Normal or not: the first 6 weeks

Pharmac seminars 2016



Children's Hospital & Community Health Tamariki Ma



The New Zealand Herald

Sunday 25th March 2012

Wednesday 28th March 2012

Baby dies after scar ruptures

Family's caesarean baby tragedy









Resuscitation (briefly)

Topics

■ Jaundice

Congenital Heart Disease

■ Hypoglycaemia

Quick small topics

GBS and other infections

Latest NZ birth stats

2008 there were <u>64,340</u> live births registered in New Zealand,
 * highest number since 1972

65,390 – 1961 = Peak (NZ pop. 2.5million 1961) 54,020 – 2002 = Low point $62,540 - 2009 (\downarrow 3\%)$ $63,900 - 2010 (\uparrow 2\%)$ $61,403 - 2011 (\downarrow 1\%)$ $57,242 - 2014 (\downarrow 2.5\% 2013)$ $61,038 - 2015 (\uparrow 7.0\% 2014)$

New Zealand women averaged 1.99 births each (4.3 in 1961)
 (2.34 Maori esp Nthld)

■ Births over death 29,430: Just sustains population without immigration (60K)

Age-specific fertility rates 1962–2014



Age-specific fertility rates 1962–2015



Source: Statistics New Zealand

NZ Stats 2016

- Median age <u>all</u> women giving birth was 30 years
- **20 24 yrs** < 30 39 years =**62/1000**(1962: 20-24 yrs = 256/1000)
- Highest fertility age group 30 34 years 125 / 1000 women
- Stable now 10 years
- Median age <u>first</u> child 28!!
- 2000 multiples (1000 1977)

- 5 years older than 1977.
- Also Stable 10 years
- 66% mothers > 30 years

The risks associated with pregnancy in women aged 35 years or older

Table IV. Delivery complications				Table V. Fetal complications			
	Age group (years)	Proportion (%)	Odds ratio (99% confidence interval)	-	Age group (years)	Proportion (%)	Odds ratio (99% confidence interval)
				Delivery after 42 weeks gestation ^{b,d}	18-34	0.16	
Induction of labour ^{b,c,f}	18-34	16.88			35-40	0.16	1.14 (0.80-1.61)
	35-40	16.88	1.04 (1.00-1.08)	Delivery before 37 weeks gestation ^{b,c,d,f}	>40	0.19	1.19 (0.57-2.50)
	>40	19.22	1.19 (1.10-1.29)		18-34	6.0	
Breech delivery ^{D,1}	18-34	0.78			35-40	6.63	1.18 (1.11–1.25)
	35-40	0.71	0.92(0.78 - 1.08)	Delivery before 32 weeks gestation ^{b,c,d,f}	>40	8.17	1.42 (1.26–1.60)
	>40	0.65	0.83 (0.56-1.22)		18-34	1.03	1 41 (1 24 1 (1)
Operative veginal delivery	18 34	11.26	0.05 (0.50-1.22)		35-40	1.55	1.41(1.24-1.61)
Emergency Caesarean section ^{a,b,c,d,e,f}	25 40	10.82	1 50 (1 42 1 57)	Stillbirth b.c.f.j	18_34	0.47	
	55-40	10.05	1.50(1.43-1.57)	Sunonui	35-40	0.61	1 41 (1 17-1 70)
	240	10.25	1.00 (1.43–1.78)		>40	0.81	1.83 (1.29–2.61)
	18-34	8.05		Delta birthweight <5th centile ^{b,c,d,f}	18-34	5.81	
	35-40	11.05	1.59 (1.52–1.67)		35-40	6.13	1.28 (1.20-1.36)
	>40	14.24	2.17 (1.97-2.39)		>40	7.63	1.49 (1.29–1.71)
Elective Caesarean section ^{a,b,c,d,e,I}	18-34	4.37		Delta birthweight >90th centile ^{b,d}	18-34	10.06	
	35-40	8.6	1.77 (1.68–1.87)		35-40	12.32	1.20 (1.13-1.27)
	>40	12.67	2.67 (2.42-2.95)		>40	11.96	1.29 (1.14–1.45)
Postpartum haemorrhage ^{b,f,g,h}	18-34	11.24		Apgar score <7 ^{a,b,d,e,r}	18-34	1.31	
	35-40	14.25	1.14(1.09-1.19)		35-40	1.42	1.16 (1.03–1.23)
	>40	17.99	1.27 (1.15-1.39)	. ∠sabdef	>40	1.61	1.19 (0.92–1.55)
Postpartum haemorrhage ≥1000 ml ^{b,f,g,h}	18-34	1.46	1127 (1110 1107)	Apgar score $<5^{4,0,0,0,0,0}$	18-34	0.23	1 20 (1 05 1 61)
	35_40	2 10	1 28 (1 16-1 41)		>40	0.28	1.01(0.60, 1.60)
	540	2.19	1.20(1.10-1.41) 1.55(1.20, 1.88)	Admission to SCBU >24 h ^{a,b,d,e,f}	18_34	5.20	1.01 (0.00–1.09)
	~40	5.10	1.55 (1.29–1.88)	Admission to bebe > 24 h	35-40	5.20	1.05(0.98 - 1.12)
				-	>40	5.92	0.9(0.85-1.15)
				Breast feeding b,d,e	18-34	61.14	3.5 (0.02
					35-40	70.08	1.76 (1.70-1.82)
					>40	66.24	1.63 (1.52-1.75)

Growth (briefly)

Case History

e

	Da	уб
Pale / Poo	or perfusion	on / Thready puls
	18% wei	ight loss
Na+	165	(135-140)
K+	3.1	(3.0 - 5.0)
Urea	23	(<5)
Glucose	3.2	(2.6 - 6.0)
Serum osmolalit	y (mOsm / l)	=
(2x[Na+]) + (2x[])	K+]) + [Urea] + [Glucose]

Osmolality 362 (270-290)

The Dew Zealand Herald HDC report: Feb 2016

16% weight loss reported 3 days after discharge

22% weight loss when readmitted 3-4 days after this

Dehydration / hypernatraemia, intracranial bleed











Resuscitation

NZRC NLS



NZRC NLS

The Golden Minute

By 1 minute a newborn should » be establishing breathing » heart rate > 100

If not intervention recommended

Most effective intervention: » <u>Ventilation!</u>

What to do about perinatal exposure to meconium?





What to do about perinatal exposure to meconium?

Chettri 2015: RCT <u>non vigorous</u> randomised to ETT suctioning or oro/nasoph only (n = 61 per grp)
 » MAS (30%), Deaths (12%) and NDT outcomes at 9 months; no difference between groups
 » (PAS 2014 abstract: n=178 Similar result)

ILCOR 2015: Insufficient human evidence to suggest routine tracheal intubation for suctioning of meconium in nonvigorous infants born through MSAF as opposed to no tracheal intubation for suctioning.

Meconium

Crying babies : Airway will be open

support breathing as needed

■ Not breathing and floppy: These babies likely in trouble

If unskilled: do not delay ventilation

If skilled:

before stimulation have a look suction below cords till clear ventilation

All: Keep warm (if asphyxiated do not overheat)



Delayed Cord Clamping

Consider in ALL well Term babies

Suggested in pre term babies <u>not needing</u> resuscitation

Delay clamping for 30-60 seconds

Cord milking < 28w; no evidence of benefit</p>

Chest Compressions

Chest compressions 3:1 (forget 2 finger method)







Fig. 2. Aortic and right atrial pressures measured during standard CPR and ventilation using a 15:2 compression to ventilation ratio. The interruptions of aortic diastolic pressure (lower border of the dark band) during ventilation is easily recognized resulting in a sub-optimal coronary perfusion pressure during that time. Right atrial diastolic pressure is seen as the most inferior border of the pressure waves. The difference is the coronary perfusion gradient. Maximal coronary perfusion occurs only a third of each compression–relaxation–ventilation cycle.

Coronary perfusion pressure = Aortic end diastolic pressure - Right atrial pressure

Key data from key studies

Reference: Case Hx: David, 1988; Moya, 1962; Thaler, 1963; Todres, 1975

P: Neonates receiving chest compressions



Chest Compressions

□ Chest compressions 3:1 (forget 2 finger method)







Jaundice!

Why we worry about SBR's Term NVD Indian baby BW 3565

☐ Thrombocytopenia ? Cause

■ Home Day 4 ?Mild jaundice Feeding OK

Represented Day 7

Opisthotonos





Source: Adv Neonatal Care © 2004 W.B. Saunders

A Three Stage Encephalopathy

Stage 1
Lethargy / poor feeding / hypotonia

Stage 2 (after first several days)
 – Fever / Hypertonia / Opisthotonos

□ Stage 3 (after 1st week)

 Muscle rigidity / paralysis of upward gaze / periodic oculogyric crisis, and irregular respirations (IQ spared)

Why we worry about SBR's

■ Mother Blood group B +ve Baby B +ve DCT –ve

 \square G 6 P D / P K screens –ve

Metabolic –ve

2 volume exchange transfusion + PTU \rightarrow 342

□ Na⁺ 143 Not weighed on readmission. \downarrow 10% when done



Kernicterus



Status: Age 9 years

- 1. Cerebral palsy (choreoathetoid)
- 2. Global developmental delay
- 3. Increasing dystonic movements / tone
- 4. Recurrent tongue lacerations
- 5. Chronic respiratory infection aspiration
- 6. Feeding (fundoplication gastrostomy February 2008)
- 7. Hiatus hernia repair
- 8. Visual impairment wearing glasses
- 9. Seizure disorder

Authors Full Name:

Stanley, TV.

NZ Kernicterus

Institution:

Title:

Source:

Department of Paediatrics, Wellington School of Medicine, University of Otago, New Zealand.

A case of kernicterus in New Zealand: a predictable tragedy?

Journal of Paediatrics & Child Health. 33(5):451-3, 1997 Oct.

NZ Kernicterus

■ Wellington case 1997 (G6PD)

Christchurch case 2000 (anti-E)

■ Auckland case: From Tonga 33/40 ABO SBR 680

Auckland case: 2005 (presented)

Auckland 2010 (Day 2 surgical abdomen, haemolysis)

Middlemore 5 year Audit

 \square SBR > 375µmols/l (375 – 609)

60% no obvious cause but ABO high in PI

When proportions compared with delivery population some PI groups overrepresented

No audiology problems or kernicterus in this period
Commonest Neonatal Problem: Definitions

Unconjugated vs Conjugated (lipophobic)

Physiological vs Pathological (first 24 hours)

Haemolytic vs Non haemolytic

Late (>2 weeks) vs Early



Haemolytic Disease newborn:

□ Rhesus disease 'D' D,d,C,c,E,e ABO protects

ABO disease (Kell / Duffy / Kidd) Coombs Test

Group O mothers: anti-A, anti-B

IgG naturally occurring

□ Group A (anti-B) or B mother (anti-A) : **IgM** naturally occurring

■ Others: G-6-PD/PK, spherocytosis and other RBC shapes



Haemolytic disease related to antigenicity of RBC Ag's

■ At Term Rh very well developed / ABO less well.

Kell (K), Duffy (Fy), Kidd (Jk), Lewis (Le), M, S have occurred

Usually anti Jk, anti-E, anti-c

Anti-Le and anti-P usually an IgM





Coombs Test

Direct Coombs test / Direct antiglobulin test Positive test result Positive test result Positive test result ("DAT") "DAT"

Indirect Coombs test / Indirect antiglobulin test



Physiological

Early discharge

Feeding problems

 \square 10% weight loss (7 – 10 days)

Breast milk jaundice



MANAGEMENT OF JAUNDICE IN THE HEALTHY TERM NEWBORN





Affix patient identification label here

Kidz 021 June 2000

CHILD & YOUTH SERVICES NEONATAL UNIT BILIRUBIN GRAPH

Late

Unconjugated (Indirect):

- Breast milk Jaundice
- Physiological / Pathological
- <u>Hypothyroidism</u>

Conjugated (Direct) Usually < 20µmols/l</p>

- Biliary atresia
- <u>Hypothyroidism</u>

Treatments (unconjugated)





Exchange transfusion (mortality 1 in 100/200)

Underlying condition

Basic Guideline

	Total bilirubin (umol/L)
Cord blood	≤ 50
Up to 24hrs	≤ 150
>1 to ≤2days	≤ 200
>2 to \leq 3 days	≤ 250
>3 to ≤7 days	≤ 300
>7 days to ≤3 wks	≤ 100 (200-300)
>3wks to ≤4wks	≤ 50
>4wks to Adult	≤ 24

Breast Milk Jaundice

True cause of prolonged unconjugated jaundice

Exact mechanism unknown

Diagnosis of exclusion; Stop feeding!!!!!

Benign?

Clinical signs

Jaundice longer than 2 weeks

	Direct bilirubin (umol/L)	Comment
≤4wks	<20 or <10%	≥30 umol/L require attention

Biliary atresia

□ 1 in 15000 – 20000 (highest in French Polynesia)

Syndromic (other associations) and non syndromic

Non Syndromic later in gestation and progressive

5 - 10% only are correctable

Aetiology

Unknown!

Many theories

Evidence that is progressive

Timing of surgery seemingly important

Clinical signs



Clay coloured stools (dark urine)

Hepatomegaly

Differential diagnosis:

Neonatal hepatitis Interlobular bil. hypoplasia

FIGURE 1 English version of the infant stool color card (first edition)

Infant Stool Color Card

No. of Booklet :

Abnormal



It is essential to observe your baby's stool color continuously after discharge from a nursery. If the stool color resembles the numbers $1 \sim 3$ (white, claycolored, or light yellowish), the possibility on your baby suffering from biliary atresia is higher. Please take this card and your baby to consult a doctor as quickly as possible. Regardless of what the stool color is, please bring this card to your doctor at 30 days of age for health check. If the baby cannot go back for health check, please fill in the number of the color resembling your baby's stool, along with the following blanks, and mail this card to our registry center.

Normal

The baby's stool color is most like No.____ Date of this kind of stool _____

Name of the	baby	Birthday
-------------	------	----------

Name of the mother _____ Tel. _____

Address

The hospital or clinic where the baby was born

If the number is No.1 \sim 3, please inform us by fax immediately. We will provide the related information and help you out.

Fax: 02-2388-1798 ; Tel: 02-2382-0886

Infant Stool Color Card Registry Center

Chen, S.-M. et al. Pediatrics 2006;117:1147-1154



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Treatment and Outcome

Surgery initially

□ 10 year survival 70% IF surgery before 60 days

□ (Other reports 25-60%). Much worse if > 100 days

Majority will eventually need liver transplant

■ 5 year survival after liver transplant = 80-90%



Other surgical conditions







Tamariki Ma

200- [L]

units

C2419 W1332













Would this baby have bilious vomiting?

Umbilical clamp





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First meconium

■ Within 24-48 hours

□ Is there an anus?

Mucous plug syndrome: 2cms mec; white cap

Other conditions: CF, hypothyroid, Hirschsprungs



Figure 14.32. Imperforate anus with a rectovaginal fistula. The large fistulous tract allows for free passage of meconium hence this infant had no abdominal distention.

Vitamin K

Phytomenadione



ACC#ML1003586-C110 Study Date:07/12/2010 Study Time:15:57:31

Children's Hospital & Community Health Tamariki Ma

> CT HEAD STANDARD C-BRAIN C- COR Migrated Study Clinically significant mark-ups or S4^orientation data may be absent.

The New Zealand Herald

Baby suffers cerebral haemorrhage after midwife failure By <u>Martin Johnston</u> 5:29 PM Monday Jun 24, 2013

> Day 5 PR = 5.2

CMDHB SE:8029 IM:24

R

9

5

The Problem

■ Early: 1st week of life:

- » bleeding in 0.25-1.7 %
- » Parenteral and oral Vitamin K virtually eliminates

□ Late: 2-12 weeks with no or inadequate Vit K prophylaxis

- » 4.4 to 7.2/100,000 births
- » breast fed, cholestatic jaundice especially important
- » Intracranial therefore often significant sequelae
- » <u>single</u> dose <u>oral</u> Vit K <u>reduces</u> 1.4- 6.4 / 100,000
- » Parenteral virtually *eliminates*

Vit K and Leukaemia

Discredited



Mothers on anticonvulsants:
» Phenobarbitone
» Phenytoin
» Carbamazepine
Sodium valproate seems OK

Mothers on anti-TB drugs: » Isoniazid » Rifampacin

Warfarin!Instrumental deliveries

Neonatal Hypoglycaemia

□ Blood sugar < 2.6 mmol/l

 \Box (Hyperglycaemia = BG > 7.0mmols/l)

Why 2.6?



□ 17 children (5 neonates) with:

• endocrine disorders (having induced hypo's with insulin)

OR

• Recurrent spontaneous hypo's

Latency of auditory/ somatosensory evoked potential in response to clicks

Archives of Disease in Childhood, 1988, 63, 1353-1358



Fig 1 Serial somatosensory evoked potentials recorded in subject 6 in relation to her blood glucose concentration. The vertical line indicates the latency of N_1 in the initial recording during normoglycaemia.

Archives of Disease in Childhood, 1988, 63, 1353-1358

Neural dysfunction during hypoglycaemia

T H H G KOH, A AYNSLEY-GREEN, M TARBIT, AND J A EYRE

Department of Child Health, University of Newcastle upon Tyne
Adverse neurodevelopmental outcome of moderate neonatal hypoglycaemia

A Lucas, R Morley, T J Cole

661 preterm infants

If moderate hypoglycaemia (< 2.6) recorded on 5+ days; 18 months corrected age scores:

» Mental / Motor Developmental scores 14 / 13 points
» Cerebral palsy / Developmental delay 3.5x

BMJ VOLUME 297 19 NOVEMBER 1988



Logarithm of days of recorded hypoglycaemia <2.6 mmol/l related to Bayley mental development index and Bayley psychomotor development index at 18 months (corrected age). Regression slopes and 95% confidence intervals (broken lines) are shown adjusted for days of ventilation, sex, social class, birth weight, and fetal growth retardation. Data shown are for both sexes and all social classes combined and for no ventilation. For infants ventilated for 1-6, 7-14, or >14 days subtract 5, 10, or 15 points respectively for mental development index and 4.5, 9.0, or 13.5 points for psychomotor development index.

BMJ VOLUME 297 19 NOVEMBER 1988

Profound / Prolonged Hypoglycaemia



At risk babies

Infants born preterm (< 37 weeks)
Infants of diabetic mothers
Small (<2.5 kg or <10th centile)*
Large (>4.5 kg)

Babies at risk after birth for hypoglycaemia:

- Hypothermic babies
- Babies not feeding well
- Babies unwell for any reason

Out of left field



Glucose B Hydroxy Butyrate Insulin 0.9 mmol/l 0.61 mmol/l 9.0 mIU/l (3-11)(0-0.27)(3-11.0)

Why screen?

□ (Supposed)

Clinical signs:

- Jitteriness
- Sweating
- Apnoea
- Seizures

Subtle and non specific

Screening and Dextrose Gel

Blood glucose measurement within one hour of birth regardless of feeding, followed by three to four hourly pre-feed measurements.

Babies at risk after birth should have a blood glucose measurement as soon as the problem is recognised.



Discontinue blood glucose measurements when:

Babies feeding well without dextrose gel

• have three pre-feed blood glucose concentrations $\geq 2.6 \text{ mmol/l}$, three to four hours apart.

Dextrose gel treatment

☐ Aim to keep baby with mother

☐ Aim to avoid infant formula

Aim to keep blood glucose within normal limits

Dextrose gel treatment

 Syringe draw up 40% dextrose gel, 0.5 ml/kg.dose to buccal mucosa then feed (breast etc)

Recheck blood glucose 30 mins after the gel
BG ≥ 2.6 mmol/l then normal cares
BG < 2.6 mmol/l then repeat steps

Then:

- BG after 30 mins or after the feeding event
- BG \geq 2.6 mmol/l then normal cares
- BG < 2.6 mmol/l (for a second time) then escalate care to paediatrics.



Cardiac Examination



Case History

■ 18 year old primigravida

Anatomy scan 20 weeks.....Normal

■ NVD at Term

LMC care: discharged at 12 hours of age

Case History

Midwife visits at 36 hours

Baby not feeding / low temp

□ Parents to EC

Saturations: 40%

Blood glucose unrecordable



Hypoxic ischaemic changes on Head MRI

■ Severe acute tubular necrosis – PD for 2 weeks

Arterial switch at 2 weeks (2nd hit)

Cost so far \$100,000

Cardiac Examination

Dysmorphic?



Central colour (pulse oximetry > 95%)

Peripheral perfusion

Pulses: Femorals / Brachials

Cardiac Examination

Auscultate heart:

» Base
» Apex
» Supraclavicular
» Back



Clinical examination

Routine newborn examination will fail to detect > 50% of infants with significant cardiac disease

Trisomy 21 » not more effective » Sensitivity of 53%¹⁰

 Ψ Wren C, et al. Presentation of congenital heart disease in infancy: implications for routine examination. Arch Dis Child Fetal Neonatal Ed. 1999;80(1):F49-53.

 φ Tubman TR, et al. Congenital heart disease in Down's syndrome: two year prospective early screening study. BMJ. 1991;302(6790):1425-7.

Clinical examination

Mild cyanosis difficult to detect

- Ethnicity
- Perfusion
- Environmental factors

Murmurs often absent

 No correlation between severity of cardiac malformation and likelihood of detecting a murmur^{*}

 Ψ Wren C, et al. Presentation of congenital heart disease in infancy: implications for routine examination. Arch Dis Child Fetal Neonatal Ed. 1999;80(1):F49-53.

Individually rare but collectively common

Difficult to diagnose

Can we screen for this?

Elza Cloete: Neonatologist Auckland

Definitions: 5 year NZ audit

Major congenital heart disease: Structural abnormality of the heart or intra-thoracic great vessels which require intervention, or is associated with death in fetal life or in the first year of life.

■ Critical congenital heart disease: Major CHD that is duct-dependent, or requires intervention or results in death at ≤ 28 days of age.

Key findings: 5 year NZ audit

- **734** cases of major CHD
- □ 353 critical
- Incidence
 Major: 2.34 per 1000
 Critical: 1.12 per 1000
- **_** Timing of diagnosis:



Late diagnosis associated with higher mortality risk (27% vs. 16%)

Antenatal ultrasound

Modest sensitivity

- Service delivery
- Availability/quality of equipment
- Skills (Tertiary v. rural, extracardiac defects, type of defect)
- Service utilisation
- Education
- Ethnicity
- Geography
- Parity
- Age
- Maternal BMI

What type of lesions we talking about?



In utero: 2 pumps in parallel

Ex utero: 2 pumps in series

Cyanotic*

- Duct Dependent, Not breathless
- Duct Dependent, Breathless
- Not Duct Dependent and Breathless

Pink

- Duct dependent and Breathless
- Breathless

Duct Dependent, Not breathless + Blue







Duct Dependent, Breathless + Blue





Emergency Treatment

PGE₁

Duct Dependent, Breathless + Pink





■ Rarely isolated (17%)

Severe isolated: heart failure weeks

+ large VSD: heart failure days
Coarctation

extension of ductal tissue into wall of aorta

Becomes more obstructive with time



Collaterals: internal mammary, scapular & intercostals

Average BP difference between upper/lower body = 30-40mmHg

Congenital Heart Disease

Breathless + Pink







Most common 2.5/1000 livebirths

□ If parent has one; 2.9% chance of VSD recurrence

Flow through VSD dependent on pulmonary vascular resistance

Resistance falls with 1st breath then over hours



□ Size of L \rightarrow R shunt determined by hole size & PVR

Failure if pulmonary blood flow 2.5x systemic

□ If VSD 50% diameter of aorta; goes into failure

■ Usually delayed until 3rd week of life

VSD-Clinical signs

No initial murmur; appears @ day3+ (maybe none!)
Most are small & asymptomatic

Tachypnoea (>60) often first sustained sign

Feedings taking longer, growth slowed

□ Vomiting / regurgitation ↑

Moribund!



Hypoplastic Left Heart

■ Hours – days: poor colour or <u>sudden</u>, profound shock

Series of operations to create single ventricle system

Long term survival 50-75%

Screening for Cyanotic Heart disease

Joining the rest of the World (USA, UK, Scandinavia, Australia)

Criteria for screening test

?

9

Important health problem

Valid Test available

Effective treatment

Benefits of screen exceed harm of test

■ Health care system can support

Screening is socially / ethically feasible ?

Cost benefit vs harm effective

What is pulse oximetry?

A tool that provides continuous, noninvasive measurement of arterial oxygen saturation levels in the blood

- Oxygenated and deoxygenated blood differ in their absorption of red and infrared light
- Light passes through a capillary bed and then light absorption is measured



Pilot study Auckland

Test time: 2- 4 minutes



Screening should be performed 2 to 24 hours after birth on all well newborn infants with a gestational age \geq 35 weeks

Perform the test on one foot



Refer all infants who fail to reach pulse oximetry targets to the paediatric service. Clinical concern at any stage warrants immediate referral.

ACTA PÆDIATRICA

EDITORIAL

DOI:10.1111/apa.13082

Newborn pulse oximetry screening is not just for heart defects

Disorder	n	(%)
Congenital heart defect	43	13
- critical	27	8
- noncritical	16	5
Pneumonia/septicaemia	55	17
Transient tachypnoea	54	17
Persistent pulmonary hypertension	6	2
Pneumothorax	6	2
Amniotic fluid aspiration	5	2
Hypoglycaemia	3	1
Pulmonary atelectasis	1	
Hyperviscosity syndrome	1	
Respiratory distress syndrome	1	
Cardiomyopathy	1	
Unclassified	1	
Transitional circulation	147	45

Table 1 Distribution of disorders causing 324 newborn infants to fail first-day-of-life

Meberg A. Newborn pulse oximetry screening is not just for heart defects. Acta Paediatr. 2015;104(9):856-7

Tongue-tie and breastfeeding: a review of the literature

Janet Edmunds RN RM BHSc(Nrg) IBCLC Sandra Miles RN RM BN MN Paul Fulbrook RN PhD MSc BSc(Hons)

Ankyloglossia breast feeding and frenotomy

Problem a true entity but:

» RCT studies seriously flawed

» Which Patients to snip (How to diagnose?: Functional vs Anatomical)

» When to snip

» Who should do the snip

» Neonatal pain

Developmental Hip Dysplasia

(Congenital Dislocation of the Hip)





L





Developmental Hip Dysplasia (Congenital Dislocation of the Hip)



1. Classic: Neurologically normal child (1 in 1000)

2. Secondary to underlying CNS disorder (rare)

Developmental Hip Dysplasia

9:1 female predominance

Positive family history (20%)

Generalized ligamentous laxity (oestrogens) / shallow sockets

■ 60% firstborn

■ 30–50% breech (extended leg, flexed hip worst)

Left hip

Developmental Hip Dysplasia
A dislocated *hip* rests in a dislocated (posterior) position and reduces only with manual effort

physical examination for a gentle clunk of the *hip* out of (adduct-Barlow sign) or into (abduct-Ortolani sign) the acetabulum shows the problem

Affected *hip* may rest in slight adduction and may have a deeper proximal thigh crease *but these signs are not constant* **Developmental Hip Dysplasia**

Baby warm, quiet, and relaxed on parent's lap

□ 1 *hip* at a time

Gentle downward pressure on knee or thigh with adduction

Feel whether *hip* goes partially or fully out (Barlow test).

■ Abduct to feel it slide back in (Ortolani test).



A click may be felt in the *hip*, but <u>very</u> nonspecific

Click often is felt in <u>normal</u> *hips* and comes from the meniscus of the knee, fascia lata, or a synovial fold.

The clunk of instability usually is lost after ~6 months, when the dislocation becomes more fixed.



■ The *hip* should be reduced within the 1st 6 weeks if the dislocation is recognized.

The earlier the diagnosis is made, the easier and safer the treatment will be



Pavlik harness \rightarrow 95% resolution by 6 weeks from newborn period

Club Foot vs Positional Talipes





Eye Examination

Are the eye(s) present!

■ Are they the same size?

Pupil reaction (present at Term)

Red reflex















Coloboma of the iris


Eye discharge

Red Eye



Neisseria gonorrhoeae

White Eye



Nasolacrimal duct obst. 6% newborns Conservative management Massage: 80% fixed 1 month May get infected

Squint



After 3 months of age needs referral

What is this rash?



Immunisations

The New Zealand Herald

Saturday Jan 19, 2013

Son's ordeal was our fault, say parents

Father says decision to refuse tetanus shot made without facts



The New Zealand Herald

Monday Feb 25, 2013

Whooping cough epidemic spreads



■ DTaP ■ DTaP ■ DTaP Charlen Constant Charlen Constant







PCV 13





Hepatitis BsAg +ve

HBsAg+ve / HBeAg+ve: Perinatal transmission 70-90%

■ HBsAg+ve / HBeAg-ve: Perinatal transmission 5-20%

Chronic infection develops in 90% of those who acquire perinatal transmission

Vaccine Efficicacy

□ Up to 90% reduction in vertical transmission

75% reduction in hepatocellular carcinoma in vaccinated population (Italy, Gambia, Asia)

At birth:

- » Immunoglobulin as close as possible to birth but can be given up to 10 days
- » HB vax as close as possible to birth up to 12 hours

Neonatal Sepsis

Infections in the first month

1. Congenital infection sustained in utero

2. Acquired during birth

3. Acquired in nursery (breast milk)

4. From household

Fever $\geq 38^{\circ}$ C if < 6 weeks

High risk group

■ Majority viral **<u>BUT</u>** bacterial infection probability = 15%

Rapid progression

Full septic workup prudent

UTI common and serious (catheter / bladder stab)

Measuring a Temperature

Rectal the gold standard < 2 years » trend now for 6 months cut off for rectal » electronic OK

 $\square > 38^{\circ}C$ Rectal = fever

Axillary and tympanic (tympanic better than axilla)
 » Both under record fever
 » For tympanic; EAM often too small in babies

Provides at best approximation of core temp esp > 39°C

Treating Fever

Paracetamol: 60mg/kg/day < 3 months (10mg/kg/dose)</p>

■ Standard dose: lower core temp by about 1°C

Aim to get child comfortable; not abolish fever

Avoid brufen (gastritis, renal, Reyes)

Recent Research

Over 200,000 children from 73 centres in 31 countries included in analysis

■ Paracetamol for fever in 1st yr of life – ?associated with:

- 46% increased risk of asthma (severe asthma ↑ increased 22-38%)
- 48% increased risk of rhinoconjunctivitis
- 35% increased risk eczema Beasley, Clayton, Crane et al (2008)
- Recent work debunks: Not strong enough evidence to change practice Lowe AJ et al, BMJ 2010



Respiratory rate: 40 – 60 asleep! (periodic respirations)

Temperature instability

– HR 90 − 160 asleep!

 \square MAP = Gestation

Listen to the (experienced) mother

"off his feeds"

"sleeping more than usual"

"fussy"

"just not right"

Group B streptococcus (agalactiae)

Causes of Neonatal Sepsis - UK



Pathogen

Enterococcus

S. aureus

Klebsiella sp.

Candida sp.

sp. E. coli Number

264

246

242

172

158

Percentage

15%

14%

14%

10%

9%

Pathogen	Number	Percentage
GBS	225	43%
E. coli	95	18%
Other Streptococci	66	13%
Micrococcus sp.	23	4%
Enterococcus sp.	19	4%

Unpublished data from the neonIN neonatal infection surveillance network.



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Characteristic	Early Onset	Late Onset	V. Late Onset
Onset	< 7 days of age (usually first 48 hrs)	7 to 30 days	30 days+
Gestation	25% <37 weeks	Often full term	Usually <30 weeks
Risk factors	Maternal intrapartum complications common	Often none	Prematurity
Source of organism	Maternal genital tract	Maternal genital tract, nosocomial, or community	Nosocomial, community
Usual clinical presentation	Nonspecific signs or respiratory distress	Focal or nonspecific signs	Focal or nonspecific signs
Case-fatality ratio	10%-20%	5%-10%	<5%



How does GBS get to infant during labour and delivery?

■ Why are newborns (esp prems) so susceptible?

How does it evade host defences?

How does bacteraemia and meningitis occur?

Neonatal Immunology

■ Maternal T cell function \downarrow during pregnancy ?tolerance

Specific GBS capsular polysccharide associated with bad disease

Transplacental immunity via passive transfer of IgG (IgG t¹/₂ 20 days; less if prem +/- sick)

higher the concentration to this polysccharide the more protection

■ <u>Majority</u> passive therefore ∝ maternal concentrations (start @ 8/40 / majority in last weeks) Group B Streptococcus (agalactiae)
Very good at binding to vaginal wall cells

May <u>induce</u> ROM and prem delivery

Penetrates intact membranes / loves amniotic fluid especially if contains meconium

 Deficiencies in neonatal neutrophil response (phagocytes)

Group B Streptococcus (agalactiae)

■ 15 – 40% women colonised (comes and goes)

- Historically occurred in 1 - 4/200 colonised women

Much lower with intrapartum antibiotics

Group B Streptococcus (agalactiae) ■ Early onset (75%) (0 – 6 days) Mean = 18 hours

 \Box 0.7 – 3.7/1000 livebirths (with antibiotics)

Late onset (7 - 89 days)

■ 0.5 – 1.8/1000 uninfluenced by peripartum antibiotics

Total (early-late) case fatality 8%



■ 80% pneumonia therefore clinical signs respiratory

Meningitis unusual but more common in late onset



Neonatal Meningitis

- Group B streptococci (Streptococcus agalactiae)

 Escherichia coli (and other gram-negative enteric bacilli)

Listeria monocytogenes

Enterococci

Neonatal Meningitis

Especially preterm babies immunocompromised

Blood-brain barrier immature

GBS & Listeria meningitis usually after the first week

Mortality / Morbidity highest in newborn

Clinical sign of GBS Bacteraemia	Percentage of infants with sign
Hyperthermia	51
Hypothermia	15
Lethargy	25
Irritability	16
Respiratory distress	33
Apnea	22
Cyanosis	24
Jaundice	35
Hepatomegaly	33
Anorexia	28
Vomiting	25
Abdominal distention	17

Maternal intrapartum treatment (early onset)

Previous GBS infected child

GBS bacteruria

Preterm labour and imminent birth

- Fever $> 38^{\circ}C$
- **PROM** > 18 hours **OR**

Positive maternal screening 35-37/40 current pregnancy