CHILDREN’S SKIN SYMPOSIUM
Saturday 15th November | The Royal Society of Medicine

Contact us:

E: portlandgp@hcahealthcare.co.uk
T: 0207 390 6057
F: 0207 390 6069

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Paediatric Facilities

- 60 dedicated paediatric beds including: 10 PICU beds, 6 cot NICU/SCBU, 11 bed day case unit & 9 bed acute rehabilitation unit

- Full range of imaging services: US, X-ray, Interventional radiology, MRI and CT (including under GA)

- Neurophysiology (EMG and EEGs)

- Audiology Services

- Neonatal Care

- Therapy Services ‘one stop shop’

- Play rooms and dedicated play therapist team
First Class Facilities
Paediatric Medical Admissions Service

- Dedicated 24/7 service for GPs to refer medical cases to The Portland Hospital for direct admission

- A senior member of our paediatric nursing team will arrange the admission in conjunction with our on call consultant paediatrician, paediatric RMO and the referring GP via a three way telephone call

- The dedicated telephone line for medical admissions is: +44 (0)207 390 8111

More information on the service including what referrals are appropriate can be found on our website:

W: www.theportlandhospital.com/GP
Paediatric Consultations

• Same day and next day paediatric appointments available Monday to Saturday

• Referrals accepted by email, fax and post – either named or unnamed referrals

• All referral requests responded to within one working day and patient contacted directly for appointment

• Audiology, Imaging and Therapy departments accept referrals directly from GPs

T: 0207 390 6057    F: 0207 390 6069
E: portlandgp@hcahealthcare.co.uk  W: www.theportlandhospital.com
Specialties

- Allergy and Immunology
- Audiology
- Acute Neurorehabilitation
- Cardiology
- Cochlear Implant Program
- Craniofacial Surgery
- Dermatology
- Endocrinology
- ENT
- Gastroenterology
- Genetics
- General Paediatrics
- Hand Surgery
- Genetics
- General Surgery
- Limb reconstruction
- Maxillofacial
- Neonatology
- Neonatal Surgery
- Neurosurgery
- Neurophysiology
- Orthopaedics
- Peripheral Nerve Injury
- Plastic and Reconstructive Surgery
- Respiratory
- Rheumatology
- Spinal Surgery
- Urology
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- Plastic and Reconstructive Surgery
- Respiratory
- Rheumatology
- Spinal Surgery
- Urology
Dermatology

Prof John Harper
Dr Nerys Roberts
Dr Mary Glover
Dr Anna Martinez
Dr Tabi Leslie
Dr Bisola Laguda
Birthmarks, propranolol and laser treatment

Professor John Harper

Children’s Skin Symposium, RSM 15th Nov 2014
Birthmarks

~ 80% of newborns

Alper JC, Holmes LB. The Incidence and Significance of Birthmarks in a Cohort of 4,641 Newborns. Ped Derm 1983; 1 (1): 58-68.
What is a birthmark?

- Naevus = birthmark [latin]
- Benign developmental irregularity of the skin present at or soon after birth
- Blood vessels, melanocytes, smooth muscle, fat, fibroblasts or keratinocytes
Blaschko’s lines
PHACES syn

Post fossa malformtions
Haemangiomas
Arterial anomalies
Coarctation of the aorta
cardiac defects
Eye abnormalities
Sternal defects
Birthmarks

- Early recognition
- Provision of accurate information
- Plan of management, if appropriate
- Neonatal Skin Screening
Propranolol for haemangiomas
TO THE EDITOR: Despite their self-limited course, infantile capillary hemangiomas can impair vital or sensory functions or cause disfigurement. Corticosteroids are the first line of treatment for problematic infantile capillary hemangiomas; other options include interferon alfa and vincristine. We have observed that propranolol can inhibit the growth of these hemangiomas. Our preliminary data from 11 children are summarized in Table 1 in the Supplementary Appendix, available with the full text of this letter at www.nejm.org.
Propranolol for haemangiomas

- only for those that could potentially cause a problem
- treatment started in hospital
- needs careful monitoring
- 1 - 3mg/kg/day in 3 divided doses
- Continue for up to one year
Propranolol for Haemangiomas
Experience at GOSH + PH

- > 350 infants treated
- Most show an excellent and rapid response to treatment
- Risk of hypoglycaemia in pre-term babies
- Wheezing, sleep disturbance
- No significant side effects
- Propranolol stopped in only 3 patients with deep ulcerations because of severe pain and non-healing
How does propranolol work?

- Blocks β2 adrenergic receptors on the surface of the endothelial cells
- Vasoconstriction
- ↓ VEGF and bFGF
- ↓ Endothelial cell proliferation
- Induces apoptosis
Present position with propranolol

Currently this treatment should be reserved for babies with haemangiomas that potentially could cause serious complications. Treatment should be initiated and supervised by a specialist centre. More research is needed to define the optimum treatment regimen and to investigate why propranolol has this beneficial effect on infantile haemangiomas. Are other beta blockers as effective and safer?
Topical Timolol for early haemangiommas

Early results very good....
Laser treatment for capillary malformations / port wine stains
• Pulsed dye laser
• 595nm
• 3-30 J/m²
• Variable pulse width 1.5-30 msecs
• Inbuilt cooling device
Vesicles and bullae in children

Dr Anna Martinez
Great Ormond Street Hospital
London, UK
November 15th RSM
Blistering skin

Differential diagnosis

Investigations

Management

Blistery neonate - could it be EB?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
- Distribution?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
- Distribution?
- Mucous membranes?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
- Distribution?
- Mucous membranes?
- Age of child – newborn
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
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Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
- Distribution?
- Mucous membranes?
- Age of child – newborn
- Drug history?
Blistering skin: general considerations

- Vesicles, bullae, erosions or ulcers?
- Crust, scale?
- Distribution?
- Mucous membranes?
- Age of child – newborn
- Drug history?
- Unwell or not?
Acquired lesions
Acquired

- Infection
- Reactive
- Autoimmune
- Mast cell disease
- Artifact and physical
Infection
Bullous impetigo

- Exfoliative toxin A produced by certain strains staph aureus
- Toxin targets desmoglein1 part of desmosomes leads to loss of cell adhesion superficial epidermis
Bullous impetigo

- Large tense pustular lesions
- Diagnosis pus/fluid lesion
Staphylococcal scalded skin syndrome (SSSS)

- Caused by same exfoliative toxin of staph aureus
- Neonates & infants susceptible
- Febrile & unwell
- Superficial sheeted flexural erosions are important clues
Staphylococcal scalded skin syndrome (SSSS)

- Peri-oral erythema
- Superficial flaccid bullae & desquamation
- Nikolsky sign positive
Staphylococcal scalded skin syndrome (SSSS)

- Intact bullae are sterile
- Swab nostrils, nasopharynx, blood cultures
- Diagnosis clinical
Staphylococcal scalded skin syndrome (SSSS)

- Frozen section of blister roof if doubt
- Prompt antibiotics and supportive care
- Mortality 5%
- Look source
Eczema herpeticum

- Disseminated infection HSV 1 or 2
- Very unwell child
- Painful punched out lesion
- Face & neck
Eczema herpeticum

- Confirm viral swabs from fresh blister
- Prompt antiviral therapy
Reactive acquired
Steven–Johnson syndrome (SJS) & toxic epidermal necrolysis (TEN)

- Considered disease continuum
- Distinguished severity & percentage body surface area affected
- SJS less 10% body SA skin detachment mucosal membranes affected >90%
- TEN over 30% skin detachment
- Overlap
SJS & TEN

- Drugs leading trigger
- Infection
- Genetic factors associated risk
- HLA types higher risk of SJS
  - HLA B1502 Asia
Pathogenesis

- Not fully understood
- Likely cell mediated cytotoxic reaction against keratinocytes leading to massive apoptosis
- Full thickness necrosis
- Nikolsky sign positive
Management

- Identify remove cause
- Eye care critical
- PICU
Acquired causes

- Physical & chemical induced
- Phototoxic
- Artifact
Mast cell disease
Mastocytosis

● Group of disorders
  excess mast cell accumulation

● Subdivided into 2 groups
  ● cutaneous
  ● systemic

● Pathogenesis not clear
Cutaneous mastocytosis-urticaria pigmentosa

- 80% present under 1yr
- Gain of function mutations in *KIT* the gene encoding c-Kit receptor
- Presents macular & papular yellow-tan lesions
Urticaria pigmentosa

- Blisters can occur
- Darier’s sign
- Avoid triggers
  - physical factors
  - drugs
- Clinical diagnosis
- Resolves by puberty
Autoimmune
Autoimmune

- Childhood bullous disease of childhood
- Bullous pemphigoid
- Dermatitis herpetiformis
Chronic bullous disease of childhood

- Acute vesicles or bullae on sites of inflamed skin
- Widespread lesions & face, trunk, genitals
Chronic bullous disease of childhood

- New blisters at periphery of resolving lesions
- String of pearls or rosettes
Chronic bullous disease of childhood

- Linear IgA antibodies bound dermal-epidermal junction BMZ
- Diagnosis direct & indirect immunofluorescence
- Treatment
Bullous pemphigoid

- IgG antibodies BMZ
- Lesions trunk and extremities
Bullous pemphigoid

- Larger hemorrhagic lesions
- Diagnosis
- Treatment
Dermatitis herpetiformis

- Cutaneous manifestations coeliac
- Can present before any other symptoms
- Intensely pruritic grouped vesicles & excoriated papules
- Buttocks & extensor surfaces
Dermatitis herpetiformis

- Diagnosis
  - Skin bx
  - Serology
- Gluten free diet life
- Dapsone
- Associated other autoimmune dx
- Increase risk lymphoma
Inherited

- Incontinentia pigmenti
- Epidermolytic ichthyosis (BIE)
- Epidermolysis bullosa (EB)
- Metabolic & porphyrias
Incontinentia Pigmenti

- X linked dominant
- Mutation in \textit{IKBKG} gene ( inhibitor of kappa B kinase subunit gamma gene ( NEMO) )
Incontinentia Pigmenti

- Teeth abnormality
- Eye abnormality
  retinal detachment
Epidermolytic Ichthyosis

- Autosomal dominant
- Mutation Keratin 1 or 10
- Blisters & erosions from birth
Epidermolytic Ichthyosis
Epidermolysis Bullosa
More on EB later!
Blistering skin

Differential diagnosis

Investigations

Management

Blistery neonate - could it be EB?
Investigations

- Depending on history & physical signs
- Swabs for bacterial, viral or fungal culture
- Fungal scrapes
- Serology

- Metabolic investigations
Skin biopsy

- Drug reactions
- Autoimmune bullous disease
- Bullous genodermatosis
Skin biopsy: autoimmune

- Lesional for H & E
- Perilesional for direct IF
Skin biopsy: autoimmune

- Lesional for H & E
- Perilesional for direct IF
- Serum for indirect IF
Blistery neonate - could it be EB?
Management: basic principals

- To pierce or not to pierce?
- Dressings
- Treatment & prevention of infection
- Importance of adequate analgesia
Management

- To pierce or not to pierce?
Management

- To pierce or not to pierce?
  - If bullae are tense and at risk of enlarging e.g. EB, TEN, then should pierce with sterile needle and leave roof intact.
Basic principles dressings

- Use non-adherent products
  - soft silicone
  - lipido-colloid
Soft silicone range

- Mepitel and Mepilex range (MHC)

- Adaptic Touch (Systergenics)
Lipidocolloid dressings

- Urgotul
- Polyester impregnated net with hydrocolloid particles & vaseline
Cooling dressings

- Intrasite Conformable
Secondary dressing & retention bandage
Other useful products

- Special fixation products
- Adhesive removers
Safe fixation
Silicone medical adhesive removers

- Appeel (Clinimed)
- Niltac (Trio Healthcare)
Management of infection
Topical antimicrobials

Flaminal range

Crystacide cream
Topical antibiotics

- Short-term use
- Problems with resistance
Systemic measures

- Only if topicals not adequate or systemic spread
- Always swab first for sensitivities
- Infection is a clinical diagnosis
Blistering skin

Differential diagnosis

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Management

Blistery neonate - could it be EB?
Epidermolysis Bullosa

- Genetic disease of fragility skin and mucosal membranes
- Incidence 1 in 17,000 UK
- National service for all patients with EB
- 2 paediatric centres
EB: referral process

- Call GOSH or BCH EB Nurses
- Give immediate telephone advice
- Courier pack with dressings and written info
- Visit within 24-48 hours, do biopsy and bloods
EB: immediate management

- Gentle handling
EB: immediate management

- Avoid incubator
- Avoid adhesive tapes, name bands, clothing seams, cord clamp etc.
EB: diagnosis

- Skin biopsy
- Bloods from baby and parents for genetic analysis
Electron microscopy

Immunohistochemistry

Mutation detection
Cannot tell what type of EB clinically
EB: diagnosis and prognosis

Severe generalized recessive dystrophic EB
EB: diagnosis and prognosis

Severe generalized recessive dystrophic EB
EB: diagnosis and prognosis

Junctional EB
severe generalised
EB: diagnosis and prognosis
Molecular analysis

- 19 different genes
- Accurate diagnosis and prognosis
- Prenatal diagnosis
Summary

- Cutaneous blisters can occur in a wide variety of clinical settings
- Infection should always be considered especially in the newborn
- Important to recognise promptly life-threatening disorders
Child suspect skin fragility

Great Ormond Street Hospital
020 7829 7808 (working hours)
020 7405 9200 ask for EB CNS mobile (out of hours)

Birmingham Children’s Hospital
0121 333 8224
Thank you
Infantile and Adolescent Acne

Dr Tabi Leslie
BSc(Hons) MBBS(Hons) FRCP(London)

Honorary Consultant, St John’s Institute of Dermatology
Guy’s & St Thomas’ Hospital, London
Consultant Dermatologist, Royal Free London
Pediatric Acne Management: Optimizing Outcomes

My tips on treatment

Dispelling myths
Evidence-Based Recommendations for the Diagnosis and Treatment of Pediatric Acne

Lawrence F. Eichenfield, Andrew C. Krakowski, Caroline Piggott, James Del Rosso, Hilary Baldwin, Sheila Fallon Friedlander, Moise Levy, Anne Lucky, Anthony J. Mancini, Seth J. Orlow, Albert C. Yan, Keith K. Vaux, Guy Webster, Andrea L. Zaenglein and Diane M. Thiboutot

Pediatrics 2013;131;S163
DOI: 10.1542/peds.2013-0490B

American Acne and Rosacea Society panel developed these recommendations for management and evidence-based treatment - endorsed by the American Academy of Paediatrics
• **Recommendations**: classification, diagnosis, evaluation and management of paediatric acne, based on age and pubertal status

• **Treatment considerations** include OTC products, benzoyl peroxide, topical retinoids, topical antibiotics, oral antibiotics, hormonal therapy, and isotretinoin

• **Simplified treatment algorithms** for adolescent, preadolescent, infantile and neonatal acne

• **Other considerations**: psychosocial effects, adherence to treatment regimens, role of diet
Paediatric Acne

• One of the most common skin conditions in children and adolescents
  – Often perceived as self-limited disease of adolescence
    • reported prevalence rate 85% in ages 12-14yrs\(^1\)
  – Detrimental psychosocial effects
  – May lead to permanent scarring

• 12 yrs no longer considered lower end of age range for acne onset
  – 78% of girls aged 8-12yrs
  – 50% of boys aged 10-11yrs\(^2\)

• Acne occurs in different ages: neonates, infants and young children
  – Differential diagnosis and systemic pathology may differ from adolescent acne

• Variations in management across primary and secondary care spectrum

1. Bhate K. and Williams HC. Epidemiology of acne vulgaris. *Br J Dermatol* 2013; 168(3): 474-485
2. Lucky AW, Biro FM, Huster GA *et al.* Acne vulgaris in premenarchal girls. An early sign of puberty associated with rising levels of dehydroepiandrosterone. *Arch Dermatol.* 1994; 130(3): 308-314
Acne Classification by Age

Be aware of differential diagnosis for each group.

Workup is based on age and physical findings.

Physical examination focused on:
- Type and distribution of acne lesions
- Height, weight, growth curve
- Blood pressure abnormalities

Signs of precocious sexual maturation or virilization should prompt workup and/or referral to paediatric endocrinologist.

**TABLE 2** Expert Panel Consensus: Pediatric Acne Categorized by Age

| Acne Type      | Age of Onset                                      |
|----------------|---------------------------------------------------|
| Neonatal       | Birth to ≤6 wk                                    |
| Infantile      | 6 wk to ≤1 y                                      |
| Mid-childhood  | 1 y to <7 y                                       |
| Preadolescent  | ≥7 to ≤12 y or menarche in girls                  |
| Adolescent     | ≥12 to ≤19 y or after menarche in girls           |

Eichenfeld et al. 2013
Neonatal Acne

• Up to 20% newborns affected

• **Self-limiting** and usually **mild** - Most cases resolve spontaneously at 1 - 3 months. May persist up to 12 months

• Erythematous papules, pustules, and sometimes comedones
  – *Malessezia* species colonizition

• Lesions typically on face

• ? caused by stimulation of **sebaceous glands** by **maternal androgens**¹

• ? high activity of neonatal adrenal glands, genetically determined **high androgen-sensitivity of sebaceous glands**²

1. Henderson CA, Taylor J, Cunliffe WJ. Sebum extretion rates in mothers and neonates. *Br J Dermatol* 2000; 142; 110-1
2. Herane MI, Ando I. Acne in infancy and acne genetics. *Dermatology* 2003; 206: 24-8
Infantile Acne

- May begin around 6 weeks of age
- Lasts for 6 – 12 months or, rarely, for years
- Presents with comedones as well as inflammatory lesions
  - Papules, pustules, or occasionally nodular lesions
- If physical examination is abnormal, hormonal workup may be necessary by paediatric endocrinologist

1. Cunliffe WJ, Baron SE, Coulson IH. A clinical and therapeutic study of 29 patients with infantile acne. *Br J Dermatol* 2001; 145(3): 463-466
# Neonatal / Infantile Acne Diagnosis

|                        | Neonatal                                      | Infantile                                      |
|------------------------|-----------------------------------------------|------------------------------------------------|
| **Onset**              | Often 2 to 3 weeks of age                    | Often 3 to 6 months of age                     |
| **Lesions**            | Pustules; less likely, comedones             | Comedones, pustules, cysts                     |
| **Possible etiology**  | *Malassezia* species colonization            | Androgens may play a role                      |
|                        | *(neonatal cephalic pustulosis)*             |                                                |
| **Course**             | Spontaneous resolution, usually by about 1 month of age | Can persist for months to years                |
| **Sequelae**           | None                                          | Scarring possible with inflammatory disease; possible association with severe acne in adolescence |

Eichenfield 2011
Mid-Childhood Acne

• Acne very rare in children aged 1 – 7 yrs

• Presents primarily on the face with a mixture of comedones and inflammatory lesions

• Endocrine abnormality should be suspected
  – Children do not produce significant levels of adrenal or gonadal androgens
  – Referral to paediatric endocrinologist is warranted
  – Rule out adrenal or gonadal/ovarian pathology including androgen secreting tumours/ congenital adrenal hyperplasia
  – Increased bone age and accelerated growth are important indicators of excess androgens

1. Cant tore-Francis JL, Glick SA. Childhood acne: evaluation and management. *Dermatol Ther* 2006; 19(4): 202-209
Preadolescent Acne

• Acne in child aged 7 – 11 years
  – Defined by age group rather than maturation / stage of puberty

• Appearance of comedonal, mildly inflammatory lesions
  – Predominance on the forehead and central face (“T-zone”)
    – Represent *adrenal awakening* as a result of normal adrenarche and testicular / ovarian maturation

• Trend towards earlier age of onset of maturation
  ➢ *Downward shift in age* at which acne first appears
Adolescent Acne

- History and physical examination most important in this age group

- Further workup usually unnecessary unless signs of excess androgens

- Polycystic ovary syndrome (PCOS) or another endocrinologic abnormality considered where acne severe, signs of excess androgens or unresponsive to treatment

Krakowski AG, Eichenfield LF. Paediatric acne: clinical presentations, evaluation and management. *Deramtol Ther.* 2006; 19(4): 202-209
Adolescent Acne
Pathophysiology
Acne is an inflammatory disease of the pilosebaceous unit – hair follicle and sebaceous gland

1. Abnormal keratinocyte proliferation and desquamation that leads to ductal obstruction

2. Androgen driven increase in sebum production

3. Proliferation of *Propionibacterium acnes* (*P. acnes*)

4. Inflammation can result in scarring and post-inflammatory hyperpigmentation
Treatment
The scars of acne!

- Acne vulgaris commonly affects children around puberty and occasionally affects infants.

- Choice of treatment depends on age, severity and whether the acne is predominantly inflammatory or comedonal.

- Inflammatory response to *P. acnes* results in permanent disfiguring scars.

- Stigmata of severe acne leads to social ostracism, withdrawal from society and severe psychological depression.

- Treatment of acne should be commenced early to prevent scarring.
OTC and Self Care

Washing, salicylic acid, benzoyl peroxide, moisturiser, sunblock

✧ OTCs can be effective for some patients with mild acne, but they must be used continuously to clear acne and prevent flares
New insights into the management of acne:
An update from the Global Alliance to Improve Outcomes in Acne Group

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Cardiff and Harrogate, United Kingdom; Singapore; Santiago, Chile; Buenos Aires, Argentina;
New Delhi, India; Kyoto, Japan; Barcelona, Spain; Caracas, Venezuela; Rio de Janeiro, Brazil;
Sydney, Australia; Toronto, Ontario, Canada; and Houston, Texas
# Global Alliance Acne Treatment Algorithm

| Acne Severity | MILD | MODERATE | SEVERE |
|---------------|------|----------|--------|
| Comedonal     | Topical Retinoid | Topical Retinoid + Topical Antimicrobial | Oral Antibiotic + Topical Retinoid +/- BPO |
| Mixed and Papular/Pustular | Oral Antibiotic + Topical Retinoid +/- BPO | Oral Antibiotic + Topical Retinoid + BPO | Oral Isotretinoin |
| Mixed and Papular/ Pustular | Oral Isotretinoin | High Dose Oral Antibiotic + Alt. Topical Retinoid |
| Nodular       | Oral Isotretinoin | High Dose Oral Antibiotic + Alt. Topical Retinoid |
| Nodular/Conglobulate | High Dose Oral Antibiotic + Alt. Topical Retinoid |

| Alternatives | 1st Choice | Alternatives | 1st Choice | Alternatives | 1st Choice | Alternatives | 1st Choice |
|--------------|------------|--------------|------------|--------------|------------|--------------|------------|
| Alt. Topical Retinoid or Azelaic acid or Salicylic acid | See 1st Choice | Alt. Topical Retinoid Antimicrobial Agent + Alt. Topical Retinoid or Azelaic acid | See 1st Choice | Alt. Oral Antibiotic + Alt. Topical Retinoid +/- BPO | See 1st Choice | Oral Isotretinoin or Alt. Oral Antibiotic + Alt. Topical Retinoid |
| Alternatives for Females | See 1st Choice | See 1st Choice | Oral Antiandrogen + Topical Retinoid/Azelaic Acid +/- Topical Antimicrobial | Oral Antiandrogen + Topical retinoid/Azelaic Acid +/- Oral Antibiotic +/- Alt. Topical Antimicrobial | Oral Antiandrogen + Topical retinoid/Azelaic Acid +/- Topical Antimicrobial |
| Maintenance Therapy | Topical Retinoid | Topical Retinoid +/-BPO |

Thiboutet et al. New insights into the management of acne: An update from the Global Alliance to Improve Outcomes in Acne Group. J Am Acad Dermatol 2009; 60: S1-50
Topical Treatment

- Benzoyl peroxide 4-10% or a topical antibiotic most effective against inflammatory acne

- Topical retinoids:
  - Adapalene, Tretinoin, Isotretinoin

- Azelaic acid 20%

- Topical antibacterials:
  - Erythromycin, Clindamycin

- Combined treatment
  - Epiduo, duac gel, treclin
Systemic Therapies

Oral antibiotics

• **Cyclines** - Lymecycline, Doxycycline, Tetracycline, Minocycline
• **Macrolides** - Erythromycin
• **Trimethoprim**

Oral anti-androgens - Dianette, Yasmin
Effects of different agents

- **Topical retinoids**: Comedolytic and sometimes anti-inflammatory

- **Benzoyl peroxide**: Antimicrobial plus weakly anti-inflammatory and comedolytic

- **Antibiotics**: Antimicrobial and anti-inflammatory

- **Hormonal agents**: Sebosuppressive

- **Oral retinoids**: Comedolytic, sebosuppressive, antimicrobial and anti-inflammatory
Isotretinoin (Roaccutane):
Targets all 4 components involved in the development of acne

Cure rate = 66%

- Shrinks oil glands
- Reduces acne bacteria
- Helps prevent clogs
- Reduces inflammation
• 0.5 - 1 mg / kg / day, cumulative dose 120 – 150 mg / kg

• Dose slowly increased as tolerated over 16 – 24 week course

• At least half of patients cured after single course

• 20% pts require repeat treatment

• Side effects
  – Teratogenic
  – Psychological
  – Other reactions

- Goodfield et al. Advice on the safe introduction and continued use of isotretinoin in acne in the U.K. Br J Dermatol. 2010: 162(6): 1172-1179
- Kar et al. Comparative study of oral isotretinoin versus oral isotretinoin + 20% salicylic Acid peel in the treatment of active acne. J Cutan Aesthet Surg. 2013: 6(4): 204-8
N-Lite Pulse Dye Laser

- Laser emitting pulse of yellow light combining with ‘porphorine’ found in bacteria creates oxygen, which destroys bacteria causing acne
- Promotes collagen aiding with healing, resulting in little or no scarring
- 30 minutes procedure and is relatively painless
- Minimum 3 sessions, results seen after 2 weeks, lasting for up to 3 months
Myths about acne

- Central discolouration in blackheads is not dirt
- Little evidence for an association between acne and poor facial hygiene
- Association between acne and high dairy diets and high glyceamic load
• **Recommendations**: classification, diagnosis, evaluation and management of paediatric acne, based on age and pubertal status

• **Treatment considerations** include OTC products, benzoyl peroxide, topical retinoids, topical antibiotics, oral antibiotics, hormonal therapy, and isotretinoin

• **Simplified treatment algorithms** for adolescent, preadolescent, infantile and neonatal acne

• **Other considerations**: psychosocial effects, adherence to treatment regimens, role of diet
Infantile and Adolescent Acne

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Honorary Consultant, St John’s Institute of Dermatology
Guy’s & St Thomas’ Hospital, London
Consultant Dermatologist, Royal Free London
Cutaneous signs of harm

Dr Mary Glover
Expert paediatrician struck off the medical register

KILLERS

Who’s to blame?

Full shame of carers exposed

WHO ARE THEY PROTECTING?

Council ‘not doing their job’
Potential obstacles

- Concern about missing a treatable disorder/being wrong
- Fear of losing a positive relationship with a family
- Discomfort of disbelieving, thinking ill of, suspecting or wrongly blaming a parent or carer.
- Divided duties to adult and child patients and breaching confidentiality.
- An understanding of the reasons why the maltreatment might have occurred
- Doubts about the benefits of the child protection process
- Fear of complaints.
Potential obstacles

- Concern about missing a treatable disorder/being wrong
  - Fear losing a positive relationship with a family
  - Discomfort of disbelieving, thinking ill of, suspecting or wrongly blaming a parent or carer.
  - Divided duties to adult and child patients and breaching confidentiality.
  - An understanding of the reasons why the maltreatment might have occurred
  - Doubts about the benefits of the child protection process
  - Fear of complaints.

NICE 2009
The questions

Skin lesion

- External damage
  - Accidental
  - Inflicted

- Manifestation of a disease process
Skin signs: accidental or not?

The most common manifestations of physical child abuse are cutaneous (90%)

Paediatr Rev 2000;21:34-35
Injury: accidental or not?

- **History**
  - Infants and young children: carers’ accounts
  - Older children and adolescents: own account

- **Age**
  - Accidental injury unusual in pre-ambulatory infants
  - Self-inflicted factitious lesions usually > 8 years

- **Site**

- **Morphology**
Bruising

- Child not independently mobile
- Relatively protected sites
  - Medial and posterior thighs, ears, neck, abdomen
- Varying stages of evolution
- Shape
  - Linear, loop, slap, circumferential
Inter-canine distance > 3cm = adult
Photograph with tape
Animal bites tend to tear and puncture
• child not independently mobile
• Areas unlikely to come in contact with hot objects by accident: backs of hands, soles of feet, buttocks, back
• Certain shapes (cigarette, iron)
• scalds in glove or stocking distribution
• scalds to limbs with symmetrical distribution
• scalds with sharply delineated borders.
Cigarette

- **Inflicted**
  - 7-10mm diameter
  - Round
  - Central crater

- **Accidental**
  - Oval or eccentric
  - Superficial
Inflicted injury or disease process?

- Skin lesion
  - External damage
    - Accidental
    - Inflicted
  - Manifestation of a disease process
3 month old
1 week tender facial swelling
Irritable
Very low platelet count
Kaposiform haemangioendothelioma
Response to vincristine
- 6 weeks old
- 2 weeks swelling right upper eye lid
- Haemangioma of infancy
- Good response to propranolol
14 weeks of age
Red raised areas – come and go
Blisters

Cutaneous mastocytosis
- 5 week old
- peri-anal rash from 2 weeks of age
- Bleeding over past week
- Haemangioma of infancy
Factitious?

Inflicted

Other

Who

How

FII?

Self

Why

How

Dermatitis artefacta?
Fabricated or induced illness (FII)

- a carer, usually the mother, fabricates illness in a child either by inducing physical signs of illness or by deliberately misleading the doctor into believing that the child is ill - DOH 2001

First described 1977 by Dr Meadows know at the time as Munchausen’s Syndrome by Proxy

Skin: Ulcers, blisters, erosions, non-healing of surgical sites, loss of nails.
Fabricated or induced illness (FII)

- McClure at al 1996
  - 2 yr prospective study UK & Ireland
    - 128 cases
    - annual incidence under 16 yr: 0.5 per 100,000
    - 77% under 5yrs
    - 6% children died as a direct result of FII
    - 12% required intensive care
    - 12% had a sibling who died previously

- Other studies
  - 10% of children with FII die
  - 50% experience long-term morbidity
Self Induced (factitial; dermatitis artefacta)

- Usually > 8 years of age
- Self induced but patients deny any role in causation
- Strongly associated with eating disorders
- Mostly females during and after adolescence
- Immature personality
Self Induced (factitial; dermatitis artefacta)

- Hollow history
- No knowledge of how the lesions appear
- No evolution ‘just appear’
- Mona Lisa smile
- Distinct from self cutting
- Parents anxious, angry and frustrated with failure of doctors to diagnose and treat successfully
Examination

- Bizarre shapes
- Odd distribution
- Accessible sites
- Blisters, excoriations, erosions, abrasions, bruises, purpura, erythema, burns
- Upper limbs initially in more than 50%, face later
mechanisms

- Deodorant spray: bullae
- Garlic: erythema, vesicles
- Hair straighteners: Linear erythema, blisters
- Suction Cup: blisters, purpura
- Caustics: erythema, blisters
Self-induced (factitial)

- Dissociative
  - Disruption in the usually integrated functions of consciousness, memory, perception
  - May be a feature of PTSD and eating disorder

- Immature personality

- Sexual abuse

- Loss of parent
What to do

- Detailed history
- Assess overall appearance of the child
- Undress fully
- Measure height and weight (growth chart)
- Observe interaction
- Discuss
CHILDREN’S SKIN SYMPOSIUM
Saturday 15th November | The Royal Society of Medicine

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