescence
5. the overall lifetime risk for the development of a malignancy is 50%

Question 8: Preferred Response: D

This child has features of NF-1. The diagnosis of NF-1 is made on the basis of having two of the following 7 criteria:

- Café au lait spots: 6 or more of >0.5 cm in diameter for prepubertal children or >1.5 cm post pubertally
- Axillary and/or inguinal freckles
- Neurofibromas: 2 or more, or 1 plexiform
- Optic glioma
- Lisch nodules
- Skeletal abnormalities, including scoliosis, pseudarthrosis, sphenoid wing dysplasia
- Family history of a 1st degree relative with NF-1, diagnosed using these criteria.

Question 8: Preferred Response: D

NF-1 is an autosomal dominant disorder and one of the most common single-gene defects.

- The gene, NF-1, which is on chromosome 17, produces neurofibronin, an inhibitor of nerve growth factor.
- Huge gene, no "common mutations", frequently mutated
- 50% spontaneous
**Question 8:** Incorrect responses

- Acoustic neuromas are a prominent feature of NF-2
  - bilateral in ~ 85% of patients and unilateral in ~6%
  - cutaneous findings are not as prominent
- The lifetime risk for the development of a malignancy in patients who have NF-1 is ~5% (NOT 50%)
  - Plexiform NFs ~5% can undergo sarcomatous change
  - CNS tumors, including optic gliomas, occur in 2-3% of affected individuals
- Pheochromocytoma is a rare tumor in NF-1 (<1% of patients)
  - common in patients who have von Hippel-Lindau disease

**Question 9:**

The next patient in clinic is the mother of a 13-mo-old boy recently diagnosed with factor VIII deficiency hemophilia. The woman is in the 1st trimester of her 2nd pregnancy, and is interested in knowing if prenatal diagnosis is available.

Of the following, the statement about prenatal diagnosis that you are MOST likely to include in your discussion is that:

A. factors VIII and IX deficiency hemophilia can be dx'd prenatally in over 95% of families
B. factors VIII and IX deficiency hemophilia can be dx'd prenatally only in 20% of families
C. neither type of hemophilia can be dx'd prenatally
D. only factor IX deficiency hemophilia can be dx'd prenatally
E. only factor VIII def. hemophilia can be dx'd prenatally

**Question 9: Preferred Response: A**

- Prenatal diagnosis for Factor XIII and Factor IX hemophilia in > 95% of families
  - Genes located near the terminus of Xq
  - Molecular diagnosis of mutations
- Affects ~1 in 5,000 individuals
  - 80-85% of patients having factor VIII deficiency hemophilia (hemophilia A)
  - 10-15% having factor IX deficiency (hemophilia B)
- Neither disorder has apparent racial or ethnic predilection
**Question 9:** Preferred Response: A

- The factor VIII gene is much larger, more complex, and has a strong predisposition to spontaneous mutation:
  - ~1/3 have new mutations
  - More than 200 different mutations
  - Common mutation: gene inversion (45% of severe cases)
- The factor IX gene is considerably smaller and less complex:
  - Over 400 mutations have been identified.
  - DNA sequencing used for dx, carriers, and prenatal dx
  - Very low rate of spontaneous mutation, so the family history almost always is positive.

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**It's lunch time in Clinic. Time for some quickies:**

**Question 10:**

Of the following, the findings that are MOST suggestive of the dx of Prader-Willi syndrome are:

1. gigantism and visceromegaly
2. hyperactivity and ataxic movements
3. hypocalcemia and a congenital heart defect
4. MR and macroorchidism
5. obesity and small hands and feet

**Question 10: The answer is E**

- A describes Beckwith Wiedemann syndrome
- B describes Angelman syndrome
- C describes diGeorge (aka velocardiofacial, Shprintzen, or 22q11.2 deletion syndrome)
- D describes Fragile X syndrome
- Children with PWS have a characteristic history:
  - Hypotonia in infancy, with poor sucking and FTT
  - Improvement in muscle tone by age 1 year
  - Development of a voracious, insatiable appetite
  - Development of obesity and related problems
PWS results from deficiency of the gene product of the SNRPN gene. SNRPN (15q11) is only expressed in the chromosome 15 inherited from the father. Thus, PWS results when a copy of paternal chromosome 15 is missing. This can be caused by:

- Deletion of pat 15q11.2 (seen in 60-70%): Use FISH to diagnose
- Maternal uniparental disomy of chromosome 15 (yielding two copies of maternal chromosome 15 and no copies of paternal chromosome 15) (seen in 20%)

Another quickie: Question 11:
All of the following are associated with Williams syndrome EXCEPT

A. “Cocktail party” personality
B. Hypercalcemia
C. Supravalvular aortic stenosis
D. Short philtrum
E. Mental retardation

Question 11: The answer is D

- WS is a contiguous gene syndrome caused by deletion of 7q11.2 (use FISH or array CGH to make diagnosis)
- Occurs in 1 in 5,000 births
- Features include all those listed in question except for short philtrum (which is seen in fetal alcohol syndrome).
- Heart disease occurs in 80%, with SVAS in 67%
A third quickie:

Question 12:
Noonan syndrome is characterized by each of the following EXCEPT

A. Autosomal recessive inheritance
B. Short stature
C. Pulmonic stenosis
D. Webbed neck
E. Low set ears

Question 12: The answer is A

- Noonan syndrome is caused by a mutation in the \textit{PTPN11} gene on chromosome 12.
- Occurs in 1 in 10,000 births
- Features include all those listed in question except for AR inheritance
- 50% also have Factor 11 deficiency causing prolonged PTT.

Yet another:

Question 13:
A child is born with esophageal atresia. Other associated abnormalities might include all of the following EXCEPT

A. Cardiac defect such as VSD
B. Absence of the left radius
C. Renal malformations
D. Hemivertebra
E. Cataracts
Question 13: The answer is E

- Esophageal atresia, occurring alone or as part of a TEF, can be isolated or part of a syndrome or association. The most common association is VACTERL.
- Association: a group of malformations that occur more commonly together than would be expected by chance, but for which no etiology can be identified.
- Features of VACTERL include:
  - Vertebral anomalies
  - Anal anomalies (imperforate, stenotic, etc)
  - Cardiac anomalies (VSD, ASD most common)
  - Tracheo-Esophageal Fistula
  - Renal anomalies
  - Limb anomalies (specifically radial ray defects)

Last quick one:

Question 14:
Which of the following is typical of Ehlers Danlos syndrome?

- A. Osteoporosis
- B. Hypotrichosis
- C. Large head and short extremities
- D. Joint laxity
- E. Progressive neurologic and ophthalmologic problems

Question 14: The answer is D

- Osteoporosis is seen in osteogenesis imperfecta.
- Hypotrichosis occurs in anhidrotic ectodermal dysplasia.
- Large head and short extremities describes achondroplasia.
- Ehlers Danlos is a disorder of collagen that leads to:
  - Joint laxity
  - Spontaneous joint dislocation
  - Hyperelastic skin
  - Poor scar formation (“cigarette paper” scars)
  - Easy bruising
Okay, really last quick one:

**Question 15:**
For which of the following disorders would you use PCR testing for trinucleotide repeats to make the diagnosis?

A. Crouzon syndrome  
B. Fragile X syndrome  
C. Cri-du-chat syndrome  
D. MELAS syndrome  
E. Down syndrome

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**Question 15: Preferred Response:** B

- **Crouzon syndrome** is a craniosynostosis syndrome, usually diagnosed clinically. This is a single gene disorder, with autosomal dominant inheritance, and DNA testing is available for mutations in FGFR-2.
- **Fragile X syndrome** is a trinucleotide repeat disorder, with an excess number of CGG repeats leading to symptoms including intellectual disability and characteristic facial features.
- **Cri-du-chat syndrome** is a continuous gene disorder, diagnosed by FISH or array comparative genomic hybridization (CGH).
- **MELAS syndrome** is a mitochondrial disorder, diagnosed by testing of specific mitochondrial DNA genes.
- **Down syndrome** is an aneuploidy syndrome diagnosed by karyotype or FISH, also detectable by array CGH.

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**OKAY, LUNCH IS OVER**

**TIME TO GO TO METABOLIC CLINIC:**
**Question 16: Patient in Metabolic Clinic:**

The parents of a 2 y.o. boy with Tay-Sachs disease (TSD) (hexosaminidase A deficiency) ask you about the availability of prenatal testing during their next pregnancy.

Of the following, the MOST appropriate statement to include in your counseling is that:

A. prenatal dx should be considered only after testing each parent to determine carrier status
B. testing can be performed on chorionic villus cells obtained as early as 10 weeks of pregnancy
C. testing is possible only if the parents' mutations in the hexosaminidase gene are known
D. the results of prenatal diagnostic testing for TSD are considered investigational
E. their risk of having another similarly affected child is no greater than that of any other couple

**Question 16: Preferred Response: B**

- AR inherited inborn error of metabolism
  - Parents of an affected child are obligate carriers (RR of 25%)
- Absence of HexA, a lysosomal enzyme
- HexA activity can be measured in fetal cells obtained by CVS, performed as early as 10 weeks gestation, or via amnio (14-16 weeks)
- Carrier frequency for TSD is highest in Ashkenazi Jews (~1:25) and French Canadians
  - 1970's - screening programs to identify carriers (measurem of hex activity in serum or isolated leukocytes),
  - Virtually eradicated the disease among this population
  - In the past 10 years most infants dx'd with TSD have been non-Jewish or have had one Jewish and one non-Jewish parent. Thus, screening of couples in whom one member is Ashkenazi Jewish is recommended.

**Question 16: Preferred Response: B**

- Deficiency of HexA activity results in accumulation of GM2 gangliosides in the CNS, liver, and spleen
- Normal for a 6 to 9 months
- Present with hypotonia, apathy, developmental delay, and an exaggerated startle response
- Cherry red spot in the maculae (not pathognomonic)
- Macrocephaly is common after age 1
- Rapid progression; death by 5 yo
- Known gene
  - Common mutations in Ashkenazim
  - Many other mutations known
Question 17: Next Pt. in Metab Clinic:
You’re seeing a 3 y.o. with Hunter syndrome (MPS-II) for F/U. His parents tell you the boy’s newborn brother has just been dx’d with this same disorder, and they’ve heard that bone marrow transplantation can “cure” the baby of his condition.

Of the following, the statement you are MOST likely to include in a discussion of transplantation for Hunter syndrome and other inborn errors of metabolism is that
A. family members who are carriers of the disorder cannot be donors
B. infections are less common than in patients who have a hematologic disorder
C. it is contraindicated in disorders that include adverse neurologic symptoms
D. it never has been successful in the treatment of enzyme deficiency disorders
E. successful transplantation may halt disease progression for some disorders

Question 17: Preferred Response: E
- Organ & bone marrow transplantation (BMT) may halt progression of some metabolic disorders
- Used more b/c better immunosuppressants & no other effective treatment strategies
- Each case must be considered individually
- Sometimes, BMT is undertaken to provide a source of enzyme that’s not produced in sufficient quantities
- Sometimes, organ tx replaces an organ that’s been damaged by the disease process

Question 17: Incorrect responses
- BMT in pts with inherited metabolic disorders carries the same risks as when performed for other indications
- Identifying a histocompatible donor frequently requires that a donor from within the family be considered.
- Carriers are acceptable donors
- Controversy whether patients with metabolic disorders that include neurologic symptoms should be candidates
Question 18: Next Pt. in Metab Clinic:
You are called down to the ED to see a previously healthy 2 y.o. boy who was brought to the ED by his mother, who reported that he has had a cold and fever for the past 2 days. He has been taking only small amounts of juice and no solid foods. When she tried to arouse him after his nap today, he was lethargic and unresponsive. Results of laboratory studies include a glucose concentration of 40 mg/dL, an ammonia level of 200 mcg/dL, and an arterial blood pH of 7.4. At this time, the MOST important study to obtain is

1. plasma acylcarnitine profile
2. plasma insulin levels
3. serum acetylsalicylic acid concentration
4. urine and stool porphyrins
5. urine organic acids

Question 18: Preferred Response: A

- Fatty acid oxidation defects
  - Defects in mitochondrial beta-oxidation of fatty acids (MBOFA)
  - Autosomal recessive
- MCAD (medium-chain acyl CoA dehydrogenase deficiency) is most common
  - Presents by age 2 with lethargy after fasting, typically associated with a URI or AGE
  - Hypoketotic hypoglycemia and hyperammonemia
  - Dx by plasma acylcarnitine profile
- MBOFA for the production of energy is only required during periods of fasting, clinical manifestations do not become apparent unless substantial fasting has occurred. Thus, there can be great heterogeneity in clinical course.

Question 18: Preferred Response: A

- Between episodes of illness, affected pts are normal.
  - 1st episode may result in death and is frequently mis-dx’d as Reye syndrome or SIDS
- Treatment includes avoidance of fasting, carnitine supplementation, and dextrose during acute episodes.
- Molecular diagnosis of MCAD is available
  - > 90% point mutation in the MCAD gene
Question 18: Incorrect responses

- An organic acidemia should be suspected in pts presenting with hypoglycemia and hyperammonemia in the presence of metabolic acidosis.
- Plasma insulin levels should be obtained in a child suspected of having hyperinsulinemia, which may cause hypoglycemia but usually is not associated with hyperammonemia.
- Urine and stool porphyrin analysis is useful in the diagnosis of the porphyrias – different sx’s (abdominal pain, vomiting, constipation)
- Acetylsalicylic acid poisoning usually presents with hyperventilation and dehydration, but hyperammonemia is not typical.

Question 19: Call from the ED

You receive an emergent call from the ED. A baby boy, DOL #4, is lethargic, with history of irritability and poor feeding for 2 days. They are concerned about a metabolic disorder. The parent has also noted some abnormal movements, that look like the baby is fencing, or bicycling. Newborn screen results are not available, but laboratory studies reveal metabolic acidosis with anion gap, normal ammonia level, and positive urinary ketones. Which do you expect to find on your examination?

A. Cataracts
B. Hepatomegaly
C. Maple syrup odor of the cerumen
D. Eczematous skin rash
E. Overriding sutures

Question 19: Preferred response: C

MSUD

- Amino Acid disorder due to a defect in the Branched Chain Ketoacid Dehydrogenase (BCKD) enzyme
- AR disorder; 3 genes BCKDH A, BCKDH B, DBT
- Symptoms 24-48 hrs of age: poor feeding, lethargy, irritability, maple syrup odor of urine and cerumen
- Progression to athetoid movements, spasticity, opisthotonus, seizures, coma
- Diagnosis: newborn screen, DNPH urine dipstick, plasma amino acids, confirmatory molecular genetic testing
- Treatment: protein-restrictive diet (especially leucine); IV hydration in crisis; some liver transplant
Question 19: Incorrect responses

- **Cataracts** can be seen in untreated galactosemia
- **Hepatomegaly** can be seen in urea cycle disorders such as citrullinemia, or also in galactosemia
- **Eczematous skin rash** can be seen in PKU
- **Overriding sutures** – common after birth, can indicate craniosynostosis, not associated with metabolic disorders of infancy

Question 20: Match each metabolic disorder with its characteristic feature

| A. Galactosemia | 1. X-linked disorder |
| B. Maple syrup urine disease (MSUD) | 2. Musty body odor |
| C. Phenylketonuria (PKU) | 3. Treated with lactose-free diet |
| D. Ornithine transcarbamylase deficiency (OTC) | 4. Missing enzyme to break down branched chain amino acids |

Question 20: Correct matches

| A. Galactosemia | 1. X-linked disorder |
| B. Maple syrup urine disease (MSUD) | 2. Musty body odor |
| C. Phenylketonuria (PKU) | 3. Treated with lactose-free diet |
| D. Ornithine transcarbamylase deficiency (OTC) | 4. Missing enzyme to break down branched chain amino acids |
Question 20 summary

- **Galactosemia**: disorder of carbohydrate metabolism; GALT deficiency; AR; sx’s: days to weeks of life (vomiting, jaundice, hepatomegaly, lethargy, bacterial infection, cataracts); dx: newborn screen, molecular; tx: lactose-free diet

- **PKU**: amino acid disorder, Phenylalanine hydroxylase deficiency; AR; sx’s: poor feeding, lethargy, light eyes/skin/hair, musty odor, eczematous like rash, untreated severe ID; dx: newborn screen; tx: low Phe diet

- **OTC**: urea cycle DO, X-linked; sx’s: 24 hrs few days, vomiting, poor feeding, lethargy, sz, hepatomegaly; dx: NOT newborn screen, ammonia, UOA, PAA, genetic testing; tx: low protein diet, alternate pathway meds to get rid of nitrogen, arginine

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GENETICS CLINIC IS OVER.

BEST OF LUCK ON THE BOARDS!