BSPGHAN

working for children with digestive and liver disorders

British Society of Paediatric Gastroenterology Hepatology and Nutrition

ANNUAL MEETING 2015

Wednesday 28th – Friday 30th January 2015

Holiday Inn Hotel, Bridgefoot, Stratford-upon-Avon

Educational Grants provided by Principal Gold Sponsors:

Mead Johnson Nutrition

MSD

Nestlé Nutrition

and Platinum Sponsors:

Nestlé Health Science
NOURISHING PERSONAL HEALTH

Local Organiser: Professor Simon Murch - Consultant Paediatric Gastroenterologist

E&OE and CPD Approved

Thanks to all of our sponsors

"How far that little candle throws his beams! So shines a good deed in a naughty world."
William Shakespeare, The Merchant of Venice

On behalf of the organising committee, the Council and membership of BSPGHAN we would like to express our gratitude for the generous support of our sponsors for this 29th annual meeting of our society. This meeting, like its predecessors, could not take place in the way that it does without your kind and greatly valued support.

At a time when there is increasing scrutiny of links between medical professionals and the pharmaceutical and nutritional industries, it is important to recognise the potential benefits to patients that can accrue from interaction between medical societies attempting to improve the delivery of care and those companies that are in a position to refine current products and develop new ones. In our specialty, challenging new disorders are being identified and there appears a realistic potential to improve outcomes in long-recognised conditions through new-generation products. This meeting offers us the chance to let you know what we think are emerging opportunities to improve the care of children suffering gastroenterological, hepatological or nutritional problems and we are happy to share the meeting with you, hoping that you may enjoy it as much as we always do.

We are grateful to the charities and patient support groups that are attending and supporting our meeting. Your input has been hugely important over the years, and we hope that it may continue for many years to come.

We hope that all those who have come to support our meeting find the time and opportunity to sample something of the atmosphere of Stratford-upon-Avon and take away memories of a place and time that was a little special.

Professor Simon Murch
Consultant Paediatric Gastroenterologist
Chair of Local Organising Committee
BSPGHAN Annual Meeting 2015

Dr Girish Gupte
Consultant Paediatric Hepatologist
BSPGHAN Treasurer

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BSPGHAN
working for children with digestive and liver disorders
British Society of Paediatric Gastroenterology Hepatology and Nutrition

President's Welcome to the 29th annual BSPGHAN meeting 2015

Dear Friends and Colleagues

Each year at the BSPGHAN annual meeting we meet a new version of "Plus ça change....." in the best possible way. Unsurprisingly, the meeting reflects the nature of the society and its continuing development flavoured by the style of the host institution. We expect and always experience a combination of successful familiarity spiced with organisational innovations and blended by the esteemed host him or herself. And we enjoy it every time.

This year the familiar can be found in the format of the programme, the timetable, the combination of gastroenterology, hepatology and nutrition, the inclusive, pan-national multidisciplinary approach to learning, and of course the football. The innovative can be found in delegate participation with the PICO session and clinical case solving, twitter interactive input, focus on microbiome and introduction of the trip to the theatre, and our hosts' remarkable personal influence is evident in the pragmatic nature of the programme contents, the developing links with BSACI and the charm in the venue and the way the meeting has been put together.

I am therefore delighted to be welcoming you on behalf of BSPGHAN council to this 29th annual meeting. I would like to thank our host, Professor Simon Murch and his colleagues in the organising committee ably supported by Mrs Carla Lloyd for what will be another fantastic conference. Whatever our interests or specialty I am sure we will all massively enjoy and learn from it.

Best wishes

Alastair Baker
BSPGHAN President

Master of Ceremony
Wed 28.01.15 - Dr Rafeeq Muhammed
Thu 29.01.15 - Dr Girish Gupte
Fri 30.01.15 - Professor Simon Murch

Local Organising Committee:
Professor Simon Murch
Dr Rafeeq Muhammed
Dr Girish Gupte
Dr Fiona Cameron
Mrs Carla Lloyd

Education Committee:
Dr Rafeeq Muhammed,
Chair of BSPGHAN Education Group
Professor Simon Murch,
Chair of BSPGHAN Gastroenterology Group
Dr Julian Thomas,
Chair of BSPGHAN Research Group
Dr Fiona Cameron,
Chair of Trainee Members' Group
Ms Kay Crook,
Chair of Associate Members' Group

Poster Walk Judges:
Dr Nadeem Afzal
Professor Stephen Allen
Dr Protima Amon
Dr Marcus Auth
Professor Bim Bhaduri
Dr Hemant Bhavsar
Dr Fiona Cameron
Dr Mike Cosgrove
Ms Kay Crook
Mr Mick Cullen
Dr Ieuan Davies
Dr Suzanne Davison
Ms Claire de Koker
Ms Jackie Falconer
Ms Vikki Garrick
Dr Richard Hansen
Dr Paul Henderson
Ms Lindsay Hogg
Dr Subra Mahadevan-Bava
Dr Patrick McKiernan
Ms Rosan Meyer
Dr Prithvi Rao
Dr Lisa Whyte

Welcome address from the local organising committee

"Why with the time do I not glance aside
To new-found methods, and to compounds strange?"
William Shakespeare, Sonnet LXXVI

Welcome one and all to Stratford-upon-Avon for the 29th annual meeting of the British Society for Paediatric Gastroenterology, Hepatology and Nutrition. It is a special society and, I think, a special place to hold the meeting.

Our society appears to have had a vigorous early life but clearly tracked much lower on the centiles than it now does. As BSPGHAN approaches mid-year maturity it has grown and evolved, moving away from untrammelled tertiary centre specialism to enfranchise paediatricians with a special interest, doctors in training and most importantly the Associate Membership with their numerous individual specialisations and skills. The burgeoning size of our society brings great advantages, including an increasing national presence and influence, but also provides challenges – not least in arranging a meeting that can provide a meaningful learning experience for delegates of very different background and clinical interest. We have thankfully so far avoided the balkanisation that afflicts larger meetings, with myriad individual sessions running in parallel and with little true shared experience. It does however demand generosity of spirit amongst delegates and a willingness to expose themselves to some learning experiences they would not ordinarily have sought. I hope that the programme offers sufficient variety and balance that all who enter into the spirit of the meeting will learn more than they might have anticipated and go away further enthused by the breadth by our great specialty. The speakers you will hear have a shared characteristic of being great communicators and there is little doubt that you will be entertained as well as educated.

We have met in many lovely places over the years and our meetings have always managed to blow away the post-Christmas January blues in a memorable way. I am so pleased that you have come to the beautiful and historic town of Stratford-upon-Avon. Those of you who manage to attend the RSC for the performance of the Midnight Truce are in for a real treat. The newly-refurbished RSC is simply the loveliest theatre you could wish for, and I would suggest it is well worth a visit by those who are unable to attend the performance. There are also numerous characterful places to visit for shopping, eating or drinking within easy walking distance.

The organising committee have supported me so much in shaping this meeting. Rafeeq has done a huge amount of work behind the scenes and has always been there to help out when problems arose. Lastly, but absolutely not least, we have been hugely indebted to Carla for her boundless support, tireless enthusiasm and wise guidance in shaping this event. The jury may be out whether we have kind hearts but she has run through fire and water on our behalf.

Let the curtain rise…

Wednesday 28th January 2015

Holiday Inn, Stratford-upon-Avon

8.00
REGISTRATION DESK OPENS

Postgraduate Day: 10.20 – 12.15

Session 1

10.20 – 10.30

Welcome and Introduction
Dr Alastair Baker, President BSPGHAN
Consultant Paediatric Hepatologist
King's College Hospital, London

Chairs:
Dr Marcus Auth - Consultant Paediatric Gastroenterologist
Alder Hey Children's Hospital, Liverpool
and
Dr Fiona Cameron - Specialist Registrar
Royal Hospital for Sick Children, Glasgow

10.30 – 10.50

Hypochondriasis by Proxy: managing the anxious internet-influenced family
Dr Karen McLachlan
Consultant General Paediatrician and
Named Doctor for Child Protection
University Hospital of Coventry and Warwick NHS Trust
Also Acting Designated Doctor of Coventry and Rugby CCG

10.50 – 11.45

Clinical problem solving cases with audience participation - keypad voting session
Dr Rafeeq Muhammed
Consultant Paediatric Gastroenterologist
Birmingham Children's Hospital
Birmingham

11.50 – 12.15

Getting a paper published
Dr Mark Beattie
Consultant Paediatric Gastroenterologist
Southampton General Hospital
Southampton

12.15 – 13.15
LUNCH AND POSTER VIEWING
(Opportunity to meet with the sponsors)

13.15 – 14.35

Session 2

Problem areas

Chairs:
Dr Franco Torrente - Consultant Paediatric Gastroenterologist
Addenbrooke Hospital, Cambridge
and
Mr Mick Cullen - Paediatric Gastro Nurse Specialist
Southampton General Hospital, Southampton

13.15 – 13.35

Management of a child with dysphagia
Dr Mike Thomson
Consultant Paediatric Gastroenterologist
Western Bank
Sheffield

13.35 – 13.55

Optimising nutrition in paediatric Inflammatory Bowel Disease
Dr Tony Wiskin
Specialist Registrar
Southampton General Hospital
Southampton

13.55 – 14.15

Nursing the child needing parenteral nutrition – from hospital to home
Ms Catriona McDonald
Paediatric Nutrition Clinical Nurse Specialist
Addenbrooke's Hospital
Cambridge

14.15 – 14.35

Weaning children off parenteral nutrition – tricks of the trade
Dr Susan Hill
Consultant Paediatric Gastroenterologist
Great Ormond Street Hospital
London

14.35 – 14.55
TEA
(Opportunity to visit exhibitor stands)

Session 3

14.55 – 15.35

How do I do it?

Chairs:
Professor Bim Bhaduri - Consultant Paediatrician
Maidstone Hospital, Kent
and
Ms Sarah Macdonald
Great Ormond Street Hospital, Great Ormond Street, London

14.55 – 15.15

Medical management of a child with severe constipation
Dr Marcus Auth
Consultant Paediatric Gastroenterologist
Alder Hey Hospital
Liverpool

15.15 – 15.35

Low-grade transaminitis – ignore or do more?
Dr Dino Hadzic
Consultant Paediatric Hepatologist
Kings College Hospital
London

Session 4

15.40 – 16.10

Abstract session

Chairs:
Dr Rafeeq Muhammed - Consultant Paediatric Gastroenterologist
Birmingham Children's Hospital, Birmingham
and
Dr Lucy Howarth - Consultant Paediatric Gastroenterologist
John Radcliffe Hospital, Oxford

15.40 – 15.50

Ready-to-use therapeutic food with balanced essential fatty acid profile, with or without fish oil, to treat severe acute malnutrition: a randomized controlled trial
Presenter: Dr Kelsey Jones, ICH

Jones, Kelsey DJ. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya; Centre for Global Health Research and Section of Paediatrics, Imperial College, London, UK; Ali, Rehema. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Khasira, Maureen A. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Odera, Dennis. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya; West, Annette L. Faculty of Medicine, University of Southampton, Southampton, UK; Koster, Grielof. Faculty of Medicine, University of Southampton, Southampton, UK Akomo, Peter. Valid Nutrition, Bantry, Republic of Ireland; Talbert, Alison WA. KEMRI-Wellcome Trust Research Programme, Kilifi, Keny; Goss, Victoria M. Southampton National Institute of Health Research Respiratory Biomedical Research Unit, Southampton, UK; Ngari, Moses. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Thitiri, Johnstone. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Ndoro, Said. Kilifi County Hospital, Ministry of Health, Kilifi, Kenya; Knight Garcia, Miguel. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya; Centre for Tropical Medicine & Global Health, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK; Omollo, Kenneth. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Ndungu, Anne. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Mulongo, Musa M. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya Bahwere, Paluku. Valid International, Oxford, UK; Fegan, Greg. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya; Centre for Tropical Medicine & Global Health, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK; Warner, John O. Centre for Global Health Research and Section of Paediatrics, Imperial College, London, UK Postle, Anthony D. Faculty of Medicine, University of Southampton, Southampton, UK; Collins, Steve. Valid Nutrition, Bantry, Republic of Ireland; Valid International, Oxford, UK Calder, Philip C. Faculty of Medicine, University of Southampton, Southampton, UK; Berkley, James A. KEMRI-Wellcome Trust Research Programme, Kilifi, Kenya; Centre for Tropical Medicine & Global Health, Nuffield Department of Clinical Medicine, University of Oxford, Oxford, UK

15.50 – 16.00

Blinded enteral feeding trials – shedding light on complex presentations
Kelly Larmour; Shergill-Bonner, Rita: Lowes, Kathryn: Holmes, Jayne: Landy, Niamh: Anna-Claire: Macdonald Sarah
Dietetics, Great Ormond Street Hospital for Children NHS Trust, London, UK

16.00 – 16.10

The composition of the gut microbiome in treatment naive children with Crohn's disease
Presenter: Dr Protima Amon, Blizard Institute
Amon, Protima[1]: Serena, Gloria[2]: Barakat, Farah[1]; Fasano, Alessio[2]:;Walker, Allan[2]: Wade, William[1]: Sanderson, Ian[1]:
[1]Blizard Institute, Queen Mary University London, UK
[2]Harvard Clinical Nutrition Research Centre, Massachusetts General Hospital, Boston, USA

16.15 – 17.15

Session 5

Factitious, induced illness and problem areas

Chairs:
Professor Simon Murch - Consultant Paediatric Gastroenterologist
University Hospital Coventry & Warwickshire, Coventry
and
Dr Peter Gillett - Consultant Paediatric Gastroentrologist
Royal Hospital for Sick Children, Edinburgh

16.15 – 16.35

The pathophysiology and causes of intestinal malabsorption
Dr Keith Lindley
Consultant Paediatric Gastroenterologist
Great Ormond Street Hospital, London

16.35 – 16.55

When to suspect factitious/induced gastrointestinal illness
Dr Sue Protheroe
Consultant Paediatric Gastroenterologist
Birmingham Children's Hospital

16.55 – 17.15

The ones we got wrong.
Dr Anna Pigott
Consultant Paediatric Gastroenterologist
City General Hospital
University Hospital of North Staffordshire, Stoke-on-Trent

And

Dr Marcus Auth
Consultant Paediatric Gastroenterologist
Alder Hey Children's Hospital, Liverpool

17.30 – 18.30

Working Group Meetings

Associate Members
Trainee Members
IBD Nurses Group

18:45-20:00

Football Challenge

19:00 – 22:30

RSC performance - The Christmas Truce

Informal Ice Breaker Dinner

18:00 onwards
Dinner in the Carvery

21.00 - late
Entertainment in G's Bar
with Cyril and Roxanne

Thursday 29th January 2015

Holiday Inn, Stratford-upon-Avon

7.45 - 9.00
Working Group Meetings

Nutrition Working Group
Hepatology
Motility/pH impedance
Coeliac Group
Endoscopy

08.55 – 09.00

Welcome and Introduction

Professor Simon Murch - Consultant Paediatric Gastroenterologist
2015 Local Meeting Organiser

09.00 – 10.00

Satellite session – Nestle

The role of protein in early nutrition for long term health
Professor A Singhal
Institute of Child Health, 30 Guilford Street, London WC1N 1EH

10.00 – 11.30

Session I - Hepatology

Chairs:
Dr Girish Gupte - Consultant Paediatric Hepatologist
Liver Unit, Birmingham Children's Hospital, Birmingham
and
Ms Sarah Tizzard - Specialist Liver Nurse
Paediatric Liver Centre, King's College Hospital, London

10.00 – 10.30

The gut-liver axis in inflammatory liver disease
Professor David Adams
Consultant Hepatologist
University of Birmingham
Edgbaston
Birmingham

10.30 – 11.00

Managing IBD Associated Liver Disease
Dr Patricia McClean
Consultant Hepatologist
Leeds General Infirmary

11.00 – 11.30

Key Note talk

Chair:
Dr Suzanne Davison - Consultant Paediatric Hepatologist,
Leeds General Infirmary. Chair of Hepatology group.

Improving long term outcomes for paediatric liver transplant recipients
Professor Deirdre Kelly
Professor of Paediatric Hepatology
Liver Unit
Birmingham Children's Hospital
Birmingham

11.30 – 12.00
TEA
(Opportunity to visit exhibitor stands)

12.00 – 12.50

Guest Lecture

Chair:
Professor Ian Sanderson - Consultant Paediatric Gastroenterologist
Royal London Hospital, London

12.00 – 12.20

The microbiome – the forgotten organ goes centre-stage.
Professor Fergus Shanahan
Alimentary Pharmabotic Centre (Apc)
Medicine Department
University College Cork
BioSciences Building
University College Cork
Ireland

12.50 – 13.30

Session III - PICO presentations

Chairs:
Dr Julian Thomas - Consultant Paediatric Gastroenterologist
Victoria Infirmary, Newcastle upon Tyne
and
Professor Stephen Allen - Consultant Paediatric Gastroenterologist
Royal Professor of Paediatrics/Honorary Consultant Paediatrician
Liverpool School of Tropical Medicine

PICO presentations

To compare outcomes in children with UC using Methotrexate or standard treatment in cases with steroid dependence or intolerance to or failure to respond to standard treatment.
Dr Dharam Basude, Consultant Paediatric Gastroenterologist
Bristol Royal Children's Hospital
Upper Maudlin Street
Bristol

Advances in diagnosis of paediatric gastro-oesophageal reflux disease
Dr Kornilia Nikaki
Wingate Institute of Neurogastroenterology
Queen Mary University of London

To establish the best method for weaning infants with short bowel syndrome from parenteral nutrition
Ms Sarah Macdonald
Great Ormond Street Hospital for Children
Great Ormond Street
London

Fish-oil based intravenous lipid emulsion (Omegaven®) as a rescue in septic infants with intestinal failure and with or at risk of developing liver disease
Dr Huey-Minn Lee
King's College Hospital
Denmark Hill
London

13.30 – 14.30

LUNCH AND POSTER VIEWING
Meet the sponsors

14.30 – 15.50

Session IV - Intestinal inflammation and growth

Chairs:
Dr John Fell - Consultant Gastroenterologist
Chelsea and Westminster Hospital, London
and
Ms Kay Crook - Paediatric Gastroenterology Nurse Specialist
St Mark's Hospital, Middlesex

14.30 – 14.50

The pathophysiology of impaired growth in inflammatory disorders
Dr Jeremy Kirk
Consultant Paediatric Endocrinologist
Birmingham Children's Hospital
Birmingham

14.50 – 15.30

Top tips for managing IBD – Twitter interactive cases
Drs Richard Russell and Richard Hansen
Consultant Paediatric Gastroenterologists
Royal Hospital for Sick Children
Glasgow

15.30 – 15.50

IBD genetic PhD
Dr Tracy Coelho
Locum Consultant Gastroenterologist/Clinical Research Fellow
Southampton General Hospital
Southampton

15.50 – 16.20

TEA
(Opportunity to visit exhibitor stands)

Session V - Plenary Abstract Session 1

Chairs:
Dr Christine Spray - Consultant Paediatric Gastroenterologist
Bristol Royal Hospital, Bristol
and
Dr Christos Tzivinikos - Specialist Registrar
Royal London Hospital, London

16.20 – 16.30

Challenges in the management of paediatric non-alcoholic fatty liver disease: a longitudinal follow-up study.
Presenter: Dr Jake Mann, Hitchingbrooke Hospital
Mann, Jake P[1]; Armstrong, Matthew J[2]. Sewel, Peter[3]: Rajwal, Sanjay[3].:McClean, Patricia:
[1]University of Cambridge, Cambridge; [2] Centre for Liver Research, Birmingham; [3]Leeds General Infirmary, Leeds

16.30 – 16.40

Outcome of live versus deceased donor liver transplantation in infants with biliary atresia
Presenter: Dr Shiv Tibrewal, Leeds
Tibrewal[1], Shiv: Davison[1], Suzanne: Prasad , Raj[2]: Attia, Magdy[2]: Hidalgo, Ernest[2]:
Alizai, Naved[2]:Rajwal, Sanjay[1]: McClean, Patricia[1]:
[1]Children's Liver Unit, Leeds Teaching Hospitals NHS Trust, Leeds.
[2]Department of Hepatobiliary Surgery and Transplantation, Leeds Teaching Hospitals NHS Trust, Leeds.

16.40 – 16.50

Transjugular intrahepatic portosystemic shunt (TiPPS) insertion for the management of portal hypertension in children: a single centre experience
Presenter: Dr Lauren Johansen, Birmingham
Johansen, Lauren[1]: McKiernan, Patrick[1]: Kelly, Deirdre[1]: Sharif, Khalid[1]: Lloyd, Carla[1]:
Olliff, Simon[2]: Mangat, Kam[2]: John, Philip[1]: McGuirk, Simon[1]:
[1]Birmingham Children's Hospital, Birmingham; [2]Queen Elizabeth Hospital, Birmingham

16.50 – 17.00

Why can't we implement Hepatitis B vaccination policy: A retrospective regional review
Presenter: Dr Mona Abdel-Hady, Birmingham
Abdel-Hady M[1], Tahir M[2], Gowen H[1], Velander N[3], Ismail KM[4], Kelly DA[1]
[1]Liver Unit, Birmingham Children's Hospital, Birmingham; [2]Public Health England, West Midlands, Birmingham; [3]Statistics, Modelling and Economics Department, Public Health England, Colindale, London; [4]School of Clinical & Experimental Medicine,University of Birmingham

17.00 – 17.10

Anti-Tumour Necrosis Factor Drug Monitoring in Paediatric Inflammatory Bowel Disease
Presenter: Dr Lisa Whyte, Birmingham
Whyte Lisa[1]: Bignell Michelle[2]: Wong Theodoric[1]: Protheroe Susan[1]: Bremner Ronald[1]: Haller Wolfram[1]: Muhammed Rafeeq[1]:
[1]Department of Gastroenterology, Birmingham Children's Hospital, Birmingham, UK; [2]Department of Biochemistry, Birmingham Children's Hospital, Birmingham UK

17.10 – 17.20

A 17-year prospective cohort study of paediatric inflammatory bowel disease patients diagnosed less than 10 years of age (Paris A1a)
Presenter: Dr Paul Henderson, Edinburgh
Henderson, Paul[1]: Rogers, Pamela[2]: Wilson, David C[1]:
[1]University of Edinburgh, Edinburgh; [2]Royal Hospital for Sick Children, Edinburgh

17.30 – 19.00

Annual General Meeting

20.00

Gala Dinner

Entertainment with
The Birmingham Blues Brothers
And dancing till early hours

Friday 30th January 2015

Holiday Inn, Stratford-upon-Avon

7.45 - 9.00
Working Group Meetings

Research
PeGHAN
Education
Gastroenterology
e-BANS

9.00 – 10.40

Session VI - Nutrition in collaboration with the Neonatal Nutrition Group

Chairs:
Dr Richard Hansen - Consultant Paediatric Gastroenterologist
Royal Hospital for Sick Children, Glasgow
and
Dr Alison Bedford Russell - Consultant Neonatologist
Birmingham Women's Hospital, Birmingham

09.00 – 09.20

Links between the microbiome and nutrition in the infant
Dr Nicholas Embleton
Consultant in Neonatal Medicine
Associate Clinical Lecturer
Newcastle Neonatal Service
Newcastle upon Tyne

09.20 – 09.40

Does mucosal inflammation contribute to malnutrition and stunting in resource-poor countries?
Dr Kelsey Jones
Imperial College London/ KEMRI-Wellcome Trust
Kilifi, Kenya

9.40 – 10.00

The many faces of milk allergy in young infants
Dr Rosan Meyer
Honorary Senior Lecturer
Imperial College, Principal Paediatric Principal Research Dietitian
Great Ormond Street Hospital for Children
London

10.00 – 10.40

State of the art lecture
How does breastfeeding protect against necrotising enterocolitis?
Professor Per Sangild
Department of Human Nutrition
University of Copenhagen
Denmark

10.40 – 11.10
TEA
(Opportunity to visit exhibitor stands)

11.30 – 12.55
Session VII

Chairs:
Dr Alastair Baker - Consultant Paediatric Hepatologist
King's College Hospital, London
and
Dr Andrew Barclay - Consultant Paediatric Gastroenterologist
Royal Hospital for Sick Children, Glasgow

11.10 – 11.30

Blended foods for tube-fed children – a safe and realistic option?
Professor Jane Coad
Professor in Children and Family Nursing
and Director of the Centre for Children and Families Applied Research
Coventry University

11.30 – 11.50

Optimising enteral nutrition to prevent malnutrition in the child with chronic liver disease
Ms Sara Mancell
Lead Children's Dietitian
Nutrition & Dietetics Dept
King's College Hospital NHS Foundation Trust
Denmark Hill
London

11.55 – 12.55
Session VIII - Plenary abstract session 2

11.55 – 12.05

The epidemiology and outcome of biliary atresia in Scotland 2002-2013
Presenter: Dr Paul Henderson, Edinburgh
Henderson, Paul[1]: Sutton, Emma[2]: Tayler, Rachel[3]: [3]Leeds Children's Hospital, Leeds; Hansen, Richard[1]: Barclay, Andrew[2]
[1]Royal Hospital for Sick Children, Glasgow; [2]University of Glasgow, Glasgow; [3]Leeds Children's Hospital, Leeds
On behalf of the Scottish Society of Paediatric Gastroenterology, Hepatology and Nutrition.

12.05 – 12.15

Acute upper gastrointestinal bleeding in childhood: development of the Sheffield scoring system to predict need for endoscopic therapy.
Presenter: Dr Mike Thomson, Sheffield
Thomson, Mike[1]: Campbell, David[1]: Narula, Priya[1]: Rao, Prithviraj[1]: Urs, Arun[1]:
[1]Sheffield Children Hospital, Sheffield

12.15 – 12.25

Should the new ESPGHAN guidelines on diagnosing Coeliac Disease also apply to asymptomatic children ?
Presenter: Dr Siba Paul, Bristol
Dr Siba Prosad Paul[1], ST7 in Paediatric Gastroenterology; Prof Bhupinder Sandhu[1], Consultant in Paediatric Gastroenterology
[1]Bristol Royal Hospital for Children

12.25 – 12.35

Biological therapy for Paediatric Inflammatory Bowel Disease in 524 patients: results from the 2014 UK paediatric biologics audit
Presenter: Ms Kajal Mortier, Royal College of Physicians
Mortier, Kajal, Royal College of Physicians, London; Merrick, Victoria M, University of Edinburgh, Edinburgh; Evans, Hannah, Royal College of Physicians, London; Muhammed, Rafeeq, Birmingham Children's hospital, Birmingham; Auth, Marcus, Alder Hey Children's Hospital, Liverpool; Elawad, Mamoun, Great Ormond St Hospital, London; Fell, John ME, Chelsea and Westminster Hospital Children's Services, London; Beattie, Robert M, Southampton Children's Hospital, Southampton; Loganathan, Sabarinathan, North-East Scotland Paediatric Gastroenterology Network (Royal Aberdeen Children's Hospital, Tayside Children's Hospital and Raigmore Hospital Combined), Scotland; Torrente, Franco, Addenbrooke's Hospital (paediatric gastroenterology), Cambridge Morris, Mary-Anne, Jenny Lind Children's Hospital, Norwich; Charlton, Charlie, Nottingham Children's Hospital, Nottingham; Croft, Nick M, Barts and The London Children's Hospital, London; Rodrigues, Astor, Children's Hospital, The John Radcliffe, Oxford; Furman, Mark, Royal Free Hospital, Centre for Paediatric Gastroenterology, London Vadamalayan, Babu, King's College Hospital (paediatric gastroenterology), London Jenkins, Huw, The Noah's Ark Childrens Hospital for Wales, Cardiff; Puntis, John, Leeds General Infirmary (paediatric gastroenterology), Leeds Mitton, Sally, St George's Hospital (paediatric gastroenterology), London Chong, Sonny, Queen Mary's Hospital for Children, Surrey; Cosgrove, Mike, Morriston Hospital (paediatric gastroenterology), Swansea Akobeng, Anthony, Royal Manchester Children's Hospital, Manchester Wilson, David C, Royal Hospital for Sick Children, Edinburgh; Russell, Richard K, Royal Hospital for Sick Children (Yorkhill), Glasgow

12.35 – 12.45

Role of nutritional support team in improving outcomes in neonatal intestinal failure
Presenter: Dr Akshay Batra, Southampton General Hospital
Akshay Batra[1]; LV Marino[1]; R.M. Beattie[1] ;Freya Pearson[1]
[1]Southampton University Hospitals NHS Trust, Southampton

12.45 – 12.55

What should we feed children who can't feed themselves?
Presenter: Ms Claire Sadlier, Cardiff
Sadlier, Claire1; Singleton, Kath[1]; Jenkins, Huw[1];
[1]University Hospital of Wales, Cardiff

13.00 – 14.00
LUNCH AND POSTER VIEWING
(Final Opportunity to visit Sponsor Stands)

14.00 – 15.40

Session IX - Food allergy – joint session with BSACI

Chair:
Dr Adam Fox - Consultant Children's Allergist and Clinical Lead
Guy's and St Thomas's Hospital, London

14.00 – 14.20

Food allergy – a multisystem disorder
Dr Helen Brough
Consultant in Paediatric Allergy, Guy's and St. Thomas' Hospital
Honorary Senior Lecturer in Paediatric Allergy, King's College London

14.20 – 14.40

Mast cells and Eosinophils
Professor Simon Murch
Consultant Paediatric Gastroenterologist
University Hospital Coventry & Warwickshire
Coventry

14.40 – 15.00

Developing an effective service for the child with complex allergies.
Dr Adam Fox
Consultant Children's Allergist and Clinical Lead
Guy's and St Thomas's Hospital
London

15.00 – 15.20

Diagnosing non-IgE-mediated food allergy.
Dr Neil Shah
Consultant Paediatric Gastroenterologist
Great Ormond Street Hospital
London

15.20-15.40

Weaning tactics for the infant with multiple food allergy
Ms Jackie Falconer
Nutrition & Dietetic Department
Chelsea & Westminster Hospital
London

15.40 – 15.50

PRIZE PRESENTATION AND CLOSE OF MEETING

Dr Alastair Baker
Consultant Paediatric Hepatologist
President BSPGHAN

and

Dr Rafeeq Muhammed
Consultant Paediatric Gastroenterologist
Chair BSPGHAN Education Group

Previous Prize winners

2008 Southampton
Alex Mowat Prize – Dr Andrew Barclay
Best Abstract Presentation – Ms Elaine Buchanan
Best Presentation – Dr Sherina Ross

2009 Sheffield
Alex Mowat Prize – Dr Johann van Limbergen
Sean Devane Memorial – Dr Jenny Epstein
Best Allied Health Professional – Ms Jackie Falconer

2010 Liverpool
Alex Mowat Prize – Dr Emer Fitzpatrick
Sean Devane Memorial – Dr Rachael Taylor
Best Poster Presentation – Dr Paul Henderson

2011 Edinburgh
Alex Mowat Prize – Dr Paul Henderson
Sean Devane Memorial – Dr Emer Fitzpatrick
Best Poster Prize – Ms Helen French

2012 Nottingham
Alex Mowat Prize – Mark Goddard
Sean Devane Memorial – Anna Gregory
Challenging Case – Lisa Whyte
Best Poster – Ms Hannah Williamson

2013 Manchester
Alex Mowat Prize – Dr Protima Amon
Sean Devane Memorial – Dr Lisa Whyte
Best Poster Prize – Dr Rana Bitar

2014 - London
Alex Mowat Prize – Dr Vandana Jain
Sean Devane Memorial – Dr Ed Giles
Best Poster Prize – Dr Bradley Keller

BSPGHAN 2015 Annual Meeting

WEDNESDAY 28TH JANUARY
Invited speaker
biographies, abstracts
and notes pages

Dr Karen McLachlan
Consultant General Paediatrician and
Named Doctor for Child Protection
University Hospital of Coventry and Warwick NHS Trust

Also Acting Designated Doctor of Coventry and Rugby CCG

This session will be a short overview of the child protection issues that can arise in children with gastrointestinal conditions and the ways in which Parents may present their children for medical attention inappropriately.

Dr Rafeeq Muhammed

Rafeeq has graduated from India and has been trained in gastroenterology units in Newcastle upon Tyne, Cardiff and Birmingham before accepting the consultant job in Birmingham Children's Hospital. Rafeeq's main interests include inflammatory bowel disease, immunology and education. Rafeeq is the chair of BSPGHAN education committee from 2013.

Getting Published: Selling the Message

Dr RM Beattie
Editor in Chief
Archives of Disease in Childhood

Most journals receive many more submissions than they are able to publish and papers are therefore selected on a combination of scientific content and relevance to the readership (often called priority). The environment is therefore competitive.
This means getting papers published is about getting the message across and many potential papers fail to get published because the message is unclear/poorly presented.
It is important to remember that journals will publish papers that are important and present novel findings that will influence further research/clinical practice or will be widely read (preferably both).
The message therefore needs to be clear.

If you want your paper published

- Make sure you have a message
- Be clear in your own mind what the message is
- Write for the reader not for yourself
- Write clearly and logically
- Consider at the start ….
  - What is the (clinical or other) bottom line of your paper– if someone reads your paper what is the take home message

Remember there is only a limited amount of time to read the increasing amount of material published. The message needs to be interesting and relevant.
Remember fewer words usually mean more clarity.
Remember to read the instructions for authors.

Remember abstracts need data.

Remember tables and figures should be well presented, clearly labelled and relevant.

This session will cover

- Why do journals want your article
- Journal Processing
- What do Editors want
- How to write a paper
- Writing style
- Structured Discussion
- Reasons for Rejection
- Changing world of Publishing

Further Reading

How to get your paper rejected. BMJ 2004;329:1469
Docherty M, Smith R. The case for structuring the scientific discussion of papers. BMJ 1999;318:1224-5
Shelton JR, Edwards SJ. The function of the discussion section in academic medical writing. BMJ 2000;320:1269-70

Dr Mike Thomson

Dr Tony Wiskin

Optimising nutrition in childhood IBD

I am a final year sub-specialty trainee in paediatric gastroenterology scheduled to take up a consultant post in Bristol in the spring. I am interested in the interplay between nutrition, growth and disease in childhood. I completed my PhD examining nutrition in childhood Crohn's disease at the University of Southampton in 2013.

The adoption of biologic agents in the management of IBD has made an impact on how we treat inflammation in children with Crohn's and Ulcerative Colitis. However, despite improvement in measures of inflammation and a greater reliance on mucosal assessment to demonstrate remission, long term linear growth and final adult height outcomes remain relatively unchanged. Control of inflammation is a fundamental aspect of management to permit weight gain and for the majority of patients clear management algorithms can be followed to achieve remission.

However, specific guidelines for nutritional management which may improve long term growth and possibly disease outcomes are rare. One of the limitations to this is the scarcity of research about what specific nutrition support to give, in what quantity and at what time. The availability of parenteral iron preparations with improved side effect profiles has led to several studies exploring how to manage anaemia and has highlighted that large numbers of patients with IBD have evidence of iron deficiency or lack of iron supply to support erythropoiesis. This volume of research does not exist surrounding what would appear a simpler question of what are the nutritional demands of children with IBD.

During a series of research studies I have assessed both resting energy expenditure and physical activity and have demonstrated energy requirements are not raised in children with IBD compared to healthy children. However it is known from clinical observation that nutrient intakes in children receiving Exclusive Enteral Nutrition (EEN) can be well above estimated requirements. The natural conclusion is that the additional energy intake will be deposited as tissue, which should equate to weight and height gain. If children do not perform sufficient physical activity or obtain the right balance of nutrients there is a risk that adipose tissue will be gained in preference to lean – persistent lean deficits are a long term problem in children with IBD. In a longitudinal study I demonstrated that while on EEN children gained both lean tissue and adipose but that this stopped on resumption of normal diet and was not clearly associated with detectable disease relapse. Higher ratios of EEN intake:REE were associated with greatest change in weight but no association was found between dietary intake and height gain. In cross-sectional study appetite satiety and meal frequency are associated with anthropometric state suggesting that on free diet children fail to maintain sufficient intake to maintain catch up growth. Strategies to improve nutrient intake and provide an appropriate balance of nutrients may lead to improved catch-up growth alongside control of disease.

Catriona Mc Donald
BA (Hons), RN Child

Lead Paediatric Nutrition Clinical Nurse Specialist

I graduated from The Buckinghamshire College of Brunel University in 1998 as an RN Child. My interest in nutrition began during my training when I first cared for children with Cystic Fibrosis. Throughout the last 16 years of my career I have experienced the delivery of nutrition in its many different forms and in many different specialities.

My first post was at Basildon Hospital rotating between A&E and the Paediatric medical ward. I then moved to Harefield Hospital and cared for children who underwent heart surgery and organ transplantation

In 2001 I came to Addenbrooke's hospital and worked in the Paediatric Intensive Care (PICU) for 8 years where I gained my PICU qualifications and was promoted to Junior Sister. Within this environment I cared for children of varying ages who had been admitted from Oncology, Neurology, Neurosurgery, surgery and medical specialities all with varying nutritional needs.

From PICU I had a brief spell in the community as a School nurse promoting healthy eating, providing advice around diet and exercise and referring on to specialist dieticians and programmes.

I returned to Addenbrooke's in September 2010 to take up the new post of Lead Paediatric Nutrition Clinical Nurse Specialist. I have been in this post for the last four years developing the nursing service in Home enteral Nutrition, Parenteral Nutrition and Home Parenteral Nutrition (HPN). In the four years that I have been in post we have trained and discharged 7 patients and their families with HPN, one of which had a successful liver and small bowel transplant. I have also taken the opportunity to gain my non medical prescribing qualification to improve the service we provide both to in patients as well as our long term patients. We aim as a team to continue to improve the provision of Enteral and Parenteral Nutrition to children, not only within the hospital, but also around the region – liaising with local teams to keep the care of children as close to home as possible

Weaning children off parenteral nutrition – tricks of the trade

Susan Hill, Great Ormond Street Hospital for Children, London

Over the past 10-20 years the prognosis for patients treated with intravenous/parenteral nutrition (PN) has hugely improved. Infants and children with severe intestinal failure (IF) can now survive throughout childhood and into adult life on PN. Prognosis is related to the underlying disease rather than the PN treatment.

However, whilst on PN the child is at risk of potentially life-threatening complications, such as fluid and electrolyte imbalances, other metabolic disorders, bloodstream infections, liver damage, and thrombo-embolic problems. It is essential to make every attempt to wean and withdraw PN at the earliest opportunity.

The four major stages in the use of PN are 1). Stabilise the patient, 2). Aim for appropriate weight gain – usually 'catch up' weight gain required 3). Maintain weight centile appropriate for patient's length/height 4). Withdraw/wean PN and institute enteral nutrition(EN). Published evidence for how to wean is limited (see review references below). Practical weaning tips include:-

- Once child is clinically stable on PN, if intestinal function not rapidly improving, the underlying IF should be fully investigated and any appropriate treatment commenced.
- Nutrition team needed to manage weaning PN effectively

- Consider intestinal motility including gastric emptying, intestinal mucosal inflammation and length of small intestine when choosing feed type and method of administration.
- Liquid EN may be administered as continuous infusion or bolus feeds, ingested orally or given via an artificial feeding device, such as a nasogastric tube, naso-duodenal tube, gastrostomy, gastro-jejunostomy or jejunostomy
- Type of EN used maybe solid food, commercially prepared liquid enteral feeds (whole protein, hydrolysate, amino acid based) or modular feed made up in a specialist diet kitchen.

- introduce EN and continue alongside PN if at all possible. Major benefits of even minimal EN (e.g. 10ml bolus twice/day include prevention of intestinal mucosal atrophy, maintenance of entero-hepatic circulation and if given orally, retention of feeding skills.
- Increase concentration of EN before volume
- Gradual increase of EN volume (e.g. 1ml/kg/24 hours initially) often needed
- Give maximum EN tolerated without unacceptable exacerbation of symptoms such as vomiting, diarrhoea or abdominal pain.
- In patients with intestinal mucosal inflammation (congenital/acquired enteropathy, SBS) hydrolysate or amino acid feed usually tolerated better than whole protein. May tolerate milk, egg, wheat, soya free solids (also low fruit in SBS).
- SBS patients may tolerate weaning onto solids with limited liquid

- Cycle PN early: may tolerate EN better when PN not infusing
- Wind down/reduce rate of PN infusion prior to stopping in neonate, infant and older child when needed
- Reduce number of infusions of PN at earliest opportunity; even infants may manage 24 hours off PN when about a third of nutritional requirements tolerated as EN
- If 6 days/week tolerated then reduce to 5 days. If poor weight gain increase nutrients in PN when infused.

- Check urine sodium regularly
- Enteral sodium supplement often required after night off PN

- High EN intake may be needed to thrive since most recovering IF patients will have persistent malabsorption

Weaning a child from PN to oral/enteral nutrition can be one of the most complex aspects of management. Even with extensive investigation and assessment of intestinal function, it is only certain whether a child can tolerate EN by reducing PN and increasing EN appropriately. If you don't succeed at the first attempt, try, try and try again.

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Olieman JF et al. Enteral Nutrition in Children with Short-Bowel Syndrome: Current Evidence and Recommendations for the Clin JAm Diet Ass 2010 110: 420-426
Barclay AR et al. Systematic review: medical and nutritional interventions for the management of intestinal failure and its resultant complications in children. Aliment Pharmacol Ther 2011:33: 175-184

Dr Marcus Auth

Medical management of a child with severe constipation

Marcus KH Auth, MD PD FRCPCH, Consultant in Paediatric Gastroenterology, Hepatology and Nutrition, Alder Hey Children's Hosp., Honorary Lecturer, Liverpool.

Childhood constipation affects > 10-20% of children in the UK. It accounts for 25-30% of consultations with paediatric gastroenterologists. 75-90% of children with constipation present with faecal incontinence as a result of retained, impacted faeces. This has significant implications for parental understanding, compliance, and drug adjustment. Successful management depends on recognition of common and exclusion of rare causes, explanation, individual tailoring of treatment, achieving compliance, and personalized continuity of care. Treatment principles are education, disimpaction, prevention of re-accumulation of faecal loading, and maintenance treatment. NICE recommends the use of PEG 3350 + electrolytes in an escalating dose regimen, and adding a stimulant laxative after 2 weeks if disimpaction is not effective, which includes senna, sodium picosulfate, bisacodyl, and docusate sodium. When appropriate prolonged medical therapy has been deemed a failure, rectal applications are indicated to prevent complications of a mega rectum, in the UK sodium citrate enema is recommended. PEG 3350 is also licenced in the UK as bowel cleansing solution, however it requires hospital admission, risk assessment for administration of a nasogastric tube, and strict control of the tube position and electrolyte control. It is important to provide advice on the expected time scale, safety, possible side-effects, and signs of under- or over-treatment of the medication. Chronicity can be debilitating and has behavioural and social consequences. More research is necessary to characterise efficient and optimal treatment for constipation in childhood. Evidence for constipation management, dosages for treatment, and references will be presented in the presentation, an extended summary is available in the online version of the Clinical Review article below :

Auth MKH, Vora R, Farelly P, Baillie C. Childhood constipation. BMJ. 2012 Nov 13;345:e7309. doi: 10.1136/bmj.e7309. PMID: 23150472

Biography:

Study of medicine at University of Frankfurt. Core training in general, visceral and transplant surgery at University of Frankfurt. Research fellow in Liver Unit at University Hospital Birmingham. MD (Frankfurt University) on immunological investigations of the human biliary ducts, and PD (Essen University) on preparation of cocultured human epithelial cells for clinical application. General paediatric training and paediatric gastroenterology, hepatology (including liver transplantation) and nutrition leading to consultant post at University of Essen. Since 2006 Consultant at Alder Hey Children's NHS Foundation Trust, Liverpool. Hosting BSPGHAN Annual Meeting in Liverpool 2010. Since 2014 leading the clinical GHN service at Alder Hey; clinical lead for IBD, polyposis syndromes, hepatology, video capsule endoscopy. 2013 clinical governance lead for medical specialties. Since 2011 local CSAC lead, and co-chair endoscopy steering group.

PI for trials in inflammatory bowel diseases (Develop, COLORS, GEM, PRED4, Biologics Homecare), coeliac disease (AbCD), eosinophilic oesophagitis (PEER), chronic viral hepatitis, pancreatitis (Europac-2), Working group member of BSPGHAN (IBD, coeliac, nutrition), ESPGHAN (Eosinophilic oesophagitis, hepatology, IBD), GPGE (coeliac).

Research and presentation awards from GPGE, GASL, ESOT, NASPGHAN, AASLD, Alex Mowat Prize. Major research grants from DFG and University of Essen for investigations on cell-matrix interactions in biliary atresia (in print).

Memberships in BSPGHAN, RCPCH, ESPGHAN, GPGE, Coeliac UK, CLDF.

Major publications in Hepatology, JPGN, BMJ, Acta Paediatrica, Liver International, Liver Transplantation, Paediatric Child Health, Klin Paediatrie (in print).

Since July 2014 BSPGHAN. Gastroenterology Chair

Dr Dino Hadzic

Low-grade transaminitis – ignore or do more?

Abnormal biochemical markers of the liver function are not infrequent in children with gastrointestinal problems. To investigate them, a holistic paediatric approach is advisable as sometimes they could indicate non-hepatic pathology, including neuromuscular or haematological problems. The commonest gastrointestinal conditions associated with genuine liver derangements are parenteral nutrition, coeliac disease and inflammatory bowel disease. The histology of liver disease caused by parenteral nutrition is reminiscent of any chronic biliary pathology, while the two others have recognized associations with autoimmune liver disease. Early detection and timely hepatological work-up are important as the long-term management differs. Therefore, monitoring hepatic biochemical markers represents a mandatory element in the management of chronic gastrointestinal disorders.

Professor Dino Hadzic

Professor Hadzic is one of the senior clinicians at Paediatric Liver Service at King's College Hospital in London. His main research interests are biliary atresia, alpha-1 antitrypsin deficiency, metabolic liver-based conditions and immunology of liver disease.

BSPGHAN 2015 Annual Meeting

WEDNESDAY 28TH JANUARY
PLENARY ABSTRACTS

Ready-to-use therapeutic food with balanced essential fatty acid profile, with or without fish oil, to treat severe acute malnutrition: a randomized controlled trial

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Background:
Ready-to-use therapeutic foods (RUTF) are lipid-based pastes that are widely used to treat acute malnutrition. Current specifications for RUTF permit high, unbalanced n-6:n-3 polyunsaturated fatty acid (PUFA) ratios, without pre-formed long-chain n-3 PUFA. The objective of this study was to develop an RUTF with balanced PUFA composition and measure its impact, with and without dietary fish oil supplementation, on children's PUFA status during the treatment of severe acute malnutrition.

Methods:
We conducted a randomized controlled trial in children with severe acute malnutrition in rural Kenya. 60 children were randomized to one of 3 arms receiving i) standard composition RUTF; ii) RUTF with balanced PUFA composition; iii) RUTF with balanced PUFA composition plus fish oil capsules. Participants were followed-up for 3 months. The primary outcome was erythrocyte PUFA composition.

Results:
Docosahexaenoic acid (DHA) composition declined from baseline in the two arms not receiving fish oil. Erythrocyte long-chain n-3 PUFA composition was markedly higher for participants in the arm receiving fish oil than for those in the arms receiving balanced-PUFA RUTF or standard RUTF alone (omega-3 index median 9.2% (interquartile range 7.4-9.9) compared to 5.0% (4.7-5.6) and 4.2% (2.7-6.2), respectively, $p < 0.001$). Both the balanced-PUFA RUTF and fish oil capsules were acceptable to participants and carers, and there were no significant differences in safety outcomes.

Conclusions:
Requirements for LC-PUFA amongst children with SAM may not be met by current specifications for fatty acid profile of RUTF or by an RUTF with balanced PUFA composition but without pre-formed long-chain n-3 PUFA. The clinical and growth implications of revised formulations need to be addressed in adequately powered clinical trials.

Registered at clinicaltrials.gov NCT01593969. Funded by the Bill & Melinda Gates Foundation and The Wellcome Trust

Blinded enteral feeding trials – shedding light on complex presentations

Kelly Larmour; Shergill-Bonner, Rita: Lowes, Kathryn: Holmes, Jayne: Landy, Niamh: Anna-Claire: Macdonald Sarah
Dietetics, Great Ormond Street Hospital for Children NHS Trust, London, UK

Background:
Medical professionals working in gastroenterology often rely on parental reporting of symptoms to inform decisions about investigations and treatment. In some cases the severity of reported symptoms is greater than one might expect given the patient's investigation results. In these cases, it is our practice to admit patients for assessment and observation of symptoms before changing their dietary treatment. From the dietetic perspective, these might be patients who are said 'not to tolerate' enteral feeds, those who 'tolerate' only minimal enteral feed volumes or those who 'tolerate' only a particular type of feed. Enteral feed changes have previously been made in an open fashion whereby families and staff are aware of the changes that have been made. This open method could introduce bias on the part of staff or families, especially if the new feed has been tried previously. Over the past year, blinded enteral feed trials have been undertaken in five patients with parental consent as part of their clinical care. The aim was to compare the outcomes of blinded feed challenges to the outcomes of previous unblinded feed challenges. The challenge was a success if the feeding plan was changed and a failure if it was not.

Methods:
Inclusion criteria: patients who were due to be admitted for an enteral feeding trial (if dependent on parenteral nutrition, PN) or for change of enteral feed were selected by the dietitian. All patients selected had previously 'failed' at least one open feed challenge while under the care of Great Ormond Street Hospital or another centre. Symptoms reported with feeds included; abdominal pain, diarrhoea, constipation, abdominal distension, bilious gastrostomy losses and changes in behaviour.

Patients were admitted for a minimum period of 2 weeks. There was an initial observation period (minimum 48 hours) when patients were fed as per their usual plan to allow for collection of baseline data. After this period, the blinded feed trial was started. Daily observations were recorded throughout admission and were documented on dedicated forms. Two sets of observation forms were completed independently, one by nursing staff and one by families. The observations were compared at the end of the trial period.

The feeds were prepared in the Special Feeds Unit daily under dietetic supervision and were sent to the ward labeled 'Blinded feed'. Feed bottles were covered with foil to conceal changes in feed appearance. From a safety perspective, the composition of the feed was kept in a sealed envelope at the back of the medical notes in case it was required by medical staff.

Results:
Blinded feed trials have been conducted in 5 children (3 males) aged 2 to 17 years. In three cases the challenges were successful and changes were made to the patients' feeding plans that were previously said to have failed. The feeds on admission were amino acid feed (3), extensively hydrolysed feed (1) and fat free modular feed (1). Of the two that failed, in one case a referral to psychology was made and further medical investigations were cancelled. The second case was a child about whom safeguarding concerns had been raised. The blinded enteral feeding trial clearly demonstrated that the child could not be fed enterally, even with water. Further investigations were undertaken and a new diagnosis was made. This child was referred for home PN and safeguarding concerns were dismissed.

Conclusions:
Blinded enteral feed trials are a useful tool in gaining a better understanding of gastrointestinal symptoms and their causes in patients with a complex medical background. They may prevent unnecessary and burdensome interventions such as modular feeds and PN and can also allay safeguarding concerns.

The composition of the gut microbiome in treatment naive children with Crohn's disease

Amon, Protima[1]: Serena, Gloria[2]: Barakat, Farah[1]; Fasano, Alessio[2]:;Walker, Allan[2]: Wade, William[1]: Sanderson, Ian[1]:
[1]Blizard Institute, Queen Mary University London, UK; [2]Harvard Clinical Nutrition Research Centre, Massachusetts General Hospital, Boston, USA

Background:
Crohn's disease is a chronic inflammatory condition affecting the gut, with a predilection for the terminal ileum and caecum. The aetiology remains elusive, but is thought to be an interaction of genetic, immunological and environmental factors. The intestine harbours a complex microbiota, both free and adherent to the intestinal surface. Studies of the intestinal gut microbiota imply that an unbalanced, or dysbiotic, microbial community is associated with a dysregulated immune response. There is evidence which indicates changes in the composition and metabolism of the gut microbiota in subjects with Crohn's disease, suggesting that the gut microbiota plays a crucial role in the pathogenesis of intestinal inflammation (1, 2). The microbiota is therefore likely to play a role in the pathogenesis and propagation of intestinal inflammation of Crohn's disease.

Methods:
Seven children newly diagnosed with Crohn's disease were studied. Stool samples were obtained prior to any treatment. We also collected stool samples from a group of seven healthy children who were undergoing investigation to give us a greater understanding of the differences between children with Crohn's disease. The faecal microbiota was characterized by amplification and sequencing of the 16S ribosomal RNA gene V4 region using the Illumina Miseq platform. The data was analyzed by means of the mothur pipeline. Sequences were clustered into operational taxonomic units (OTUs) at a distance of 0.03.

Results:
Crohn's disease patients were found to have significantly reduced OTU richness compared to healthy subjects ($P<0.0002$, Student's t test), confirming previous studies showing reduced bacterial diversity in patients with Crohn's disease (3). Furthermore, the microbiome in Crohn's disease patients was significantly different from that of healthy children [$P=0.01$ (parsimony comparison of ThetaYC statistic)]. The relative abundances of bacterial genera differed greatly between healthy children and Crohn's disease patients. Healthy children had higher proportions of the genus Bacteroides than Crohn's disease patients, whilst the reverse was true for Prevotella and Escherichia / Shigella.

Conclusions:
We have demonstrated there are significant differences in the composition of the gut microbiome in newly diagnosed children with Crohn's disease compared with healthy controls. This may suggest that if changes in the microbiota are associated with health, future studies can try to maintain that microbiota, perhaps using probiotics and/or established and new treatments for Crohn's disease with the aim of achieving long term remission in children with Crohn's disease.

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3. Manichanh C, Rigottier-Gois L, Bonnaud E, et al. Reduced diversity of faecal microbiota in Crohn's disease revealed by a metagenomic approach. Gut 2006;55:205-21.

BSPGHAN 2015 Annual Meeting

WEDNESDAY 28TH JANUARY
Afternoon Invited speaker biographies, abstracts and notes pages

Dr Keith Lindley

The pathophysiology and causes of intestinal malabsorption
Keith J Lindley

Assimilation of enteral nutrition is dependent upon both digestion and absorption of ingested foods. These processes are dependent upon the integrity of the gastrointestinal epithelium and mucosa, the presence of normal gastrointestinal endocrine and exocrine secretions and an appropriate gastrointestinal neurohumoral environment. Deficiencies of these elements can be primary or secondary to other pathologies.

In the case of carbohydrates these processes involve digestion of polysaccharides to oligosaccharides through a mixture of intralumenal and brush border hydrolysis, hydrolysis of oligosaccharides to monosaccharides by brush border disaccharidases and the active absorption of glucose, along with sodium and water, utilising an electrochemical gradient or the passive absorption of fructose via a facilitator transporter.

Protein digestion is principally via intralumenal proteases/peptidases and active absorption as amino acids, di- and tri- peptides through specific transporters.

Fat absorption is dependent upon emulsification and intralumenal lipolysis. This is dependent upon normal motility, the presence of adequate amounts of bile salts and of lipases which function at the lipid / aqueous interface. Medium chain fatty acids released in this way can be absorbed paracellularly along with other water soluble nutrients utilising the bulk flow of liquids driven by the transcellular active transport of osmotically active molecules. Long chain fats are re-esterified within the epithelial cell and packaged with apo-proteins to reach the systemic circulation via the lymphatics.

Specific transporters exist for a wide variety of vitamins, minerals and nutrients.

Primary abnormalities of the above processes can arise through an absence of epithelial transporters and digestive enzymes, defects in epithelial assembly / integrity, defects in development of specific epithelial lineages (eg enteroendocrine cells) and defects in intra-epithelial trafficking. In addition primary disorders of intralumenal digestion can arise as a consequence of disordered bile flow and disordered pancreatic secretion.

Secondary disorders arise either through inflammation with or without disruption of epithelial and villus architecture and as a result of disordered motility. This is of course the largest group which will be encountered in clinical practice.

The talk will discuss normal GI absorptive physiology and illustrate the consequences of deficiencies of the elements of the absorptive system upon clinical manifestations of malabsorption.

Keith J Lindley

Consultant / Hon Reader in Gastroenterology at Great Ormond Street Hospital for Children and UCL Institute of Child Health since 1997. Currently Clinical Lead for diagnostic, interventional and therapeutic endoscopy and of the a Paediatric GI physiology investigative laboratory.

Over 100 publications in basic science and clinical paediatrics. Former convenor of the GI section of the Physiological Society, member of the ESPGHAN GI committee and (a very long time ago) council member of BSPGHAN !

When to suspect factitious/induced (F.I.I) gastrointestinal illness

Dr Sue Protheroe, Birmingham Children's Hospital.

"Any illness can be feigned; any symptoms can be falsified. Any medical test can be misleading or misinterpreted."

What is F.I.I?
Falsification, exaggeration or induction of a medical condition in a child is a medical diagnosis and responsibility for recognition lies with the Paediatrician. The common thread is that the doctor plays a role perpetuating the condition by failing to recognize the problem in a vulnerable child and causing inadvertent harm with a greater degree of invasive medical attention than is necessary. This may include endoscopy, surgical procedures, insertion of venous lines, artificial feeding, anesthesia or prolonged hospital admission. While most perplexing presentations to gastroenterologists do not have F.I.I, cases of factitious or induced feeding difficulties or failure to gain weight and unexplained diarrhoea rank among the more common presentations of F.I.I. Inducing signs or illness by poisoning, over medication, starving by active withholding or withdrawal of food, alleged allergies, blood loss or mimicking dysmotility syndromes may involve falsification of records or charts, interference with investigations or specimens, tampering with central venous lines, or enteral tubes. There can be a delay recognizing F.I.I since it is not uncommon for the child to have an underlying genuine medical condition such as gastro oesophageal reflux, chronic diarrhoea, vomiting, or have survived premature birth or an acute illness. Older children may support their parents/carer in the perplexing presentation, to the point of being complicit with deception.

What isn't F.I.I?
The definition of F.I.I excludes the cases of harm not caused by deception. Parental health care seeking for a child varies enormously and trying to reassure an unreassurable mother can be a challenge, especially withstanding pressure to continue to investigate. Our practice of working with parents and seeking "patient or parent satisfaction" can lead us to follow parent's requests to pursue medical care. However, if carers with extreme anxiety exaggerate symptoms and signs and encourage the doctor to pursue tests to rule out or treat any identifiable disorder (or to confirm a (false) belief about a child's ill health), then the child may still be suffering harm, although parents do not intend deception. To avoid "missing something" or "being wrong," paediatricians can favour testing to rise to the challenge to make a diagnosis as the "specialist" or to avoid complaints. An unsatisfied parent may demand a second, third, or fourth opinion, another test or diagnostic study, or even surgery to "find" the problem. Establishing as quickly as possible what is wrong when faced with a perplexing presentation and addressing parental concerns in another way rather than proceeding with potentially painful or harmful tests in certain circumstances can prevent escalation to F.I.I.
When to suspect F.I.I?
A cautionary flag might be raised if parental reassurance is unattainable and has been followed by an ever-accelerating trajectory of interventions that are not clearly clinically indicated.
The following features are not diagnostic of F.I.I but raise concern: -

- The diagnosis does not match the objective findings and there is an unlikely history of events or clinical features that are bizarre or do not make sense, perhaps with a discrepancy between the history, physical findings or investigations (eg lack of dehydration despite huge daily stool losses, inconsistent fluctuations in the condition, atypical organism in the central line blood culture), and

One or more of the following: -

- Reported symptoms and signs are unverifiable (eg pain only observed or appears when in presence of carer)
- Inexplicably poor response or non-compliance with treatment, unusual intolerance to those treatments or to multiple foods
- New symptoms reported on resolution of presenting problem (eg haematemesis followed by haematuria)
- Few normal daily activities or using aids beyond expected need
- Obstetric or birth history given is incorrect. (between 30% and 70% of those who falsify illness in children also falsify illness in themselves)
- Sibling has or had an unusual or unexplained illness or death
- Various opinions sought at different clinics and disputed by carer despite a definitive clinical opinion and investigations which might have been done to little or no avail, and child continues to presented for investigation and treatment with a range of symptoms.

In the case of severe motility disorders, children with factitious conditions are more likely to display

- Daily abdominal pain
- Accelerating disease trajectory
- Reported history of pre term birth
- Absence of dilated bowel on abdominal x ray
- Normal antroduodenal manometry
- No urinary tract abnormality.

A focus on the carers motive(s) isn't necessary for a diagnosis; there is no specific behaviour but high rates of early childhood adversity, unusual illness or somatization disorders are reported and 14-30% have professional ties to health care professions. Carer characteristics overlap considerably with those of caregivers who are advocates for their children with genuine illnesses, but reports describe carers who are either: -

A] Comfortable in the hospital, friendly with staff, emotionally strong and unusually calm despite medical setbacks, and receive admiration from medical personnel and other parents. The caregiver might not express relief or pleasure when told their child is healthy or does not have a particular illness or when told that child is improving and may seek another medical opinion. This friendly, supportive carer may publicly solicit sympathy or donations or benefits because of the child's rare illness. They can transition into a more hostile individual if doctors are seen as unresponsive or challenging to their concerns.

B] Another variant is the carer whose aims appear diametrically opposed to those of professionals. They may be demanding, confrontational, manipulative, and/or obvious in their lies. A carer may resist reassurance that child is healthy, perseverate about borderline abnormal results of no clinical relevance despite reassurance, insist on procedures or threaten to leave the system or litigate if requested tests are not received.

Challenges for clinical assessment
Separation of primary, secondary and tertiary care may contribute towards a delay in recognizing F.I.I. since transferring the patient away for an opinion to a Gastroenterologist may miss local social factors and medical continuity can be challenged particularly with the Consultant of the Week arrangement. Case notes are often cluttered or massive, and entries subjected to copy-and-paste behaviour carrying forward imprecise accounts, while electronic notes may give less opportunity to document personal observations or interactions. Overreliance on the history in gastroenterology, discomfort of suspecting a parent, (especially if this might prove unjustified), being uncertain when to mention suspicions, what to write, and what to say to parents can prevention concerns being pursued, especially when health outcomes are measured in terms of patient satisfaction and the Internet can be used to draw attention to doctors with whom families are not satisfied. Multiagency management according to the frameworks (below) are key in the crucial task of diagnosis and collaborating to keep children healthy and safe.

RCPCH guidelines Fabricated or induced illness by carers (F.I.I): a practical guide for paediatricians. London: Royal College of Paediatrics and Child Health, 2009
Child Protection Companion, 2nd ed. London: Royal College of Paediatrics and Child Health 2013.
http://www.rcpch.ac.uk/Policy/Child-Protection/Child-Protection Publications
HM Government. Department of Health guidelines on fabricated or induced illness by carers http://dcsf.gov.uk/everychildmatters/safeguardingandsocialcare/safeguardingchldren/safeguarding/
HM Government. Consultation process on Working together to safeguard children http://www.dcsf.gov.uk/consiultations/index.cfm?action-consultationDetails&consultation=1667&external=no&menu=1
NICE. When to suspect child maltreatment. NICE Clinical guideline 89.UK: NICE, 2009.
General Medical Council. Protecting children and young people: the responsibility of all doctors. London. GMC. 2012.
A normal gastrointestinal motility excludes chronic intestinal pseudoobstruction in children. Cucchiara S et al Dig Dis Sci. 2000 Feb;45(2):258-64.

Dr Anna Piggot and Dr Marcus Auth

The ones we got wrong

BSPGHAN 2015 Annual Meeting

THURSDAY 29TH JANUARY
Invited speaker
biographies, abstracts
and notes pages

Professor David Adams

David Adams is Professor of Hepatology and Dean of Medicine for the College of Medical and Dental Sciences at the University of Birmingham. He is also director of the Centre for Liver Research and the National Institute for Health Research (NIHR) Birmingham Liver Biomedical Research Unit and lead for translational research in the MRC Centre for Immune Regulation.

Professor Adams' clinical interests are transplant hepatology and autoimmune liver disease. His laboratory research interests are focused on mechanisms of immune-mediated liver disease. After initial training in hepatology in Birmingham he continued his immunology training with Dr Stephen Shaw at the Experimental Immunology Branch of the National Cancer Institute, Bethesda, USA before being appointed to the Chair of Hepatology in Birmingham in 1997. He was made a Fellow of the Academy of Medical Sciences in 2000.

He has a long-standing interest in understanding how leukocyte-endothelial interactions regulate the recruitment of effector cells in chronic liver disease and his group have defined molecular mechanisms used by hepatic endothelium to control the entry of leukocytes from the blood. They have recently begun to use this information to develop cell therapy for liver disease by targeting pathways involved in the recruitment of damaging effector cells or by promoting the recruitment of therapeutic cells including dendritic cells, stem cells and regulatory T cells that may be used to manipulate immune responses in patients in vivo.

Dr Patricia McCLean

Biography

Dr Patricia McClean MD, FRCP, FRCPCH is a consultant in paediatric hepatology and clinical lead for the Children's Liver Unit in Leeds Teaching Hospitals NHS Trust. She trained in Northern Ireland and Cambridge before coming to Leeds as a Consultant Paediatrician in 1992. She developed the liver service which was then designated as one of the 3 national children's liver services in 2000. Within the team in Leeds she has taken the lead in metabolic liver disease running a joint clinic with the metabolic paediatricians from Manchester. She is involved in clinical research in cholestatic liver diseases, transplantation, transition to adult services and metabolic liver disease.

Professor DA Kelly

Improving outcomes in paediatric liver transplantation

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The successful development of pediatric liver transplantation has dramatically changed the prognosis for many infants and children dying of acute or chronic liver failure. As the emphasis moves from immediate survival, and the prevention and management of early post-operative complications, attention has focussed on long term outcome and quality of life, (QoL), reducing the deleterious effects of essential immunosuppression, prevention of chronic infection and rejection and managing a smooth transition from childhood to adolescence and adult life.

Overall survival has improved due to advances in medical and surgical management, which has led to a change in the indications and case mix of children requiring liver transplantation in the UK.

Survivors will require regular monitoring both at the specialist centre and with their local hospital in order to ensure early identification of rejection and technical complications, prevention of chronic infection, such as CMV and EBV and reducing the adverse effects of immunosuppression especially renal dysfunction, hypertension, hyperlipidemia and the development of malignancy, such as post-transplant lymphoproliferative disease

Issues which need resolution include the shortage of organ donors, the aetiology of indeterminate graft hepatitis and fibrosis, the detection and prevention of adverse cognitive or psychological functioning and the management of late technical complications, recurrent disease, adherence and transition to adult care.

It is important to pay close attention to nutrition, bone metabolism, endocrine function and psychosocial development. Annual or five yearly assessments of cognitive function, educational achievement and patient and family perceptions of quality of life may allow appropriate intervention.

Finally the management of adolescent transition to adult care, with all the significant issues of non-adherence requires a multidisciplinary team approach, involving both adult and paediatric providers, to be successful.

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McDonagh JE, Kelly DA. Trans-plan-sition! Transplantation and transition. Pediatr Transplant 2007 Sep: 11(6):578-581

Biography

Professor Deirdre Kelly is Professor of Paediatric Hepatology at the University of Birmingham, Consultant Paediatric Hepatologist and Founding Director of the Liver Unit for Birmingham Children's Hospital NHS Foundation Trust. She is a graduate of Trinity College, Dublin.

She has trained in both adult and paediatric gastroenterology and hepatology. She set up the Paediatric Liver Unit at Birmingham Children's Hospital in 1989 which provides a national and international service for children with liver failure and undergoing liver transplantation, transforming survival and outcome for these children. Until 2008, the Unit was the only national unit to be designated for small bowel and liver transplantation in the UK.

Professor Kelly runs an active research programme focusing on viral hepatitis in children, molecular genetics of inherited liver disease, quality and outcome of life following liver and/or intestinal transplantation. She is on the Council of the General Medical Council (2013-). She was a Commissioner on the Care Quality Commission (2008-2013) and Healthcare Commission (2007-2009).

She was Medical Director of the Children's Hospital (2000-2007). She has been President of the European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN) (2006-2010), British Society for Paediatric Gastroenterology, Hepatology and Nutrition (BSPGHAN) (2004-2007) and the International Paediatric Transplant Association (IPTA) (2002-2005).

Professor Kelly is author of several textbooks about paediatric liver disease and has published many original articles and chapters on liver disease, liver transplantation and viral hepatitis.

Key note Speaker

Professor Fergus Shahanan

Fergus Shanahan is Professor and Chairman of the Department of Medicine at University College Cork (UCC), National University of Ireland. Born and educated in Dublin, he attended medical school at University College Dublin where he graduated with honours in 1977. After internship and residency in internal medicine in Dublin, he did a two-year fellowship in clinical immunology at McMaster University in Ontario, Canada, followed by a two-year fellowship in gastroenterology at the University of California, Los Angeles (UCLA). After completion of his fellowship at UCLA, he was appointed to the faculty there, rising to the rank of Associate Professor before making the decision, in 1993, to return to his native Ireland.

Together with colleagues from several departments and different faculties within University College Cork and Teagasc (a research agency of the Irish Ministry of Food and Agriculture), Dr. Shanahan led a team of clinicians, clinician-scientists, and basic scientists to successfully compete for seed funding from Science Foundation Ireland to create a multi-disciplinary research center, the Alimentary Pharmabiotic Centre, which investigates host-microbe interactions in the gut in health and disease. Under Dr. Shanahan's directorship, the center now has a membership of 168 staff, scientists, and students and has expanded its funding and research base by securing research alliances with indigenous and multinational companies within the food and pharmaceutical sectors.

Dr. Shanahan has published more than 450 scientific papers and several on the medical humanities and has co-edited several books. He is a Fellow of the Royal College of Physicians in Ireland, Canada, and the United Kingdom as well as of the American College of Physicians. He served as President of the Irish Society of Gastroenterology from 2007-2009. He was recently named to the "Irish Life Science 50" a list of the top 50 Irish and Irish Americans in the life science industry. In 2013, Science Foundation Ireland named him as its Researcher of the Year.

His interests are in mucosal immunology, gut microbiota, inflammatory bowel disease, and most things that affect the human experience.

Dr Jeremy Kirk

Biography

Paediatric training in London including time as Research Fellow at St Bartholomews along with the 2 paediatric registrars Ian Sanderson and Simon Murch!

Currently Consultant Paediatric Endocrinologist (Honorary Reader) at Birmingham Children's Hospital. Previously Secretary of the BSPED 2002-2008, and Subspecialty Representative on RCPCH Council 2007-12. Since 2012 involved in the NIHR as Clinical Director (CD) of the Clinical Local Research Network for Birmingham and Black Country (BBC-CLRN), and since March 2014 as CD of the West Midlands Clinical Research Network (WM:CRN).

Have a special interest in puberty and also growth and growth hormone (GH). Wrote the initial national GH shared care guidelines and also Expert Advisor to the NICE 2010 paediatric GH guidelines.

Dr Richard Russell and Dr Richard Hansen

Top tips for managing IBD – Twitter interactive cases

Richard K. Russell on behalf of the BSPGHAN IBD working group accompanied by Richard Hansen who will control the live twitter feed

Note: This presentation will feature innovative interactive content delivered via Twitter. To ensure you are able to take part, please install the Twitter app on your mobile phone and sign up for a Twitter account. Follow @BSPGHAN and search for and follow tweets on the #BSPGHAN hashtag during the meeting.

This presentation will feature a number of interactive cases which will allow discussion and interaction via real clinical cases. These are designed to illustrate common rather than rare situations. While the specific contents of each case will be discussed on the day we would like to highlight recent and forthcoming publications together with dates for educational meetings which will continue we hope to enhance the education of BSPGHAN members on current and future developments in paediatric IBD.

Key publications:

1. 2013 updated IBD standards - www.ibdstandards.org.uk/uploaded_files/IBDstandards.pdf
2. Ruemmele FM, Veres G, Kolho KL et al. Consensus guidelines of ECCO/ESPGHAN on the medical management of pediatric Crohn's disease. Journal of Crohn's and Colitis 2014;8(10):1179-207.
3. IBD audits 2014 - www.rcplondon.ac.uk/projects/inflammatory-bowel-disease-audit
4. Ruemmele FM, Hyams JS, Otley A et al. Outcome measures for clinical trials in paediatric inflammatory bowel disease: an evidence-based, expert-driven practical statement paper of the paediatric ECCO committee. Gut 2014;e-pub.
5. Fell JM et al. Management of Ulcerative Colitis. Archives of Disease in Childhood 2015
6. Kammermeier J et al. Management of Crohn's Disease. Archives of Disease in Childhood 2015

IBD UK educational events 2015

1. Progressing paediatrics: Difficulties in managing Inflammatory Bowel Disease in Young People RCPCH London 17.3.15
2. IBD audit "regional" network meeting 18.3.15, RCP London
3. 2nd Digestive Disorders Federation 22 -25 June 2015 at ExCeL, London (7 IBD sessions within the programme plus much more)
4. ECCO paediatric IBD workshop, Glasgow 25.11.15

CV/Biography

RICHARD K. RUSSELL MB ChB, MRCPCH, MRCP, PhD.

Current Occupation:
Consultant Paediatric Gastroenterologist and Honorary Clinical Senior Lecturer
Dalnair Street, Yorkhill Hospital, Glasgow 2007 - present.

Qualifications:
MB ChB The University of Edinburgh 1988 – 1993.
MRCPCH 1998.
PhD The University of Edinburgh, 2007.

Papers (Peer Reviewed Journals):
70 papers, 15 letters and 3 book chapters published

Full Academic Profile:
www.gla.ac.uk/departments/childhealth/ourstaff/drrichardrussell

Current Grant Support includes:
Co-applicant on Medical Research Council Strategic Grant: (G0800675) The Paediatric-Onset Inflammatory Bowel Disease Cohort and Treatment Study (PICTS)

Personally awarded NHS research Scotland career fellowship 2012.

Relevant previous Experience and Current Post:

I am currently one of 5 consultant paediatric gastroenterologists working in Yorkhill childrens hospital in Glasgow caring for children with gastroenterology, hepatology and complex nutritional problems from the West of Scotland. I am the UK chair for IBD within paediatrics (BSPGHAN) and have been involved with setting up a number of specific IBD developments locally and nationally including: transition clinics, patient support days, paediatric involvement in the UK IBD audit and the development of specific patient treatment pathways.

My main research interest is inflammatory bowel disease and I have presented and published widely on this subject. I have previously lead a national multicentre study investigating the genetics of early onset inflammatory bowel disease in Scotland that resulted in the award of a PhD. The study has recruited more than 800 children with IBD from across Scotland. The work has now moved forward to form part of an international adult/paediatric genetics consortium.

I am actively involved currently in several clinical research studies. I am chair of the BSPGHAN IBD working group and a member of the ESPGHAN IBD "Porto" group and a member of the Paediatric European Crohn's and Colitis committee. I am one of 2 paediatric members of the UK IBD audit group.

Dr Tracy Coelho

In September 2013, CICRA awarded the Dave Casson Clinical Research Fellowship (PhD) to Dr Tracy Coelho, a paediatric gastroenterology grid trainee based at University Hospital Southampton (UHS) NHS Trust. Tracy was delighted to take up this post and join the very active local Paediatric IBD research team that represents a collaboration between the University of Southampton and UHS. Over recent years, CICRA has been supporting research at Southampton exploring the genetic, nutritional and immunological basis of IBD. Tracy started his CICRA-funded research work in the immunology and genetics of IBD from September 2013 during his final year of paediatric gastroenterology training. Recently in September 2014, he was appointed as a clinical consultant, joining a dynamic and a research-driven team of paediatric gastroenterologists at Southampton. The appointment as paediatric gastroenterology consultant is currently part-time (50%), which allows him to continue with his research in IBD. The key focus of Tracy's research is on the genetics and immunology of IBD. Doing a PhD alongside a consultant job is a huge challenge. In his presentation, Tracy talks about the challenges involved in maintaining a fine balance between being a dedicated clinician and an enthusiastic researcher.

Selected plenary abstracts

Thursday 29th January 2014

Challenges in the management of paediatric non-alcoholic fatty liver disease: a longitudinal follow-up study.

Mann, Jake P1; Armstrong, Matthew J2; Sewel, Peter3: Rajwal, Sanjay3:McClean, Patricia
1University of Cambridge, Cambridge; 2 Centre for Liver Research, Birmingham; 3Leeds General Infirmary, Leeds

Background:
Non-alcoholic fatty liver disease (NAFLD) affects up to 10% of children in the western world, and in conjunction with obesity is becoming a public health concern. Despite this, the natural history of paediatric NAFLD is unclear due to a lack of longitudinal studies. In the UK, there are no established guidelines to identify those at risk of progressive liver disease (non-alcoholic steatohepatitis (NASH)) and that require transition to adult hepatology services.

Methods:
We performed a retrospective review of children consecutively diagnosed with NAFLD at a tertiary referral hepatology service in the UK, between 2003 and 2014. Patients had been referred from either primary care physicians or general paediatricians with incidental, asymptomatic, abnormal liver function tests (LFTs) and/or fatty liver on ultrasound scan (USS). Patient characteristics, metabolic features and complications of liver disease were recorded at baseline and follow-up.

Results:
100 patients with primary NAFLD were followed-up for a mean 2.5±2.1 years. 77% (77/100) were male and mean age at presentation was 12±2.8 years. 59% were Caucasian and 41% were Asian. 67% were referred due to abnormal LFTs and 28% due to an abnormal ultrasound.

On presentation, median BMI standard deviations (SDs) was 1.9 (range 0.45-3.0, 98th centile), ALT 71.5 (range 5-421) IU/L, cholesterol 4.3 (range 1.3-6.2) mmol/L, and bilirubin 8 (range 2-44) µmol/L. 12% (13/100) had type 2 diabetes confirmed by oral glucose tolerance test or HbA1c.

20/100 patients had undergone biopsy. 65% (13/20) of patients had biopsy-proven NASH and 25% (5/20) had moderate-severe fibrosis.

At the end of follow-up, there was a trend towards increasing severity of the metabolic syndrome. 65% of patients had an increase in HbA1c (mean increase 9±23 mmol/mol) and 56% had an increase in cholesterol (mean increase 0.4±0.4 mmol/L), but these changes were not statistically significant.

There were no liver related complications during follow-up. At the end of follow-up, 66/100 patients had been discharged from the paediatric clinic. 79% (52/66) were under the care of their primary care practitioner, including 9 patients with NASH and fibrosis on biopsy. Only 3% (2/66) had transitioned to the care of an adult hepatologist, with the remaining 18% (12/ 66) under another secondary care specialist.

Conclusions:
There is a need for validated non-invasive scoring systems to help identify children who require liver biopsy and long-term follow-up. With the collaboration of adult colleagues, those with NASH ± fibrosis should undergo long- term follow-up to aid our understanding of disease progression. However, this will require additional resources in adult hepatology.

Outcome of live versus deceased donor liver transplantation in infants with biliary atresia

Tibrewal[1], Shiv: Davison[1], Suzanne: Prasad , Raj[2]: Attia, Magdy[2]: Hidalgo, Ernest[2]: Alizai, Naved[2]:Rajwal, Sanjay[1]: McClean, Patricia[1]:
[1]Children's Liver Unit, Leeds Teaching Hospitals NHS Trust, Leeds.
[2]Department of Hepatobiliary Surgery and Transplantation, Leeds Teaching Hospitals NHS Trust, Leeds.

Background:
Biliary atresia (BA) is the most common indication for liver transplantation (LT) in infants. Surgical advances have led to live donor (LD) as well as deceased donor (DD) LT being possible options. Our programme commenced DDLT in 2000 and introduced LDLT in 2007.

Aim:
To compare outcome of DD and LDLT in infants with BA listed for LT at age < 1year.

Method:
All infants with BA listed for LT between 2000 and 2013 were studied. From 2007, families were counselled regarding the possible option of LDLT. All infants were on DD waiting list until time of LT, irrespective of whether LD was being considered. Outcome data was entered prospectively into unit database for national audit purposes

Results:
50 infants (M1:F1) were listed for LT at median age 190 (62-351) days, 18 in Pre-LD era. 7 had BASM, and 7 had no previous Kasai. 3 died awaiting DDLT, (1 in Pre-LRD era) and 47 underwent LT (33 DD 14LD). Patient and graft survival 1 year post LT was 45/47 (96%) and 41/47 (87%), and long term (median FU 6y 4m) was 45/47 (96%) and 38/47 (81%).

Comparing DDLT and LDLT, median time from listing to LT was for DDLT 36 (4-176) and LD 76 (20-216) days. Post-LT admission duration was DD 23.5 (16-128) and LD LT 22 (15-59) days. Primary non-function (PNF) occurred in 4/33 (12%) following DDLT, (all underwent re-LT) and 0/14 after LDLT. After 1yr FU, patient / graft survival are 97% (32/33) / 85% (28/33) for DD and 93% (13/14) / 93%(13/14) for LD recipients. Two children died, one (DD) due to PNF and sepsis after re-LT, the second (LD) due to hepatic artery (HAT) and portal vein thrombosis (PVT) recurring after re-LT.

Excluding 4 children who lost grafts to PNF, complications at 1yr FU in 29 DD and 14 LD were compared. HAT (5) or stenosis (1) occurred in 3/29 (10%) DD and 3/14 (21%) LD recipients (p=0.18). Of these, 1 (LD) underwent re-LT, 4 (2DD:2LD) thrombectomy and revision and 1 (DD) angioplasty. PVT or stenosis occurred in 3/29 (10.3%)

DD and 6/14 (43%) LD recipients (p=0.01). Of these, 5 (4LD) had PV stenosis treated by angiographic venoplasty. Biliary complications occurred in 4/29 (14%) DD and 5/14 (56%) LD recipients (p=0.1). Acute rejection requiring treatment occurred in 9/27 (33%) DD and 5/14 (36%) LD recipients (p=0.65).

Conclusion:
LT for infants with BA has a good outcome with 96% 1 yr survival being maintained during FU. Although LDLT led to a higher incidence of vascular complications, requiring intervention, particularly portal vein stenosis requiring angiographic venoplasty, likelihood of PNF or re-transplantation was reduced. Overall mortality of 3/50 on waiting list was not seen in those in whom LD was possible.

Transjugular intrahepatic portosystemic shunt (TIPSS) insertion for the management of portal hypertension in children: a single-centre

Johansen, Lauren[1]: McKiernan, Patrick[1]: Kelly, Deirdre[1]: Sharif, Khalid[1]: Lloyd, Carla[1]: Olliff, Simon[2]: Mangat, Kam[2]: John, Philip[1]: McGuirk, Simon[1]:
[1]Birmingham Children's Hospital, Birmingham; [2]Queen Elizabeth Hospital, Birmingham

Background:
TIPSS has an established role in the management of portal hypertension in adults. There is however a paucity of evidence outlining the indications for and long term outcomes of TIPSS in childhood. Our aim was to assess the use of the TIPSS procedure in the management of portal hypertension at a supra-regional paediatric liver unit.

Methods:
Retrospective review off all children (age 0-18 years) that had undergone a TIPSS procedure at Birmingham Children's Hospital since 1995. Subjects were identified from the liver unit database and radiology information system. Data was collated and analysed following review of patients' medical records and imaging.

Results:
35 TIPSS procedures were performed on 34 patients, over a 19 year period. The median age at time of TIPSS was 12 years (range 7 months-17 years). 20 of the procedures were performed to palliate symptoms of recurrent or active variceal bleeding; 6 were done as a bridge to transplant and 9 were performed to both palliate symptoms and act as a bridge to transplant. In 2 cases the procedure was performed as an emergency measure due to life threatening bleeding. There was successful placement of a covered stent with creation of a porto-systemic shunt in 29 cases. In 6 patients it was a failed procedure. In 16 cases there was a measurable reduction in hypersplenism with a rise in platelet count and a reduction in spleen size. Complications occurred in 11 cases and included encephalopathy (3), hepatic pseudo aneurysm requiring placement of a covered stent (1), secondary infarction and liver failure (1), bile leak (1), infection (1) and minor symptoms (4). In all but 5 patients variceal bleeding came under control; of the remaining patients 3 had persistent bleeding but at a reduced volume, 1 had persistent hypersplenism requiring splenic artery embolization and 1 had no improvement and so required early liver transplantation. 5 of the initially successful TIPSS later required further intervention due to thrombus formation and occlusion (2) or narrowing of shunt (3). 1 patient has been lost to follow up. Of the remaining patients 14 have subsequently undergone liver transplant and 6 have died (3 from sepsis and 3 from progressive deterioration of liver disease whilst awaiting transplantation).

Conclusion:
This study represents the largest series of paediatric TIPSS procedures to date. It demonstrates that TIPPS can be successful in palliating patients with variceal bleeding and also acting as a bridge to later transplantation. However, it is not without risk and therefore patients must be appropriately selected and counselled for the procedure.

Why can't we implement Hepatitis B vaccination policy: A retrospective regional review

Abdel-Hady M[1], Tahir M[2], Gowen H[1], Velander N[3], Ismail KM[4], Kelly DA[1]
[1]Liver Unit, Birmingham Children's Hospital, Birmingham; [2]Public Health England, West Midlands, Birmingham; [3]Statistics, Modelling and Economics Department, Public Health England, Colindale, London; [4]School of Clinical & Experimental Medicine,University of Birmingham

Introduction
Viral Hepatitis B (HBV) affects 180,000 individuals in the UK. Most children are infected by vertical transmission from an infected mother during the antenatal, intrapartum or postpartum period. Timely HBV immunisation schedule may prevent persistent HBV infection in over 90% of these infants.

UK Department of Health policy mandates antenatal screening of pregnant women for HBV infection, followed by immunisation of at risk babies according to an agreed schedule which includes a 12 month sample to detect efficacy combined with a booster immunisation.
Completion of the vaccination programme is dependent on effective and timely communication between health care professionals in primary and secondary care and compliance from a transient, mainly ethnic minority population.

Previous data from the UK has indicated that there are inconsistencies in the delivery of HBV vaccination with up to 50% of identified at risk infants who did not receive the complete vaccination schedule.

The aim of this study was to audit the uptake of HBV immunisation in high risk infants born between 2007-2012 in the West Midlands region and to identify barriers for effective vaccine delivery.

Methods
We identified babies born to women who were found to be HBV positive from 2007-2012. Anonymised data were collected from (West Midlands Public Health England, Child Health Information system (CHIS) and hospitals database). Information collected included demographics, vaccination schedule, and post vaccination HBV serology testing.

Descriptive frequency distributions and cross-tabulations were produced.

Results
11/17 areas in the West Midlands submitted data. Data were incomplete across all the areas. 961 children were identified. Data on post code/ PCT were missing in 98 (10%). Data on ethnicity were missing in 268 (28%) and not stated in 150 (16%). Ethnicity included; African (16%), Chinese (11%) and Pakistani (10%).

Vaccination dates were not recorded in 312 (32%), and serology test results were not available in 733 (76%). The majority (63%) had their vaccination at hospital based clinics. 696/961 (72%) had a complete course of vaccination (3 or more) of whom only 460 (66%) received the vaccination on schedule. In the remaining 265 (28%), less than 3 vaccinations were recorded. Only 20% of the babies who had a complete course of immunisation (151/696) had a post vaccination serology test. 131/151 were immune and 94 (71%) of the immune children had the vaccination on schedule. 5/151 were found to be infected (HBsAg positive) of whom 2 did not have the vaccination on schedule. The remaining 15 were either partially immune or not immune requiring a booster dose.

The areas with highest rates of correct vaccine delivery had either GP or health visitor led service. In contrast, the lowest rates for vaccine delivery were paediatric clinic

Conclusions
Less than 50% of high risk infants born to HBV carrier women had their hepatitis B vaccination on schedule. This is likely due to the variable pathways and service delivery models involved. Data were unavailable for a large number of the infants, which makes it difficult to ascertain the number of successful vaccination and babies who may require booster dose of the immunisation. There is a need for a unified system of vaccine delivery across the West Midlands with a single central database to ensure effective vaccine delivery and recording to those high-risk infants.

Anti-Tumour Necrosis Factor Drug Monitoring in Paediatric Inflammatory Bowel Disease

Whyte Lisa1: Bignell Michelle[2]: Wong Theodoric[1]: Protheroe Susan[1]: Bremner Ronald[1]: Haller Wolfram[1]: Muhammed Rafeeq[1]:
[1]Department of Gastroenterology, Birmingham Children's Hospital, Birmingham, UK;
[2]Department of Biochemistry, Birmingham Children's Hospital, Birmingham UK

Background:
Commercial assays to measure anti-Tumour Necrosis Factor (TNF) drug and antibody levels have been available only in the last few years. The lack of familiarity with these tests and cost implication has limited their widespread clinical use. There is not much information available about the value of anti-TNF drug monitoring in paediatric inflammatory bowel disease (IBD) practice.

Methods:
We have done a retrospective analysis of anti-TNF drug monitoring in patients with IBD over 12 months. We have correlated anti-TNF drug levels with the presence of anti-TNF antibody, concomitant treatment with Azathioprine and the clinical remission status. We have also looked at the cost effectiveness of anti-TNF drug monitoring.

Results:
89 anti TNF drug and antibody levels were done in 47 patients in the last twelve months (43 patients had Crohn's disease, 4 had ulcerative colitis and 1 had IBD unclassified). All these patients were receiving Infliximab. 14 patients (30%) had below therapeutic levels of Infliximab and all were receiving Infliximab 5 mg/Kg dose. 12 of these patients had Crohn's disease and all these 12 patients were on concomitant treatment with Azathioprine. 7 of these patients (58%) had anti-Infliximab antibody positivity. Out of the 12 Crohn's disease patients with low Infliximab levels, 3 were in deep remission with mucosal healing and these three patients were positive for anti-Infliximab antibodies. In these 3 patients treatment with Infliximab was stopped. Of the 12 patients with Crohn's disease and low levels of Infliximab, 9 (75%) were not in clinical remission. 4 of these patients had antibody positivity and dose escalation to 10 mg/kg achieved clinical remission in only one of these patients. 5 Crohn's disease patients who were not in clinical remission with low Infliximab levels and no anti Infliximab antibodies achieved clinical remission with Infliximab dose increase to 10 mg/kg. 4 patients had Infliximab concentrations higher than the therapeutic range and all these patients were on 10mg/kg dose.

15 patients (31%) had anti-Infliximab antibody positivity and all these patients had Crohn's disease. 7 of these patients had low Infliximab levels, 2 had high Infliximab levels (on 10 mg/kg dose) and 6 had Infliximab levels in therapeutic range. 14 of these patients were receiving concomitant Azathioprine. Out of these 15 patients, 9 were in clinical remission.

Overall cost savings of about 9000 pounds per year were achieved by Infliximab dose adjustment, even after considering the cost of these investigations and the cost of increased dose of Infliximab in some patients.

Conclusion:
A significant number of IBD patients receiving Infliximab treatment have sub-therapeutic levels of Infliximab. Majority of the patients with low levels of Infliximab are not in clinical remission and have anti- Infliximab antibodies. Dose escalation of Infliximab is successful in achieving clinical remission in patients with low infliximab levels without the presence of anti-Infliximab antibodies. We have not seen any beneficial effect of concomitant treatment with Azathioprine in achieving therapeutic levels of Infliximab or the development of Infliximab antibodies. Anti-TNF drug level and antibody monitoring are very useful and cost effective in the management of children with inflammatory bowel disease

Conflict of Interest
LW, MB TW, WH and RB have no interests to declare. SP had received consultation fee and meeting sponsorship from MSD. RM had received speaker fee, educational and research grants from MSD.

A 17-year prospective cohort study of paediatric inflammatory bowel disease patients diagnosed less than 10 years of age (Paris A1a)

Henderson, Pau[1]l: Rogers, Pamela[2]: Wilson, David C[1]:
[1]University of Edinburgh, Edinburgh; [2]Royal Hospital for Sick Children, Edinburgh

Background:
The recently published paediatric inflammatory bowel disease (PIBD) Paris classification highlights that patients diagnosed before their 10th birthday (Paris A1a) have a different clinical phenotype at presentation than those diagnosed aged 10-16 years (A1b). However, data regarding accurate incidence rates, disease natural history, medication use and surgery is required.

Methods:
All A1a PIBD patients within our prospective, regional PIBD database from South-East Scotland (SES) diagnosed from 08/97-07/14 had data recorded regarding demographics, phenotype data, details of medical therapy and surgery; data at last paediatric follow-up (FU) was also collected. Accurate incidence data was generated using publicly available population data for SES. Statistics were performed in GraphPad Prism and R with Poisson regression analysis for incidence data.

Results:
121 A1a PIBD patients (77 Crohn's disease [CD], 28 ulcerative colitis [UC] and 16 IBD-unclassified [IBDU]) were identified in the 17yr period (32% of entire PIBD cohort); 52% were male. Total FU was 816 patient-years; median FU was 7.1yrs (IQR 3.6 – 9.6). The incidence of A1a PIBD in SES during 2000-2013 was 4.4/100,000/yr (CD 2.8/100,000/yr; UC 1.0/100,000/yr; IBDU 0.6/100,000/yr); there was no significant increase in incidence between 2000-2006 and 2007-2013 (p=0.577). One patient was diagnosed <2yrs (male with UC requiring only 5-ASA treatment to date), 34 at 2-5 years and 86 at 6-9 years. At diagnosis 20% of CD patients diagnosed at 2-5yrs had panenteric diseasee, 36% isolated colonic disease, and 16% had isolated oral and/or perianal disease; all had inflammatory behaviour. At a median of 8yrs FU phenotype was similar; although 2 (8%) progressed to pan-enteric disease and 1 (4%) to penetrating disease. In the older CD cohort (diagnosed 6-9yrs) more (41%) had panenteric disease at diagnosis (45% at FU); 25% had isolated colonic disease; and 17% of this group progressed to penetrating/fistulising disease at follow-up. The younger age-group were less likely to have panenteric disease at diagnosis (p=0.002) or progress to penetrating disease (p<0.001). 73% of A1a UC patients had E3 disease at diagnosis; this figure was 77% at a median of 5.5yrs follow-up. 61% had exposure to thiopurines, 26% to methotrexate, and 25% to anti-TNF therapy; successful remission at anti-TNF therapy induction was achieved in 57% of patients. 20 patients (17%) had IBD-related surgery; 16 CD patients (median of 6.6yrs from diagnosis) and 4 UC patients (median 3.2yrs from diagnosis).

Conclusions:
A third of patients diagnosed with PIBD present before 10 years of age, with the incidence of very early-onset disease remaining stable over time. The requirement for immunosuppression, especially biological therapy, is significant and represents a high disease burden in these patients. Intriguingly, differences exist between those diagnosed <6 years of age and at 6-9 years with the older group more likely to have panenteric CD at diagnosis and to progress to penetrating disease.

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Links between the microbiome and nutrition in the infant

Dr Nicholas Embleton

BSPGHAN winter meeting 2015

There has been an explosion of interest in the last decade of scientific study relevant to neonatal nutrition. The areas of work amalgamated under the Developmental Origins of Health and Disease (DOHaD) umbrella seek to explore the relationships between exposures (often, but not exclusively, nutritional) in pre- and early post-natal life, and determine how these impact on later life health and chronic disease. Additionally, there has been a growing interest in how early life colonisation of the gastro-intestinal (GI) tract may impact on later health and disease, with evidence for example, that altered colonisation patterns due to delivery by caesarean section may themselves impact on later health. These issues are of major importance to the >15 million babies born prematurely around the world every year, in whom necrotising enterocolitis (NEC) and sepsis are now responsible for more deaths after the first week of life than any other pathology. There is increasing evidence to show that perturbations to the GI microbiota are strongly associated with both NEC and sepsis, and emerging data to show that certain interventions (e.g. probiotics, breast milk, lactoferrin etc.) may modulate disease via microbiotic mechanisms. The Neonatal Nutrition Network (N3) is a multi-disciplinary UK wide group that is helping coordinate a series of studies examining these issues. Mechanistic work, is seeking to explore how changes in gut microbial community structures may relate to functional changes using metabolomic analysis of stool and urine. Through these collaborations researchers aim to develop a better understanding of how to improve nutritional status in preterm infants.

Does mucosal inflammation contribute to malnutrition and stunting in resource-poor countries?

Kelsey Jones
Wellcome Trust Centre for Global Health Research
Imperial College, London

Undernutrition (including acute malnutrition and stunting) remains the most important risk-factor for childhood mortality, responsible for 45% of all deaths among children under-five worldwide. Stunting is associated with long-term loss of growth and developmental potential, reduced educational achievement, poor economic productivity in adulthood, and low-birthweight and mortality in subsequent generations. It a key factor entrenching poverty.

While stunting has often been thought to result from the dual burden of chronic dietary insufficiency and frequent enteric infections, interventional studies have shown that addressing these factors results in minimal improvement. Work from the Gambia revealed a close relationship between growth failure and the severity of a syndrome of chronic sub-clinical small-intestinal inflammation called environmental enteric dysfunction (EED, previously environmental or tropical enteropathy). EED is practically ubiquitous where children live in poverty. It is thought result from high-level ingestion of pathogenic and non-pathogenic microorganisms due to contaminated water supplies and inadequate sanitation facilities. The inflammatory component has been considered to be a healthy adaptive response to microbial challenge at the gut mucosal surface but an alternative hypothesis is that functionally redundant inflammatory activation actually triggers growth failure itself – exactly as occurs in paediatric IBD.

A randomised controlled trial of the use of mesalazine in Kenyan children with severe acute malnutrition and stunting provided data in support of this 'maladaptive' model. Mesalazine was safe, and resulted in modest reductions in gut and systemic inflammatory activation without exacerbating gastrointestinal symptoms or promoting microbial translocation. Cytokine-driven reduction of insulin-like growth factor-1 (IGF-1) release was evident.

EED affects hundreds of millions of children worldwide. If its association with growth failure is in fact causal, then it is indirectly responsible for an enormous burden of ill-health. The application of expertise gained from the study of aetiology, pathogenesis and natural history of other inflammatory bowel diseases of childhood presents a timely and important opportunity to understand the syndrome better and to start to understand what, if any, specific treatments might be beneficial.

Funded by The Wellcome Trust

Dr. Rosan Meyer

Honorary Senior Lecturer, Imperial College, Principal Paediatric Principal Research Dietitian, Great Ormond Street Hospital for Children

Rosan completed her degree in Dietetics in South Africa and specialised in paediatric nutrition in the United Kingdom, focusing on nutritional support, feeding behaviour and allergy. In 2004 she went on to finish her Masters in Paediatric Nutrition, focusing on paediatric gastroenterology, allergies, nutritional assessment, feeding support and performed research on the use of feeding protocols on the intensive care unit. In 2008, she completed her PhD in energy expenditure in critically ill children at Imperial College London. She currently is the principal research dietitian at Great Ormond Street Hospital for Children, leading a project on the impact of gastrointestinal food allergies on children and their families.

She is also module leader for the Food Hypersensitivity Module that forms part of the MSc in Allergy at Imperial College London, and is honorary senior lecturer in paediatrics at the same university. In addition she Chair of both the specialist Food Allergy and Intolerance Group of the British Dietetic Association and the European Section of the committee of the International Network for Diet and Nutrition in Allergy.

Per T. Sangild
Curriculum Vitae

Current employment
Professor, Faculty of Health and Medical Sciences,
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Academic profile, in brief:
Responsible for research consortia focused on developmental nutrition, gastroenterology and endocrinology in infants and animals during health and disease (Clinical Nutrition). Target analytical areas are digestion, histology, immunochemistry, enzymes, metabolism. Academic teaching in research philosophy, nutrition and developmental physiology.

Academic degrees:
- 1986: MSc (Agricultural Science), Faculty of Life Sciences, University of Copenhagen
- 1990: PhD (Animal Physiology), La Trobe University, Australia & Univ. Copenhagen
- 1996: DVSc (Veterinary Medicine), Faculty of Science, Univ. Copenhagen, 7 papers
- 2009: DMSc (Human Medicine), Faculty of Health, Univ. Copenhagen, 20 papers

Academic employment:
- Lecturer asssistant, Dept. of Anatomy and Physiology, Univ. Copenhagen, 1986
- PhD stipendiate, Dept. of Anim. Sci. Anim. Health, Univ. Copenhagen, 1987-1989
- Research Fellow, Dept. of Anim. Sci. Anim. Health, Univ. Copenhagen, 1989-1990
- Post Doctoral Fellow, Dept. Physiology, Univ. of Cambridge, UK, 1991-1992
- Research Fellow, Division of Reproduction, Univ. Copenhagen, 1992-1993
- Research Fellow, Division of Internal Medicine, Univ. Copenhagen, 1993-1994
- Senior Research Fellow, Division of Reproduction, Univ. Copenhagen, 1994-1996
- Associate Professor, Division of Animal Nutrition, Univ. Copenhagen, 1996-2004
- Visiting Professor, Dept. of Zoology, Nutrition, Univ. Hong Kong, 2004-2005
- Professor of Human Nutrition, Head, Preventive Nutrition, Univ. Copenhagen, 2005-2006
- Professor Clinical and Experimental Nutrition, Head, Univ. Copenhagen, 2007-2014
- Professor of Pediatrics (20%), Copenhagen Univ. Hospital (Rigshospitalet), 2013-
- Professor of Comparive Pediatrics and Nutrition, Univ. Copenhagen, 2015-
- Adjunct Professor, Sun Yat-sen University, Guangzhou, China. 2015-

Research leadership and funding:
Per Sangild has led 50 separate projects (total 2007-2014: 110 mio. DKK external funds), Extensive collaboration among local, national and international partners. International experience from longer research stays in UK, Australia and China. He has lead several university groups (20-25 staff) and organized many national/international scientific meetings.

Some key scientific achievements:
- Shown that glucocorticoid hormones regulate gut maturation in fetuses and newborns
- Characterized the response of the gut to nutrient intake in fetuses
- Development of preterm pig models of necrotizing enterocolitis and short bowel syndrome
- Documented the effects of a novel growth factor, GLP-2, on gut immaturity problems
- Clarified effects of enteral and parenteral nutrition for preterm neonates using piglet models
- Documented the effects of bovine colostrum on gut maturation in preterm pigs and infants

Publications:
140 peer-review, 300 abstracts, 45 book chapters. First/corresponding author on 90 peer-review publications, ~75 international publications last 5 yrs. Some reviews:

Sangild PT et al. (2014). Animal models of infant short bowel syndrome.
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Sangild PT et al. (2013). Preterm pigs as models in pediatric gastroenterology.J Anim Sci.91:4713-29.
Sangild PT. (2006). Gut responses to enteral nutrition in preterm infants and animals. Exp Biol Med. 231:1695-711.

Professor Jane Coad

Professor Jane Coad is Professor in Children and Family Nursing and Director of the Centre for Children and Families Applied Research (CCFAR), Coventry University, UK. Jane has a strong background in both art and nursing and undertakes a number of surveys and projects. These include innovative e-technology survey and evaluations and qualitative art-based participatory research, including handling large data sets, with children, young people and families. User-led approaches include consultation, collaborative and user-led projects. Predominantly, research is focused on children and families that have long term, complex and palliative care needs, and settings have included acute, community and public health settings in the UK and internationally such as Ireland, USA and Sri Lanka. In terms of professional recognition, Jane was awarded a Royal College of Nursing Fellowship in 2013 for lifetime research and leads on a number of local, national and international groups holding substantive posts.

Ms Sara Mancell

Selected plenary abstracts

Friday 30th January 2014

The epidemiology and outcome of biliary atresia in Scotland 2002-2013

Henderson, Paul[1]: Sutton, Emma[2]:Tayler, Rachel[3]: Hansen, Richard[1]: Barclay, Andrew[2]
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[3]Leeds Children's Hospital, Leeds
On behalf of the Scottish Society of Paediatric Gastroenterology, Hepatology and Nutrition

Background:
Biliary atresia (BA) is a rare and poorly understood liver disease of infancy that is fatal if not treated through early biliary drainage via the Kasai procedure. BA surgery was rationalised to three UK centres in 2002 following data supporting improved outcomes in institutions performing greater than five Kasai procedures per year(1). We have previously shown that outcomes in Scottish children were worse than expected in the years following initial rationalisation (2). We aimed to expand the post-rationalisation cohort of BA cases in Scotland to examine epidemiology and outcomes.

Methods:
Outcomes of the previously published 2002-2009 incident cohort(2) was first expanded. New incident cases of BA, with a Scottish postcode at birth and born between 2010-2013, were obtained using data from specialist nurse/team knowledge in the three Scottish regional paediatric gastroenterology networks. New data collection focussed on demographics, details of Kasai and outcomes (particularly 2 year transplant-free survival [2YTFS]). Accurate regional and national population data was obtained from the General Register Office for Scotland and statistics performed in R with Poisson regression analysis for incidence trends.

Results:
48 infants were initially identified, of whom 5 were excluded from outcome analysis (three with Kasai procedures performed in Edinburgh, one with less than 1yr follow up post-Kasai and one child born outside Scotland). Three infants required immediate liver transplantation; 43 infants underwent Kasai. Median age at Kasai in the full cohort was 55 days (range, 19–96) and showed significant improvement from 61 days in 2002-2009 to 49 days in 2010-2013 ($p<0.0001$). Of those with available data, 45% cleared their jaundice (bilirubin <20umol/l) six months post-Kasai; 2YTFS was 40%. BA incidence in Scotland was 0.68/10,000 live births and was relatively stable over time (0.66/10,000 in 2002-2009 and 0.71/10,000 in 2010-2013). However a cluster of cases was identified in Lanarkshire where the incidence was 1.26/10,000 live births, significantly higher than the rest of Scotland (0.59/10,000, $p=0.033$).

Conclusion:
BA atresia incidence appears relatively stable in Scotland but with an unexplained cluster of cases in the Lanarkshire region; examination of this cluster may provide epidemiological insight into disease pathogenesis Despite a significant reduction in time to Kasai portoenterostomy, the 2YTFS in Scotland remains disappointing, is lower than the pre-rationalisation figure of 65%(2) and is not currently an endorsement of centralisation of BA surgery in Scotland.

References
(1) McKiernan P, et al. Lancet 2000; 355(9197): 25-29.
(2) Tayler R, et al. Arch Dis Child 2013; 98(5): 381-383.

Acute upper gastrointestinal bleeding in childhood: development of the Sheffield scoring system to predict need for endoscopic therapy.

Thomson, Mike[1]: Campbell, David[1]: Narula, Priya[1]: Rao, Prithviraj[1]: Urs, Arun[1]:
[1]Sheffield Children Hospital, Sheffield

Background/Aims:
Acute upper gastrointestinal bleeding (AUGIB) is a rare and potentially life threatening condition in childhood. In adults with AUGIB validated scoring systems exist but these are not applicable to children. The aim of this study was to construct a clinical scoring system to accurately predict the need for endoscopic haemostatic intervention.

Methodology:
A retrospective data collection occurred over a three year period at a tertiary children's hospital. A total of 69 patients who had had endoscopic assessment were divided into Group 1 (no endoscopic haemostasis required) and Group 2 (endoscopic haemostasis required). A wide range of clinical parameters were collated including: pre-existing conditions; melaena; haematemesis and degree; transfusion requirement; parameters of hypovolaemia; presenting haemoglobin (Hb); Hb drop over 24 hours; platelet count; coagulation indices; liver function tests; and urea/electrolytes.

Results:
Parameters which reached statistically significance for endoscopic intervention (Group 1 v 2) were: presence of significant pre-existing condition; melaena; large haematemesis; heart rate (HR) >20 mean HR for age; prolonged capillary refill time; Hb drop of more than 20 g/l; need for fluid bolus; need for blood transfusion (Hb<80g/l); and need for other blood products. Using these parameters a number of scoring models were tested with multiple regression, and the most predictive resulted in a scoring system constructed with a total=24 and a cut off for intervention of 8.

The scoring system consists of the following:
significant pre-existing condition: 1; presence of melaena: 1; history of large amount of hematemesis: 1; heart rate more > 20 from the mean heart rate for age: 1;prolonged capillary refill: 4; haemoglobin drop of more than 20 g/l: 3; need for a fluid bolus: 3; need for blood transfusion (haemoglobin < 80g/l): 6; need for other blood product: 4.

Using this model would have resulted in 4 false negatives in the interventional group and 3 false positives in the non-interventional group. Hence: PPV of 91.18% (95% CI: 76.3% to 98.04%); NPV of 88.57% (95% CI of 73.24% to 96.73%); sensitivity of 88.7% (95% CI: 73.24% to 96.73%); and specificity of 91.18% (95% CI of 76.3% to 98.04 %.)

Conclusion:
In our study population, we were able to formulate a scoring system with good positive and negative predictive value for endoscopic haemostatic intervention in AUGIB in children. This may prospectively be studied and potentially lead to appropriate endoscopic intervention in this paediatric emergency.

Should the new ESPGHAN guidelines on diagnosing Coeliac Disease also apply to asymptomatic children?

Dr Siba Prosad Paul1, ST7 in Paediatric Gastroenterology; Prof Bhupinder Sandhu[1], Consultant in Paediatric Gastroenterology
[1]Bristol Royal Hospital for Children

Aims and Objectives:
In 2012 ESPGHAN guidelines for diagnosing Coeliac Disease (CD) were modified and recommend that in symptomatic patients a diagnosis of CD can be made without small-bowel biopsy if anti-tissue transglutaminase antibody (TTG) titre is greater than 10 times upper limit of normal (>10xULN) and HLA-DQ2 and/or DQ8 is positive. The aim of this study is to examine the relationship between TTG levels and histological grading in asymptomatic patients with newly diagnosed CD to establish whether the recent CD guidelines could be reliably applied to these patients

Methods:
Prospective data was collected at diagnosis on all asymptomatic children diagnosed with CD during March 2007 – September 2014. Our laboratory's ULN is 10U/ml. The relationship between the modified Marsh criteria histological grading and contemporaneous TTG levels was analysed. Data was also collected on the age of children and reason for initial serological screening. Cost benefit of extending the new diagnostic criteria to asymptomatic children with suspected CD was estimated based on biopsy costing £1340.00 and TTG and HLA-DQ2 costing £65.00 per patient.

Results:
94 asymptomatic children were diagnosed with CD. 63/94 children (67%) had TTG titres >10xULN and all these had small bowel enteropathy (sensitivity= 100%). Table 1 shows the histological grading with contemporaneous TTG titres . 45/63 (71%) had TTG >200U/l and this was associated with greater likelihood of Total villous atrophy (Marsh 3c)

The mean age at diagnosis was 9.1 years (1.75 years – 17.25 years). Reasons for serological screening were: Diabetes Mellitus (n=32), family history of CD (n=22) and Down's syndrome (n=5). Estimated cost saving to the Health Service for each child = £1,275.

	Pre NST	Post NST	p-value
Number	49	80	
Birth weight (gms)	866.5	822.5	NS
Gestation age (weeks)	26	26	NS
PN duration (day)s	39	40	NS
Weight at birth SDS	-0.307	-0.239	NS
Weight at discharge SDS	-2.16	-1.37	0.03
Change in SDS from birth to discharge	-1.37	-1.83	0.04
Length of stay	86.9	58.89	0.03

Conclusion:
All 63 asymptomatic children with TTG>10xULN had biopsy proven CD. 45/63 (71%) had TTG titres >200U/ml and were more likely to have total villous atrophy (Marsh 3c). Our study suggests that the ESPGHAN criteria for diagnosing CD via the serological pathway should be extended to asymptomatic children with resultant cost benefit to the health service and convenience for the family.

References:
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Biological therapy for Paediatric Inflammatory Bowel Disease in 524 patients: results from the 2014 UK paediatric biologics audit

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Background:
The biological therapy audit aims to measure the efficacy, safety and use of anti-TNF therapy in patients with inflammatory bowel disease (IBD) in the UK, including children.

Methods:
A prospective audit; all UK paediatric IBD (PIBD) teams providing biological therapy asked to identify patients newly starting biological therapy from 12/9/11 to 28/4/14. Disease severity was assessed using Physician Global Assessment (PGA) +/or Paediatric Crohn's Disease Activity Index (PCDAI).

Results:
22 of 25 (92%) UK specialist PIBD centres in the UK submitted data included in the analysis plus 8 additional UK paediatric centres. 524 patients were included; 429 Crohn's disease (CD), 76 ulcerative colitis (UC) and 19 IBD unclassified (IBDU). The commonest indication for starting therapy was active luminal CD 78% (355/458) or chronic refractory UC/IBDU 57% (58/102). The majority of patients were on concomitant co-immunosuppression with a thiopurine or methotrexate at time of starting biologic; 79% (386/488) infliximab (IFX) and 78% (58/74) adalimumab (ADA). 429 CD datasets were analysed in further detail (267 male); median age at diagnosis 12 years (IQR 9, 14). 396 initial treatments with IFX and 63 with ADA; 30 CD patients received both IFX and ADA. Median time from diagnosis to treatment was 1.42 years (IQR 0.63-2.97). At initial treatment, PGA was mild 8%, moderate 54%, severe 37% (N=179); median PCDAI score (N=255) was 28 (IQR 20, 38). The data suggest a disparity between disease severity scoring with PGA vs PCDAI; median PCDAI (28) suggests much milder disease than moderate-severe PGA in 91%. Where response to induction was documented, 77% CD patients demonstrated response and 65% full remission. Where reason for stopping biological therapy was documented, loss of response accounted for 25% and side effects or adverse events for 13% cases. When compared with data from the adult arm of this audit, children with CD receive treatment with a biologic significantly earlier in disease course than adults; 1.42 vs 5.23 years (p<0.001).

1389/1414 IFX treatments were seen for follow up at some point; median time from initial treatment to follow up was 167 days (IQR 46, 350). 10% (32/316) of CD patients experienced ? 1 adverse event; 3% of all follow-up treatments were associated with an adverse event (43/1480). No deaths or malignancies were recorded. 12% CD patients were prescribed IFX in accordance with NICE guidelines (TA187 criterion 1.5).

Surgery rates 6 months pre and post initiation of biologic (all IBD) were comparable; 7% pre, 5% post (N=524). Drainage of perianal abscess was significantly less common in CD after initiation with biologic 26% (27/102) vs 7% (3/42) p=0.01.

Conclusions:
Our audit suggests biologics are generally safe and effective treatments for IBD but the current length of follow up of this national cohort is relatively short. Disparity between disease severity scoring tools needs to be addressed, potentially by use of weighted PCDAI for CD patients. Documentation of indication for starting biological therapy is largely outwith NICE guidance. Adverse events are uncommon but secondary loss of response is an important clinical issue; longer term follow up data for this is required. Continued audit is essential to assess trends over time as clinical practice changes and use of biologics for UC and IBDU evolves.

Role of nutritional support team in improving outcomes in neonatal intestinal failure

Akshay Batra[1]; LV Marino[1]; R.M. Beattie[1] ;Freya Pearson [1]
[1]Southampton University Hospitals NHS Trust, Southampton

Role Of Nutritional Support Team In Improving Outcomes In Neonatal Intestinal Failure.

Objectives and Study:
The provision of parenteral nutrition (PN) forms part of standard practice in neonatal intensive care units to support nutrition and growth. Providing adequate nutrition during this vulnerable stage of development is of paramount importance and dictates the long term outcomes. The neonatal population is the largest single patient group receiving PN and to date, there are no standardised guidelines or funding arrangements in the UK to support its use. The aim of this retrospective cohort study was to look at the impact of provision of support via a multidisciplinary team on growth, mortality, length of hospital stay and time taken to achieve enteral autonomy.

Methods:
In our unit a multidisciplinary neonatal nutrition support team (NST) was established in 2011. This comprised of a neonatologist, pharmacist, dietician pediatric surgeon and gastroenterologist. Neonates receiving PN for greater than 28 days were identified from Badgernet. Outcome data was compared between those born between January 2009 to December 2011 to those between January 2011 to December 2013. This included anthropometry, outcome, duration of PN and length of stay in hospital.

Results:
A total of 128 cases were identified where neonates had received PN for greater than 28 days. The demographics are as in the following table.

Conclusion:

TTG titres (U/ml)	Modified Marsh Criteria identified on histology		
	3a	3b	3c
100 - 150 (n=13)	2	9	2
151 - 200 (n=5)	0	1	4
>200 (n=45)	7	13	25

Since the inception of a Neonatal NST in 2011, there has been a significant improvement in discharge weights and length of stay, demonstrating the efficacy of such an MDT. For further improvements to be achieved additional resources may be necessary to monitor growth within this risk group.

What should we feed children who can't feed themselves?

Sadlier, Claire[1]; Singleton, Kath[1]; Jenkins, Huw[1];
[1]University Hospital of Wales, Cardiff

Background:
Enteral tube feeding has been practiced for centuries, but in recent years its use has increased significantly. A wide range of formula feeds have been developed by feed companies, delivered in a sterile, liquid form, and it is now accepted practice to use a ready made sterile feed for all children requiring enteral tube feeding. However a growing number of parents are choosing to give their children pureed/blended diet (PBD) via gastrostomy rather than the formula feeds prescribed, despite professional groups not endorsing this approach.

Methods:
In order to ascertain how common is the use of PBD in our cohort of gastrostomy fed children we sent a questionnaire to parents to discover whether they had tried giving PBD diet, the benefits and what problems they had encountered.

Results:
83 questionnaires were sent and 50 returned, giving a response rate of 60%

18 said that they had heard of this method of feeding.

5 used PBD of which 4 use it exclusively.

1 had tried PBD but stopped because the dietitian had warned them about the risk of infection

All parents reported improved stool consistency, some also reported better weight gain, hair and nails growth, more energy, better feed tolerance.

No complications were reported by parents.

11 respondents would like to know more about pureed/blended diet.

Conclusion:
Parents are challenging the established view that a sterile formula feed is the best way to feed their child, and are learning about PBD through internet/social media. Benefits of giving PBD are being reported, and the anticipated risks appear to have been exaggerated. It is time for professional bodies to reconsider their advice about PBD.

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FRIDAY 30TH JANUARY
Invited speaker
biographies, abstracts
and notes pages

Dr Helen Brough

Dr Helen A Brough MSc (Allergy) MA (Hons) MB BS FRCPCH

Consultant in Paediatric Allergy, Guy's and St. Thomas' Hospital
Honorary Senior Lecturer in Paediatric Allergy, King's College London

Meeting Secretary, BSACI 2015 Annual Meeting

Helen studied Medicine at King's College, Cambridge University followed by clinical training at the Royal Free & University College London Medical School. Helen completed Paediatric Allergy and Immunology Higher Specialist Training at Guy's and St. Thomas' Hospital, King's College London and Great Ormond Street Hospital as the MADEL Clinical Lecturer in Paediatric Allergy. Helen completed an MSc in Allergy gaining a distinction at the University of Southampton and has submitted her PhD at King's College London.

Helen's clinical and research interests are in food allergy prevention, diagnostics and biomarkers, immunotherapy, asthma, eosinophilic gastrointestinal disorders and eczema. She is Principle Investigator for the Pronuts study at King's College London which is evaluating the rate of challenge proven co-existent peanut, tree-nut and sesame seed allergy. Helen set up Guy's and St. Thomas' Integrated Children's Allergy services in the community with an educational program for primary care clinicians; she also runs specialist Joint Children's Allergy & Respiratory clinics and Joint Children's Allergy and Gastroenterology clinics at St. Thomas' Hospital.

Helen is the Meeting Secretary for the British Society of Allergy and Clinical Immunology (BSACI) Annual Meeting and is on the Health Advisory Board for Allergy UK. Helen has written a book in Paediatrics (Rapid Paediatrics and Child Health) and several publications on the impact of environmental peanut exposure on the development of peanut sensitization and allergy and genetic biomarkers of peanut allergy. She was awarded 'Health Professional of the Year' runner up in 2010 by Coeliac UK and the BSACI Barry Kay Award in 2013.

Professor Simon Murch

Mast cells and eosinophils

Both mast cells and eosinophils may be recruited as part of a type-2 (Th2) reaction. These cells of innate immune lineage show significant evolutionary conservation and demonstrate overlapping functions. They are able to synthesise a variety of mediators, and share the capacity to release stored mediators rapidly. Eosinophils have been better recognised for many years, likely due to their ready recognition using standard histological techniques. They both play important roles in host defense, best recognised in protection against intestinal helminths.

While both cells function in many ways, two shared areas of important gastrointestinal concern are their ability to alter neural function, particularly motility, and to promote fibrosis. Mast cells share a critical transcription factor with the interstitial cells of Cajal that act as intestinal pacemakers, and are localised together with intestinal neural cells – thus effectively hard-wired into control of GI motility. Their secreted products specifically localise on enteric neurones, both altering motility and potentially altering sensory function to establish a state of visceral hyperalgesia. Similarly eosinophil products can directly alter neural function acutely and chronically. It is perhaps not surprising that the emerging role of mast cells and eosinophils lies in gut motility disorders.

Although mast cells may be triggered by antigens binding to specific IgE, they are also involved in the delayed reactions characteristic on non-IgE-mediated food allergies. Both mast cells and eosinophils may be locally adapted or may be newly recruited from bone marrow. Their recruitment pathway is a matter of significant current concern, not least because of the emergence of eosinophilic oesophagitis as a common disorder relatively resistant to treatment. This review will examine the methods by which these cells are recruited to the intestine, how they are activated and what are the consequences in both intestinal and neural function.

Biography

I went into paediatrics late, having spent the first years of my career in the Royal Naval Medical Services, aiming for a career in cardiology and trying to avoid too much time spent away at sea. An attachment with Prof June Lloyd at St George's rapidly converted me to paediatrics, and then a period as Lecturer with Prof John Walker-Smith at St Bartholomew's further differentiated me towards paediatric gastroenterology. I was privileged to work in the lab of Prof Tom MacDonald at Barts when we were uncovering the role of TNF-α in IBD and I have maintained an interest in mucosal immunology ever since. I have worked for the last 10 years as Professor of Paediatrics at Warwick Medical School and single-handed consultant Paediatric Gastroenterologist. I have previously been Gastroenterology Representative on BSPGHAN Council and remain a member of UK and international working groups on coeliac disease and eosinophilic disorders. My particular clinical and research interests are in allergic gastrointestinal responses, coeliac disease, protein losing enteropathy and the mucosal pathology of malnutrition disorders in resource poor countries.

Dr Adam Fox

Consultant Paediatric Allergist
MA(Hons), MD, MSc, MB, BS, DCH, FRCPCH, FHEA, Dip Allergy

Adam read Medicine and Neuroscience at Cambridge University before completing his clinical training at University College, London. Having completed specialist training in Paediatric Allergy in 2006, he is now a consultant and joint clinical lead of Allergy at Guy's & St Thomas' Hospitals NHS Foundation Trust, London, the UK's largest specialist allergy service.

Adam chaired the Department of Health commissioned RCPCH National Care Pathway for Food Allergy in Childhood and was part of the National Institute of Healthcare and Clinical Excellence (NICE) clinical guideline development group for Assessment and Diagnosis of Food Allergy in Children. He continues as an Expert Advisor to NICE. He is secretary of the BSACI and a member of the Paediatric Medicine Clinical Reference Group advising the NHS Commissioning Board for the strategic planning of specialist services. He is also a trustee and Chair of the Advisory Board of Allergy UK, member of the Anaphylaxis Campaign Clinical and Scientific Panel and the Advertising Standards Authority. Adam has previously been a commissioning editor for the journal Pediatric Allergy & Immunology and an external advisor and examiner for Southampton University MSc in Allergy.

Adam's research interests relate to numerous aspects of allergy especially food allergy and immunotherapy. He is a Reader in Paediatric Allergy at King's College London and his doctoral thesis on Peanut Allergy was awarded the Raymond Horton-Smith prize by Cambridge University.

Adam has an interest in medical education and he is director of the King's College London Allergy Academy. His clinical role involves the management of children with multiple allergic disease including food allergy, asthma and rhinoconjunctivitis as well as children with difficult eczema where food allergy plays a role.

Adam was recently included in 'The Times Magazine: Britain's Best Children's Doctors' (2012) and was awarded 'Paediatric Allergist of the Year' by Allergy UK in 2007.

Dr Neil Shah

Jackie Falconer BSc (Hons) RD
Lead Gastroenterology Paediatric Dietitian

Jackie graduated with a BSc (Hons) degree in management in 1986. She then went on to further study at Kings College in London and qualified as a state registered dietitian in 1991. She has worked in clinical dietetics since 1992, specialising in paediatrics in 1995. For the past 19 years she has worked in the area of paediatric gastroenterology at the Chelsea and Westminster NHS Foundation Trust and her particular areas of interest are short bowel syndrome, inflammatory bowel disease, non IgE mediated food allergy, coeliac disease and gastro-oesophageal reflux. She also holds a private clinic once a week for infants and children with gastro-intestinal dietary associated conditions.

Jackie held the position of chair of the Associate (nurses and dietitians') Members of the British Society of Paediatric Gastroenterology, Hepatology and Nutrition (BSPGHAN) from 2003–2007. She is also a member of the Allied Health Professionals (AHP) group of the European Society of Paediatric Gastroenterology, Hepatology and Nutrition (ESPGHAN). She has presented and chaired both nationally and internationally. More recently she has been elected to serve as the AHP representative on the ESPGHAN GI committee helping in policy and guideline development for Europe.

Jackie is registered with the Health Professions Council (HPC) in the UK and the British Dietetic Association (BDA).

BSPGHAN 2015 Annual Meeting

WEDNESDAY 28TH JANUARY
Poster abstracts

Team 1

Poster Walk Round

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G2

Interstitial Cells of Cajal in Human Tissues

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Background:
Interstitial cells of Cajal (ICC) are non-neuronal effectors of gastrointestinal motility. Since the marker, cKit, was identified in 1992 their importance has been increasingly recognised. ICC losses or pathology have been implicated in an increasing number of gastrointestinal disease states including Inflammatory Bowel Disease, Idiopathic Gastroparesis, and Hirschsprung's disease.

Hundreds of studies have now investigated ICC in a wide range of disease processes, yet normal function and distribution remain incompletely understood in human tissues. Methodological variation impairs the ability to compare studies. The aim of this study was to determine which methodologies have been used to identify ICC.

Method:
A structured review of the literature over a 21-year period (January 1992 – May 2013) was performed to identify studies examining the role of ICC in human motility. Papers were retrieved electronically, with those unavailable locally being obtained from the British Library. Data, recorded by two independent assessors, included the clinical condition, methodological variables, and the numbers of subjects.

Results:
20,802 abstracts were initially identified, from which 392 met our search criteria (126/392 review/editorial articles and 266/392 original articles). 391/392 papers were retrieved and analysed.

Of 57 different conditions examined, 28 affected paediatric patients and 33 investigated gastrointestinal pathology (paediatric and/or adult subjects). The most common gastrointestinal pathologies studied included Hirschsprung's disease and Slow Transit Constipation. A further 97 papers examined 'normal' tissue samples from adults and children.

253/266 original articles were immunohistochemistry-based (247/253 using cKit as an antibody). Antibodies were derived from over 30 manufacturers with additional variation in concentration used. Functional correlation (e.g. electrophysiology) was uncommon (17/266).

A mean number of 16.3 patients (range 1 – 80) and 7.6 controls (range 0 – 108) were examined. Control samples were often taken from "pathological" tissue, however this was not always clear.

Conclusion:
A wide range of conditions have been examined for ICC abnormality. The number of antibodies used, and inconsistency in their concentration may lead to variation in affinity and in results. Correlation of distribution with function was performed in only a small number of studies using electrophysiology. The number of control specimens was low and often not true control tissue.

Optimised techniques may allow multi-centre accrual of normative data and facilitate future studies. Correlation of distribution with function should be developed to enable a more accurate understanding of ICC (patho)physiology. Given the increasing number of gastrointestinal conditions now associated with ICC dysfunction, this is likely to lead to patient benefit.

G10

Superior Mesenteric Artery Syndrome: A Diagnostic Difficulty

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Introduction:
Superior Mesenteric Artery (SMA) Syndrome is a rare cause of small bowel obstruction, due to the compression of the duodenum between the abdominal aorta and the superior mesenteric artery. It usually follows rapid weight loss resulting in a lack of retroperitoneal and visceral fat.

Case Presentation:
We report two very different cases of interest, of teenagers who presented to general paediatrics with gastrointestinal symptoms following weight loss. Both were subsequently diagnosed with SMA syndrome and followed a successful refeeding programme through the use of nasojejunal tube to bypass the obstruction.

Conclusions:
These cases highlight the diagnostic difficulties associated with the rare SMA syndrome. Delayed diagnosis can result in complications relating to electrolyte disturbances, bowel obstruction and perforation.

G29

Diagnostic Yield of Paediatric Gastrointestinal Endoscopy at a Training Centre in UK: Results of Service evaluation.

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Background:
Endoscopy is integral to the diagnosis and management of many gastrointestinal problems in children. Recently the number of endoscopic procedures performed has increased considerably worldwide [1], raising questions about their appropriateness and cost-efficacy. The aim of this evaluation therefore was to determine diagnostic yield of endoscopy in a paediatric population in a large tertiary centre.

Methodology:
147 randomly selected cases were assessed from April 2012 to October 2014. 3252 endoscopic procedures were performed on 2471 children during this period. Indications for endoscopy, endoscopic and histopathological findings were collated and the endoscopic diagnostic yield and contribution to the management was evaluated.

Results:
The mean age was 9.58 (0.5-16.5) years, M:F ratio 1:1.42. Indications included: abdominal pain (66.6%); diarrhoea (42.4%); bleeding PR (27.4%); weight loss 19.6%; mucus PR 19.6% and urgency (9.6%) and nocturnal symptoms (9.8%) were also noted. Other indications included vomiting/ suspected reflux (20.9%) and feed aversion (12%).

The positive diagnostic yield was 18.9% for oesophagogastroduodenoscopy (OGD) alone, 32.6% for ileocolonoscopy (IC) alone and 39.21% when both occurred.

	HISTOPATHOLOGY		
Endoscopy	Positive	Negative	
Positive	45	24	PPV = 65.21%
Negative	18	60	NPV = 76.9%
	Sensitivity = 71.4%	Specificity = 71.4%	

The pre-test probability of making a positive the diagnosis prior to endoscopy was 42.8% with likelihood ratio of a positive test of 2.49. Using Fagan's likelihood ratio nomogram a post-test probability of 65 is calculated indicating a high degree of diagnostic contribution.

In 45% of the patients management was actively changed due to endoscopy and histopathology findings, and management contribution occurred in all patients.
No adverse events were noted in this cohort.

Discussion:
The number of endoscopy procedures performed world-wide has increased considerably recently raising questions regarding cost effectiveness. [2] In children a positive diagnosis is important but so may significant negative findings in terms of patient management and reassurance. Hence the relatively low positive diagnostic yield of OGD (18.9%) and IC (32.6%) in this cohort must be interpreted in this clinical context.

Overall endoscopic procedures had good sensitivity (71.4%) and specificity (71.4%) with NPV of 76.9% and PPV of 65.2% in our Centre. Of course appropriate selection of patients is contributory to this. Various studies have suggested that the diagnostic yield of endoscopic procedures improve if indications and appropriateness are critically assessed with use of Guidelines e.g. NASPGHAN, EPAGE (European Panel on Appropriateness of Gastrointestinal Endoscopies) and ASGE (American Society of Gastrointestinal Endoscopy) guidelines. [3, 5]

Therefore pre-procedural use of empirical therapy (e.g. PPI trial) and/or non-invasive tests (e.g. faecal calprotectin) may refine endoscopy use.

Conversely the negative endoscopies in this cohort were not necessarily inappropriate as they led to exclusion of suspected disorders with consequent reassurance and change in management.

Conclusion:
To improve the diagnostic yield of endoscopic procedures we recommend adherence to well established Guidelines for appropriateness and indication of endoscopy in children but it should be noted that a significant negative finding may be as important as a positive diagnosis in the care of these families.

References:
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G32

The pH study as a tool to guide management of suspected gastro-oesophageal reflux disease in paediatric patients

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Background:
The 24h oesophageal pH study is considered to be the gold standard for the assessment of gastro-oesophageal reflux (GOR). At our institution it has been used to quantify the disease extent in children with suspected GOR disease (GORD) failing to respond to medical therapy, when the diagnosis is in doubt or, in particular, when they are felt to be potential candidates for fundoplication surgery. The aim of this study is to assess the influence of this tool in the management of patients with suspected GORD.

Methods:
All children undergoing the pH study at our institution from January 2012 to December 2013 were included. Data was retrospectively collected from electronic hospital databases and clinician letters. This included: indication for pH study, endoscopic findings, pH study findings and subsequent management.

Results:
87 patients were included in the study (median age 4.6 years, range 7 weeks - 17.2 years). The most common indication for the study was persistence of symptoms despite optimal medical treatment (66%). Histology from endoscopic biopsies was normal in 44% of cases; it showed oesophagitis in 28% and other pathology in the rest. The median Demeester score from pH studies was 15.1 (range 0.6-203). Based on the pH study, 25% of patients had clinically significant GORD, 21% had mild disease and 54% had insignificant or no disease.

Overall, this led to no change in medical management in 42 patients (48%). Only 10 patients (11%) were immediately referred for fundoplication, with another 7 (8%) referred after a further trial of medical therapy. The pH study findings guided subsequent medical management in 9%. In the rest of cases, subsequent treatment was based on clinical assessment or endoscopic findings.

Out of the 17 referred for surgical consideration, only 12 proceeded to surgery, 11 of whom had 'severe' GORD on the pH study. 8 of the 12 had underlying neurodevelopmental or severe respiratory disorders.

6 patients were not started on a proton pump inhibitor until after the pH study was performed.

Conclusion:
There may be an overuse of the pH study in our institution, with it having no direct influence on management in most children and very few proceeding to surgery. Possible explanations include failure to optimise medical therapy in all patients prior to the pH study or the study being used as a tool to reassure parents and clinicians.

Among children whose GORD is confirmed to be severe enough on a pH study to warrant surgery, 75% had significant underlying neurodevelopmental or respiratory disorders. This group of patients appear to benefit from a low threshold for a pH study to plan surgery, being unlikely to respond to medical therapy. Consideration should be given to performing an endoscopy without a simultaneous pH study, initially, in other children not responding to medical treatment to reduce problems associated with the study (e.g. overnight hospital stay).

G39

Hypermagnesaemia induced by bowel-cleansing agent

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Background:
There are no established standarised guidelines for bowel cleansing prior to colonoscopy in children. The lack of evidence from systematic investigations leaves the possibility open for a shorter and simpler regimen. Saline laxatives that use sodium picosulphate and magnesium citrate as the active ingredient are equally effective as polyethylene glycols (PEG) but more tolerable. Hypermagnesaemia is a rare but potentially life threatening finding in paediatric patients. We describe a girl who developed severe hypermagnesaemia to administration of Picolaxâ with normal renal function.

Case report:
A 5-year-old girl with diarrhoea, following bone marrow transplantation for leukaemia, was under investigation for gut graft versus host disease. As per the guidelines, she received 2 doses of senna and Picolaxâ with 10 hours apart. She developed hypothermia, bradycardia and weakness. Her serum Magnesium was 5.45mmol/L with concomitant hypocalcaemia. She was treated with intravenous Calcium Gluconate and her symptoms rapidly improved. She had a normal renal function (estimated GFR), euvolaemic with normal electrolyte or medications that would impair renal function except cyclosporin. Eventually she underwent endoscopy 3 days later which showed significant colitis with histology confirming features consistent with graft versus host disease.

Conclusion:
Hypermagnesaemia usually occurs in patients with renal impairment or as a result of iatrogenic overdose but may develop even in patients with normal renal function. Also bowel preparation based on safety profile, patient co-morbid conditions and currently prescribed medications should be taken into account. Although clinical guidelines are intended to reduce the complications but do not address every clinical situation. This case highlights a need for careful clinical judgement and the risk of complications from bowel preparations with comorbidity. It might be worth considering minimal purgation or use PEG-based solutions with potentially less physiologic disturbances.

N2

Long term parenteral supplementation of Vitamin A in preterm neonates: Time for a change of practice?

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Background:
Premature infants have lower levels of Vitamin A at birth compared to term infants and are therefore at greater risk of deficiency, particularly if on long term parenteral nutrition. The recommended intake of Vitamin A in preterm infants remains controversial and is based on expert opinion.The only intravenous preparation currently available in the UK, Vitlipid N (Fresenius Kabi, Runcorn, UK) provides 920 IU/kg/day at the recommended dose of 4ml/kg/day. This study aims to assess the adequacy of this dose of Vitamin A in infants on parenteral nutrition (PN) for >28 days.

Methods:
We identified neonates who received PN for > 28 days between January 2009 and December 2013. Those who were on >50% of total calories via parenteral route and had their serum vitamin A, D and E concentrations measured at 4-6 weeks of age were included in the study. Additional data collected from case notes included; demographics, gestational age, diagnosis, duration of PN and anthropometry. Vitamin A deficiency was defined as serum concentration below 200µg/L (0.7µmol/L) and severe deficiency, with depleted liver stores, as below 100µg/L (0.35µmol/L).

Results:
43 cases fulfilled the inclusion criteria and were included. 39 of them were premature.The most common indication for PN was gut immaturity associated with preterm delivery in 24 cases followed by presence of congenital or acquired gut disorders in 19. At 4-6 weeks of age, 31/43 (72%) had vitamin A deficiency; with 15/43 (35%) having severe deficiency. There was a higher proportion of preterm infants who were deficient 30/39 (77%), of whom 4/5 (80%) remained deficient at 90 days, despite ongoing supplementation. None of the infants included were deficient in vitamin D or E.

Table 1: Demographic and anthropometric profile of babies on long-term PN

	Vitamin A deficiency	No Vitamin A Deficiency
	Median (25th- 75th percentile)	Median (25th- 75th percentile)
Number (total 43)	31	12
M:F	18:13	7:5
Gestation Age (weeks)	26 (25-28)	30.5 (25.25 – 35.5)
Birth weight (gms)	815 (695 – 1090	1760 (803-2742)
Change in SDS score for weight between birth and discharge	-1.40 (-2.05 to -0.62)	-1.6 (-3.62 to -0.57)

Conclusion:
This study shows that preterm infants who receive PN > 28 days have Vitamin A levels that are below the currently accepted range. As Vitlipid N is a fixed preparation, there is no scope of increasing Vitamin A dosage without altering the levels of vitamins D and E, without exposing infants to toxic amounts. The authors recommend that additional studies are required to determine appropriate recommended nutrient intakes for this vulnerable population thereby preventing vitamin A deficiency.

N7

Incidence of evidence of refeeding syndrome

Sara Zaher and Dr.Susan Hill
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Background:
Re-feeding syndrome (RFS) is one of the complications associated with the introduction of parenteral nutrition (PN) especially in under-nourished patients. The primary aim of this review was to determine if RFS occurs among children receiving PN at a specialist children's hospital prescribed in accordance with international guidelines. The secondary aim was to identify the timing of the onset of occurrence of RFS.

Method:
Patients newly starting PN from December 2013-June 2014 were included. Plasma electrolyte levels including phosphate, potassium and magnesium as well as serum albumin levels were monitored over 10 days from starting PN. Information including gender, age, weight and weight for age percentiles were collected for each patient. The definition of under-nutrition was based on the weight for age percentile.

Results:
A total of 97 patients were reviewed. 36 patients or 37% developed hypophosphatemia and 32 patients or nearly 33% developed hypokalemia while only 21 patients or 22% had hypomagnesaemia. Malnutrition at baseline appeared to significantly affect the biochemical marker of RFS, specifically hypophosphatemia, (p-value < 0.014). In contrast, malnutrition before PN initiation did not significantly affect the incidence of hypokalemia or hypomagnesaemia, (p >0.05). Also our results showed that an albumin level of <20g/l was associated with a 2.59 chance of re-feeding hypophosphatemia and 3.21 chance of hypomagnesaemia compared to albumin ? 20g/l. The results indicated that RFS usually occurred between the 2nd and 7th day of PN initiation. Finally, no significant increase in body weight after 10 days of PN compared to baseline was recognized. In contrast, a significant improvement in nutritional status was recorded after 10 days, p-value <0.001.

Conclusion:
RFS is a frequently encountered complication of PN up to 7 days after starting treatment. Professionals caring for patients requiring PN support must recognize the risk factors of RFS.

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G5

A Tale of 2 Meckel's

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Meckel's Diverticulum is a congenital remnant of the omphalomesenteric duct and is the most common gastrointestinal congenital malformation, found in 2% of the population. Whilst most cases are found incidentally on laparotomy, symptomatic Meckel's presents less frequently.

We describe 2 contrasting symptomatic presentations of proven Meckel's Diverticulum. One, a textbook classic, the other a serendipitous find.

Case one
A 3 year old girl presenting with recurrent episodes of painless rectal bleeding. A Meckel's technetium scan proved to be positive with a characteristic spot distant from the stomach. The Meckel's was surgically resected. The patient made a full recovery with no further episodes of bleeding.

Case two
An 11 year old girl with a 2 year history of abdominal pain and refractory anaemia. Faecal calprotectin was raised and inflammatory bowel disease was suspected. Upper and lower gastrointestinal endoscopy was unremarkable, therefore a video capsule endoscopy was performed to further assess the small bowel, which showed inflammation.

The video capsule was retrieved surgically by open laparotomy after failure of passage and found to be sitting in a Meckel's Diverticulum. Following resection the patient's symptoms have fully settled.

In conclusion, of the many cases of suspected gastrointestinal bleeding that are seen in general paediatrics, those with a Meckel's Diverticulum may not present in the characteristic way and prove to be a diagnostic challenge.

G15

Long term outcomes of anti-TNF treated paediatric inflammatory bowel disease patients after transition to adult services

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[1]Child Life and Health, University of Edinburgh, Edinburgh; [2]GI unit Western General Hospital, University of Edinburgh

Background and aims:
Anti-TNF therapy use in paediatric IBD (PIBD) is increasing worldwide with evidence of short-term effectiveness and safety. Longer term outcomes are less clear due to lack of follow-up (FU) of young people after transition to adult services. We aimed to assess effectiveness and safety of anti-TNF therapy in PIBD post-transition to adult care.

Method:
A retrospective case review of PIBD patients treated with anti-TNF (Infliximab (IFX) or Adalimumab (ADA)) in the SE Scotland regional PIBD unit at RHSC, Edinburgh and then transitioned to the regional adult specialist IBD centre at WGH, Edinburgh, during 01/01/00-30/09/13. Data (including outcome and duration of anti-TNF therapy, dose escalation, adverse events and discontinuation/re-start of therapy) were collected prior to, at the time of transition from PIBD services, and to study end point (transfer out of region, loss to FU, death or ongoing adult care at 31/07/2014), a minimum 10 months FU.

Results:
34 children (30 Crohn's disease (CD), 4 ulcerative colitis (UC)) had anti-TNF exposure in PIBD services then a median (range) duration of FU post-transition of 2.9 (0.7-9.3) years. At transition, 19/34 were still on anti-TNF (12 IFX and 7 ADA). 10/12 IFX were in steroid-free remission (SFR) and 8/10 remained in SFR at last FU; 2 discontinued IFX due to loss of response (LoR), both then had ADA with 1 ADA LoR and 1 SFR on ADA at last FU. 1/12 IFX patient with moderate disease at transition then achieved remission, had planned drug withdrawal (PDW) then relapsed, restarted IFX and had mild disease at last FU. 1/12 IFX patients had response with mild disease post-induction IFX but transitioned and had primary non-response (PNR) by 6 doses IFX, and switched to ADA with moderate disease activity at last FU. 3/7 ADA patients at transition were in SFR; 1 stopped as PDW in prolonged remission but 2 stopped due to adverse events. 2/7 ADA patients with mild disease at transition then achieved sustained remission up to last FU. 2/7 ADA patients had moderate disease at transition; one had ADA LoR and moderate disease at last FU, the other gained remission so had PDW, relapsed but was in remission at last FU. There were no deaths or malignancies associated with anti-TNF use.

Conclusion:
Anti-TNF therapy post-transition is effective at maintaining remission in those with remission prior to transition, although some may require a 2nd anti-TNF agent to maintain remission. Those previously not in remission can achieve sustained remission, usually if just starting anti-TNF maintenance at transition; others do not and have a more complicated disease course. Loss of response remains a key reason for stopping anti-TNF therapy.

Conflict of Interest: *BSPGHAN trainee chair

G25

Parent, Patient and Professional Perception of Issues facing Children Living with Stomas

Burdall, Oliver: Bohr, Claire: Spray, Christine: Cusick, Eleri:
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Background:
There has been little formal research into psychological or social issues, rather than surgical complications, facing children with stomas. We aimed to assess what issues patients, parents and the professionals involved felt were important.

Methods:
Thematic analysis from grounded theory was undertaken with purposive sampling through focus groups and interviews for school age children who have had a stoma formed in the last 2 years, their parents and members of paediatric surgical team. Each session included whole group discussion, separate parent group discussion and set tasks and games; including ranking the best and worst things about having a stoma. Discussion was structured to prompt dialogue within the group around key areas: information provided, school life, sport, social life and body image. The format was repeated with members of the paediatric surgical team.

Results:
Twenty-five families were invited of whom 3 attended focus groups or interviews (two boys and one girl, 11-16 years, 3 mums and one dad) and 3 are scheduled to attend in January. Eighteen members of the paediatric surgical team took part. Stoma bag leaks or fear of it leaking were identified as the most significant problem by all children. Issues with body image or clothes, missing school and not being able to do things with friends, in particular sleep overs, were also listed by all children. Despite feeling very well informed preoperatively, they felt the reality differed greatly from the expectations. All families were nervous managing the stoma at home for the first time and none of the children were changing their bags independently. All children had withdrawn socially in some way and stopped playing sports. Mothers identified feelings of loneliness, isolation and guilt following the operation. However, only one child listed skin irritation as a significant issue despite all three having suffered with it and none ranked any surgical complications and repeat visits to hospital as major problems despite two undergoing repeat operations. The surgical teams felt there would be large variation in the patient's social and sporting activities following stoma formation. Issues around body image were listed independently by all of the professional group. The majority also listed fear of being different from their peers and smell in the top three issues these children would face. Only more senior members of the teams identified that stoma leaking and problems with stoma management would be a real problem. The surgeons felt that patients and their families were well informed/prepared upon discharge and that older children would be caring for their stomas independently. The majority of the professional group also listed technical issues, complications or skin irritation amongst the worst problems for a child with a stoma.

Conclusion:
Medical complications were not cited as significant issues for the children, possibly reflecting expectation

G30

Paediatric Inflammatory Bowel Disease Nurses' Experiences of using the Paediatric Inflammatory Bowel Disease Patient Held Record in Clinical Practice

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Background:
The Paediatric Inflammatory Bowel Disease Patient Held Record (PIBDPHR) is a tool that was initially conceptualised to enhance the care and education of children with IBD attending Alderhey Children's Hospital. The PIBDPHR was adopted as a national project by the RCN/BSPGHaN Paediatric IBD nurses group. A literature review was performed and no studies were identified on using a PHR in PIBD. Therefore a study was devised to develop an understanding of how the PIBDPHR was used in practice from the perspective of paediatric IBD nurses.

Methods:
26 paediatric clinical nurse specialists, who participated in the national adoption of the PIBDPHR were identified to take part in a two phased mixed methods study, of which 5 had left their post and 1 was ineligible. Only 1 nurse per paediatric IBD service was invited to participate.

Phase 1 - An e-survey was sent to the eligible nurses (N=20). The survey had 3 sections – demographics, service use and challenges/benefits/solutions. were invited to self-select to participate in Phase 2. Data gathered in Phase 1 was used to inform Phase 2 of the study.

Phase 2 - Interviews with six nurses focused on gaining a deeper understanding and allowed an interrogation of the responses given in phase 1. Data were analysed using descriptive statistics and thematic analysis, as appropriate.

Results:
Phase 1
12 Nurses (60%) completed the e-survey although 1 nurse did not receive any copies of the PIBDPHR. The PIBDPHR was used for newly diagnosed patients (100%), patients who had difficult to manage disease (91%), patient and parent education (64%) and transitional patients (55%).

The nursing challenges in implementing the PIBDPHR included remembering to use the record (55%) and the time involved in using it (36%). Barriers described by the participants included engaging medical staff in their use (45%) and patients forgetting to bring the record to clinic (64%).

Phase 2
6 participants (55%) self-selected to participate in Phase 2.

The PIBDPHR was found to be a good record for blood monitoring (100%), and gave the opportunity to develop a consistency for information giving (100%). 83% of participants cited the rationale for using the PIBDPHR was the need to empower patients.

When interviewed in Phase 2 there was an acknowledgement by 5 participants that they would use the PIBDPHR

G31

Adolescent Colonoscopy Sedation vs General Anaesthetic: what do patients prefer?

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Background:
Colonoscopy is routinely performed in children and adolescents under general anaesthetic (GA). Within our polyposis and inflammatory bowel disease transition service we have developed a pathway for adolescents aged 14-17 allowing them to choose between GA and sedation for colonoscopy. A Standard Operational Procedure (SOP) and patient information leaflets were developed to safely implement this service.

Methodology:
A two stage data collection tool was developed for sedation colonoscopy.

Stage 1 - was completed by the paediatric nurse who was present throughout procedure and reviewed the patient following recovery. Data collected included colonoscopy completion, patient tolerance, length of procedure and overall length of admission.

Stage 2 - A questionnaire, based on recognised satisfaction tools was given to each patient, once they had fully recovered, following the procedure.

Inclusion criteria - Adolescents aged 14 to 17 years who elected to have their colonoscopy under sedation rather than GA between June and October 2014.

Results: Between June 3rd and September 10th 2014 15 adolescents, mean age 16 (14-17) years underwent endoscopy under sedation. 4/15 had previous experience of sedation colonoscopy.

100% caecal intubation (100% TI intubation) was achieved with minimal sedation, maximum dosages of sedation used were 2.5mg midazolam and 50mg fentanyl. There were no adverse events related to the procedure or sedation

The mean duration of colonoscopy was 32 (10-55) minutes, and the mean time to full recovery was 56 mins (0-120mins). Time to full recovery was assessed on the ability of the adolescent to hold a conversation. There was wide variation in time to discharge from procedure completion from 45mins to 240 mins (mean 117mins), this due to different practices in paediatric day care and endoscopy suite.

14 patient experience questionnaires were returned. 78% (11/14) experienced mild or very mild discomfort during the procedure, with the same number reporting no discomfort post procedure. No adolescent described feeling pain during or post procedure.

Patients felt well informed despite only 20% (3/15) having a colonoscopy patient information leaflet prior to the procedure, however 100% of patients said they were given enough information about the procedure. Pre-colonoscopy 50% of patients were anxious about the procedure but post colonoscopy 100% said they would prefer sedation colonoscopy in the future

No visits to the endoscopy unit were undertaken it was not clear whether patients were given the opportunity and declined or were never offered this opportunity.

Conclusion:
All patients within our cohort stated that they would prefer sedation endoscopy in the future. All patients were minimally sedated and could remember the procedure, some in great detail.

There are some areas that need to be reviewed such as written information giving, which may reduce anxiety pre procedure, and the wide variation of time to discharge.

Colonoscopy can be done under sedation in 14 – 17 year age group with good outcomes.

G41

HSP: A spot of bother

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Background:
Henoch Schonlein Purpura (HSP) is the most common systemic vasculitic presentation in paediatrics. Widespread IgA immune complex deposition leads to a classic, well recognised triad of symptoms: purpuric rash, colicky abdominal pain and arthritis. The resulting small bowel oedema can precipitate intussusception in a small number of cases.

Methods:
We describe the clinical course of a 5 year old boy presenting with a 10 day history of colicky abdominal pain, bilious vomiting and hypertension. Initial abdominal ultrasound scan revealed a small bowel mass and possible malignancy was suspected. CT abdomen revealed generalised small bowel wall thickening and oedema, suggesting an inflammatory pathology. Surgical exploration and gastrointestinal endoscopy were considered due to the impressive radiological findings. Following a period of conservative management for bowel obstruction, abdominal symptoms started to settle nearly three weeks after the initial presentation. This coincided with the new onset of a classical non-blanching HSP rash to the lower limbs. Renal function and urinary parameters were normal throughout. Abdominal symptoms resolved with conservative management and he is being followed up to monitor renal parameters.

Conclusion:
This case demonstrates the atypical chronology with which HSP symptoms can manifest, with significant abdominal symptoms and radiological features preceding the onset of classical rash by several weeks. Furthermore, this case illustrates the importance of correlation with the clinical picture when bowel wall thickening is demonstrated. The differential diagnosis of small bowel thickening is varied and includes malignancy, inflammatory bowel disease and vasculitis. Although a high index of suspicion is warranted for malignancy, where findings appear more generalised HSP should be considered even in the absence of the classic features. A degree of tenacity is required to monitor for an evolving HSP picture, avoiding unnecessary invasive procedures.

N5

Use of body composition measurements in paediatric patients: identifying malnutrition and predicting clinical outcomes

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Background:
Sick children have a high risk of malnutrition both on admission and during their hospital stay. Weight (WT) and height (HT) are commonly used for screening and nutritional management of these children. However, these measurements do not distinguish between the different body tissues that might shift drastically in disease1 and influence the response to treatment and patient recovery2. Thus, measurements of body composition (BC) have been proposed to better identify children at risk of malnutrition and guide nutritional management more effectively.

Objectives:
To determine baseline BC in children admitted to a tertiary paediatric hospital, and examine associations with length of stay (LOS) and weight loss during admission.

Methods:
Children aged 5-18yrs admitted to a tertiary referral hospital with expected stay >3 days had measurements of WT, HT and BC (LM by Dual Energy X-ray Absorptiometry (LMDXA) and bioelectric impedance analysis (LMBIA); FM by DXA and 4-site skinfold thicknesses (SFT)) within 48 hours of admission. Standard deviation scores (SDS) were calculated from UK BC reference data3. Diagnosis, nutritional management, predicted LOS and mobility were recorded, with WT and actual LOS at discharge.

Results:
128 children (mean age 10.7yr; 49.2% male) were studied; 54.7% from medical and 45.3% from surgical wards, most with multiple and chronic diagnoses. Median LOS was 7 (range 1-65) days. On admission (Table 1) children had significantly low HT and LM, with high BMI and SFTs. 19% had LMDXA <-2SDS, compared to 12% LMBIA and WT, and 2% BMI.

	n	Min	Max	SDS a	<-2SDS b	>2SDS b	Acceptability c
HT	121	-5.54	2.26	-0.62 (1.5)*	17	2	95
WTN=31	128	-5.62	4.62	-0.18 (1.7)	12	7	
BMIN=18	121	-3.38	5.38	0.30 (1.4)*	2	12	
Biceps SFT	102	-2.2	2.17	0.46 (0.9)*	1	2	65
Triceps SFT	103	-3.14	1.97	0.17 (0.9)	1	0	
FMDXA	103	-3.24	3.39	0.19 (1.2)	5	6	93
LMDXA	103	-5.58	1.98	-0.91 (1.5)*	19	0	
LMBIA	90	-4.14	2.28	-0.72 (1.3)*	12	2	90

* 1-sample t-test, $p<0.05$; a mean(SD); b %; c score/100

Lower WT and LMDXA were predicted by immobility (wheelchair use), and artificial/liquid parenteral or enteral nutrition (EN/PN). Lower BMI, FM, and LMBIA were also predicted by EN/PN; with a greater effect for full versus partial EN/PN. Baseline WT, BMI and BC did not correlate significantly with actual or predicted LOS. Children with higher baseline WT, BMI and FM lost more WT by discharge.

Conclusions: Children admitted to this tertiary paediatric hospital are short with abnormal BC characterized by low LM and variable FM, frequently not apparent from WT/BMI, with implications for nutritional management. DXA and BIA were feasible and acceptable techniques whilst SFT were less so. Immobility and nutritional support were the most significant predictors of abnormal BC. BC measurements did not correlate with LOS and WT loss during admission, probably reflecting the limitations of generic outcomes for this heterogeneous group. Further research will target specific patient groups using disease-specific outcomes to establish the usefulness of BC measurements for the nutritional management of these children.

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BSPGHAN 2015 Annual Meeting

WEDNESDAY 28TH JANUARY
Poster abstracts

Team 3 Poster Walk

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Self bougienage in children with oesophageal stricture: A case series and video demonstration
IN CHILDREN WITH OESOPHAGEAL STRICTURE; A CASE SERIES

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Background:
Oesphageal strictures in children are usually benign. Aetiology include oesophageal atresia (57%); caustic ingestion (21%) and peptic oesphagitis (12%).The initial treatment for long segment oesophageal strictures is either balloon dilatation or surgical dilatation. Most need multiple dilatations with associated complications related general anaesthesia and oesophageal perforation. We report 3 children who were successfully trained to swallow esophageal dilators as an alternative treatment modality for this complex problem.

Methods:
All these children required multiple dilations on regular intervals. All children were taught to swallow the chosen Maloney Dilator by a consultant paediatric surgeon (SP). Cases 2 and 3 were also able to view a video demonstration by patient 1.

Results:

Case 1:
A 15 year old girl, with underlying VATER anomaly, had an oesophageal stricture following trachea-oesophageal fistula repair. She had a 4 cm mid-oesophageal stricture which was further aggravated by a significant oesophageal dysmotility. She has had multiple oesophageal dilatations since the first year of life. Self bougienage was started at the age of 8, and since she has not needed any further surgical dilatations. She has suffered occasional episodes of odynophagia. Upper endoscopy has shown no recurrence of stricture.

Case 2:
An 11 year old boy diagnosed with Barrett's oesophagus at 7 years of age and an oesophageal stricture secondary to ulcerative oesophagitis. He required multiple balloon dilatations, most recently in October 2013. He was trained to self dilatation and discharged with a size 28 Fr dilator. He has remained asymptomatic since.

Case 3:
A 15 yr old boy diagnosed with ulcerative oesphagitis at age of 12 years developed a 6 cm lower oesophageal stricture confirmed by barium and endoscopy; Oesophageal biopsies were normal. He required recurrent balloon dilatations. He started performing self-bougineage at 14 years of age. At present he does his self-dilatations 1-2 per week and remains totally asymptomatic.

We were unsuccessful in training a young 7 year old boy recently.

Conclusion
With appropriate patient selection and careful instruction oesophageal self-dilation is a safe and effective alternative treatment to current balloon or surgical dilatations. We have shown that such dilatations could be started as early as eight years of age.

G16

A new method to estimate catheter length for oesophageal multichannel intraluminal impendance monitoring in children

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Objectives and Study:
Multichannel intraluminal impedance combined with pH (MII-pH) is the gold standard test for diagnosing gastro oesophageal disease. Accurate catheter placement is essential to prevent erroneous recording of reflux events. In this study, we proposed a simplified method (GOSH Table) to estimate the length of insertion of MII-pH catheter from nose trill to the point where the pH sensor is approximately two vertebral bodies above the diaphragm. We compared our results to Strobel and Monreau formulae.

Methods:
Retrospective data were collected from children who underwent MII-pH studies in the department of gastroenterology at Great Ormond Street Hospital between January to October 2014, including plain X-ray after initial catheter insertion and the position adjusted to align the pH sensor at two vertebral bodies above the diaphragm (desired catheter position). Children were divided into three age groups G1: 1 month to 3 years; G2: 3-10 years and G3: over 10 years.

Results:
One hundred and forty four children were included with mean age was 5.1 (±4.5) years, 73 males and 71 females. The data were analysed as correlation (r) and mean difference (MD) between Strobel formula, Moreau formula, GOSH table and desired catheter position (DCP). The results are shown in table

	N	Correlation, r	p-value	Mean of Differences	95%CI. of the Difference	
					Lower	Upper
Total	144					
DCP - Strobel*0.87		0.843	<0.001	-1.6	-9.0	5.9
DCP - Moreau*0.87		0.851	<0.001	-0.4	-6.4	5.6
DCP - GOSH		0.951	<0.001	-0.6	-4.0	2.8
G1	67					
DCP - Strobel*0.87		0.555	<0.001	-0.4	-7.3	6.5
DCP - Moreau*0.87		0.591	<0.001	-0.1	-5.5	5.2
DCP - GOSH		0.915	<0.001	-0.3	-2.9	2.4
G2	50					
DCP - Strobel*0.87		0.816	<0.001	-2.4	-6.7	2.0
DCP - Moreau*0.87		0.816	<0.001	-0.8	-4.5	3.0
DCP - GOSH		0.784	<0.001	-0.9	-4.6	2.8
G3	27					
DCP - Strobel*0.87		0.430	0.025	-2.9	-14.1	8.3
DCP - Moreau*0.87		0.430	0.025	-0.4	-10.2	9.3
DCP - GOSH		0.809	<0.001	-0.9	-4.9	3.2

Conclusion:
GOSH Table is an accurate method to estimate the insertion length of MII-pH catheters from nares to a point of approximately two vertebral bodies above the diaphragm in children. Although radiography is required to confirm final catheter position, using GOSH Table will reduce the need for repeated catheter manipulation after initial insertion and will eliminate the use of a mathematically complicated formulae

G28

A Novel RFX6 mutation in a child with neonatal diabetes, annular pancreas, gallbladder aplasia and intestinal atresia

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Introduction:
Mitchell Riley syndrome is a rare condition caused by homozygous or compound heterozygous mutations in the RFX6 gene on chromosome 6q22. It is characterised by neonatal diabetes, intestinal atresia, pancreatic abnormalities, and biliary hypoplasia. 9 patients with RFX6 mutations and Mitchell Riley syndrome are reported in the literature, most of whom had a poor clinical course, 5 of the 9 dying before 6 months of age.

We report a case with a novel RFX6 intronic mutation leading to the Mitchell Riley syndrome.

Case Description:
An antenatal diagnosis of duodenal atresia was made on the second twin in a dichorionic diamniotic pregnancy (in vitro fertilisation). On day 2 of life, at planned laparotomy, she was noted to have an annular pancreas and absent gallbladder. Preoperatively, pigmented stool and bile had been noted, but postoperatively she became acholic with a rising conjugated hyperbilirubinaemia.

Subsequently intraoperative cholangiogram revealed a patent but hypoplastic biliary tree with absence of gallbladder. Liver histology on day 51 showed ductular and lobular cholestasis with marked hepatocellular, sinusoidal, and portal siderosis. Immunostaining for insulin, glucagon & somatostatin was negative in the heterotopic pancreatic tissue isolated in the duodenal specimen.

The child had refractory blood glucose levels from birth with undetectable C-peptide at 2 months of age.

Feed intolerance with frequent loose stools and poor weight gain were also problematic. Gastrointestinal biopsies on day 174 showed focal active inflammation, villus atrophy and borderline epithelial tufting, with gastric and large bowel eosinophilia. Pancreatic exocrine insufficiency was excluded. By 6 months of age only 30% of calories were tolerated enterally and she remains PN dependent. With the clinical phenotype a mutation in RFX6 was suspected, however initial testing for previously reported mutations was normal. Subsequent testing discovered a novel intronic mutation, c.1556-40T>G. This mutation is predicted to create a cryptic splice acceptor site in intron 14 and cause aberrant splicing

Discussion:
The RFX6 gene encodes a winged helix transcription factor that is expressed in the developing pancreas and in the gut endoderm. It is downstream of NGN3 and studies in mice suggest that this gene is specifically required for the differentiation of islet cells for the production of insulin. There are nine previous case reports of RFX6 mutation in the literature. There appears to be genotype-phenotype correlation, with the longest surviving cases (9 years and 6 years respectively) being a compound heterozygote and a milder homozygous variant.

At 8/12 of age the infant reported here has done well. The conjugated hyperbilirubinaemia has resolved, enteral feed tolerance is slowly improving, and blood glucose is controlled via a subcutaneous insulin pump (HbA1c 6.3%) with near normal developmental progress. Our case may suggest that this genotype confers a milder phenotype, or that with careful multidisciplinary management of each affected system, a good outcome may be achieved.

G34

How Quickly Should We Endoscope Children With Haemophilia Who Present With Gastrointestinal Bleeding?

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Background:
Gastrointestinal (GI) bleeding is an uncommon presentation in children with haemophilia. Clinicians may be dismissive of gastrointestinal bleeding in this group due to the false assumption that their bleeding is secondary to their underlying coagulation disorder. However, usually it is secondary to genuine gastrointestinal pathology and the resultant bleeding has the potential to be life-threatening. We aimed to review children with haemophilia presenting with GI bleeding to a tertiary paediatric gastrointestinal unit in order to gain a better understanding of GI bleeding in this condition.

Method:
The case notes of children with haemophilia presenting to a tertiary gastrointestinal unit with GI bleeding over a 7 year period (2005-2012) were retrospectively reviewed.

Results:
Five patients were identified: all had haemophilia A and two were classified as severe and on prophylaxis therapy with recombinant Factor VIII. The median age at presentation was 9 years (range: 2 -15 years). Four patients presented with melena and one with epistaxis and haematemesis, they all had low haemoglobin at presentation. One patient was clinically unstable during admission requiring resuscitation with fluid and blood transfusion. All patients underwent upper gastrointestinal endoscopy: four had a duodenal ulcer, 2 of whom were positive for rapid urease test (CLO). All patients responded to medical treatment and had normal endoscopy findings 3 months after treatment. The remaining patient had a normal endoscopy and a subsequent positive Meckel's scan, his Meckel's diverticulum was surgically removed with resolution of bleeding.

Conclusion:
Gastrointestinal bleeding secondary to haemophilia is rare. Erosive gastritis and duodenal or gastric peptic ulcers with or without Helicobacter pylori infection is the likely cause of bleeding in these patients. We recommend early endoscopy in association with helicobacter pylori testing aiming to initiate timely and appropriate treatment and avoid life threatening bleeding in these patients.

H7

Body image perception in young people with chronic liver

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Purpose:
Body image (BI) is a normative discontent in adolescence. Researchers have hypothesised that the physical effects of chronic liver disease (CLD) and its treatment would exacerbate BI dissatisfaction in this population, but this has never been investigated.

Methods:
The study investigated BI in young people (mean age 19.1 years) with CLD of 'transition age' (16-24 years). 4 validated questionnaires were used to assess these constructs in 80 young people (42 female) with various diagnoses of CLD; the Multi-dimensional Body Self-Relations Questionnaire (MBSRQ), the Screening Tool for Psycho-social Distress (STOP-D), The Coping Responses Inventory (CRI) and the Basel Assessment of Adherence Scale to Immunosuppressives (BAASIS). The role of surgical scars and immunosuppressive medication side-effects in appearance dissatisfaction was examined using linear regression. The study also investigated the association between medication non-adherence with appearance dissatisfaction, psycho-social distress, and coping response style.

Results:
Compared to the general population young females with CLD are less satisfied with their overall appearance. Young males are more dissatisfied with discrete parts of their body (particularly muscle tone and mid-torso) but not their overall appearance. No evidence was found that immunosuppressive medication side-effects or scarring from surgery impacts upon BI. High levels of psycho-social distress were reported; 46% screened positively for depression, 51% for stress, 39% for anxiety, 23% for anger and 18% for perceived lack of social support. Young people who reported feeling more vulnerable to physical illness (Health Evaluation) reported higher psycho-social distress. Just 26.7% reported full adherence to their immunosuppressive regimen over the past 4 weeks, which challenged comparisons between 'adherent' and non-adherent' individuals.

Conclusion:
BI perception is poorer in young people with CLD compared to the general population and our findings indicate high rates of non-adherence. Psycho-social factors should be considered alongside physical indicators of health. Detection of, and subsequent early intervention for issues could prevent these impacting upon the self-management of their condition and potentially influence their overall outcome.

H16

Blau Syndrome: A rare cause of Hepatosplenomegaly

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Background:
Blau syndrome (BS) is a rare granulomatous inflammatory condition first described in 1985. BS presents with a recognized classic triad of arthritis, skin rash and uveitis. It is caused by mutations in the nucleotide-binding oligomerization domain containing 2 (NOD2) gene located on the long arm of chromosome 16. Mutations may be sporadic or inherited in an autosomal dominant fashion.

We present a case of a 5 year-old girl referred to our department for investigation of hepatosplenomegaly. She was diagnosed with JIA a year prior following a 6-month history of general malaise, lethargy and joint swelling. Clinically, she had hepatosplenomegaly with tenosynovitis of her wrists and ankles. Biochemically, he liver function tests were completely normal. Her inflammatory markers including erythrocyte sedimentation rate and serum amyloid A were elevated. She was subsequently treated with corticosteroids and methotrexate.

Methods:
We performed a complete liver work-up including liver function tests, creatine kinase, serum autoantibodies, immunoglobulins, complement levels, caeruloplasmin, urinary penicillamine challenge, angiotensin converting enzyme levels (ACE), viral serology and liver biopsy. Biochemically, the only positive findings were of a mildly raised ACE, positive anti-SLA antibodies (low titres) and elevated complement C3 levels. The liver biopsy revealed portal granuloma with mild inflammation.

Her father was similarly diagnosed with JIA aged 6 and remains on treatment for arthritis. He developed liver disease aged 22 and liver biopsy revealed portal granulomas with fibrosis. A putative diagnosis of Sarcoidosis was made and he was treated with corticosteroids. 5 years later; his liver disease has progressed with severe portal hypertension with variceal bleeding and cholestasis. A repeat biopsy demonstrates an obliterative venopathy with granulomatous cholangiopathy.

Results:
Given the similiarities in presentation, the presence of granuloma in his liver biopsy and the progression of liver disease. We suspected a genetic cause of her hepatosplenomegaly. Our search led us to suspect a diagnosis of BS.

Genetic analysis was subsequently performed and she has been found to have 2 pathogenic variants in her NOD2 gene confirming BS. Her father's genetic results are currently awaited.

Conclusions:
To date, there is no evidence for the effective treatment of BS. Patients have been managed with various combinations of corticosteroids, methotrexate and monoclonal antibodies to tumour necrosis factor and interleukin-1.

Our patient has not suffered a rash or uveitis, the commonest and most significant cause of morbidity in BS. The progression of liver disease in her father is worrying and poses difficult questions for her future immunosuppressive management.

In conclusion, a diagnosis of BS should be suspected in children with evidence of arthritis, systemic granuloma with or without hepatosplenomegaly

N6

Measuring weight and height on admission to hospital - Practice in the UK and Ireland

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Background:
Malnutrition is known to impair physical and mental health as well as recovery from disease and therefore leading to an increase in mortality, hospital stays and hospital costs1. Young children and infants are categorised as those being at high risk of malnutrition as well as those patients with gastrointestinal, respiratory and renal disease2. Early identification of malnutrition in children admitted to hospital is core to timely intervention and improved outcomes. The Royal College of Nursing (RCN) have issued a position statement that calls for all nurses to use triggers, including anthropometric and dietary information, to identify children at risk of malnutrition3.

Aim:
To investigate the relationships between reported nutrition practices and the prevalence of malnutrition in the admitted children. Secondly, variation in practice since 2011 was explored.

Methods:
The multi-centred, multi-national nutrition audit, the Children's Nutrition Survey (CNS) collected data on the nutrition related practices of specialist and general children's units within the UK and Ireland. In addition anonymous routine nutrition related clinical data were recorded for children admitted over a 72 hour period in April 2011-2013. Data includes age, gender, admitting diagnosis, anthropometric measurements and dietary pattern information. Data from three years (2011-2013) was collated in SPSS v22 and descriptive analysis is reported here.

Results:
A total of 63 centre reported on nutrition related practices and 2114 anonymised routine clinical data sets within the period 2011-2013. Weight was reported as being recorded the majority of the time; however recording of height on admission was reported to be infrequent (Table 1). When the routine clinical data was investigated this trend was further supported with 91% (N=1933) of children having a recorded weight but only 41% (N=861) having a recorded height. Acute under-nutrition, as defined using a weight z-score of -2SD, was seen in 10% (N=192) of cases while chronic under-nutrition, as defined by a height z-score of -2SD, was seen in 11% (= 93).

Table 1. Nutrition related practices as reported in the CNS for 2011- 2013*

	Are nutrition related practices audited		Plan for management of patients at risk of malnutrition		Is there training on referral criteria to dietetics		Are patients weighed on admission		Is height routinely recorded on admission	
	Yes N (%)	No N (%)	Yes N (%)	No N (%)	Yes N (%)	No N (%)	Yes N (%)	No N (%)	Yes N (%)	No N (%)
Total N=63	25 (39.7%)	38 (60.3%)	30 (47.6%)	33 (52.4%)	36 (57.1%)	27 (42.9%)	62 (98.4%)	1 (1.6%)	25 (39.7%)	38 (60.3%)
2011 N=31	9 (29.0%)	22 (71.0%)	14 (45.2%)	17 (54.8%)	18 (58.1%)	13 (41.9%)	30 (96.8%)	1 (3.2%)	13 (41.9%)	18 (58.1%)
2012 N=18	9 (50.0%)	9 (50.0%)	8 (44.4%)	10 (55.6%)	9 (50.0%)	9 (50.0%)	18 (100.0%)	0 (0.0%)	7 (38.9%)	11 (61.1%)
2013 N=14	7 (50.0%)	7 (50.0%)	8 (57.1%)	6 (42.9%)	9 (64.3%)	5 (35.7%)	14 (100.0%)	0 (0.0%)	5 (35.7%)	9 (64.3%)

(10 (0.0(35. (64 (64.(35(50. (50 (57. (42 N= 0.0)) 7) .3) 3) .7) 0) .0) 1) .9) 14

*Values reported as N(%)

The poor compliance with basic procedures such as weighing and measuring children on admission to hospital will result in limited identification of under-nutrition. This could have significant impact on planning the appropriate clinical and nutritional management of these children with increased clinical, financial and personal consequences. The poor use of audit may be compounding this issue.

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BSPGHAN 2015 Annual Meeting

THURSDAY 29TH JANUARY
Poster abstracts

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G4

An inverse relationship between H pylori infection rates and the incidence of IBD: Could this be contributory to the rising incidence of PIBD?

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Background:
Accumulating evidence suggests that H. pylori infection is protective against several autoimmune conditions. This effect may be through steering away the host immune machinery from a pro-inflammatory Th1/Th17 response and promoting a regulatory T-cell environment [1]. A meta-analysis of 23 studies suggested a protective role of H pylori infection against IBD development and an inverse relationship between H pylori infection and IBD has been suggested [2]. The overall prevalence of H pylori infection in developed countries is less than 10% compared to up to 50% in developing countries. In this study, we present the prevalence of H pylori infection in a PIBD research cohort and explore its relationship to the rising incidence of IBD. We also assessed the prevalence of other gastro-intestinal infections one year preceding the onset of IBD.

Methods:
Cases of PIBD were identified from an existing research database, recruiting patients from a geographically defined area in Southern England. The epidemiological data on the prevalence of the infection prior to the onset of IBD were obtained from the research database recorded prospectively at the time of recruitment.

Results:
254 Paediatric patients with IBD were identified, 59% with Crohn's disease, 31% ulcerative colitis and 10% IBDU. All patients had IBD diagnosed over the last 15 years (1999-2014). H pylori infection was diagnosed in 2 patients (<1% of patients) preceding the onset of IBD. The other gastro-intestinal infections diagnosed in patients preceding the onset of IBD by one year was as follows: C-difficile-3 patients, Salmonella-1, giardia-1 and none with shigella and campylobacter.

Conclusion:
The prevalence of H pylori infection in our cohort was less than 1%. This is in keeping with the overall trend of falling rates of H pylori infection in Western Europe. Through a previous study from our centre, we have demonstrated that the incidence of PIBD has increased by almost 50% over the last decade[3]. Whilst the reasons for this increase remain unclear, our data is reflective and supportive of the view that diminishing rates of H pylori infection in the developed countries may be contributory to the rising incidence of IBD.

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G14

Incidence of Paediatric Inflammatory Bowel Disease in Scotland continues to rise

**Cameron, Fiona L[1]; Henderson, Paul[1]; Jagger, Fiona[2]; Rogers, Pamela[3]; Hansen, Richard[4]; McGrogan, Paraic[4]; Loganathan, Sabari[5]; Russell, Richard K[4]; Wilson,David C[1];*
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Background and aims:
The worldwide incidence of paediatric-onset inflammatory bowel disease (PIBD) is rising, with Scotland having the highest rate in the UK. Scottish PIBD data over the last 40 years has shown a consistent increase, including a 76% rise over 13 years around the millennium (1). The aim of this study was to calculate current PIBD incidence rates in Scotland and to determine if the temporal trend of significant increase has been maintained.

Methods:
Historical data from 2003-2008 (cohort 1) was compared to prospective, nationwide data of all incident cases diagnosed in paediatric services (under 16 years of age) from 2009-2013 (cohort 2). Age-sex adjusted incidence rates were calculated using population data from the General Registrar's Office for Scotland. Cases were classified as Crohn's disease (CD), ulcerative colitis (UC) or inflammatory bowel disease unclassified (IBDU) and diagnosed according to the Porto criteria. Statistical analysis was performed using Poisson regression.

Results:
A total of 436 patients were diagnosed with PIBD over six years in cohort 1 (265 CD, 115 UC, 56 IBDU) compared to 478 children over five years in cohort 2 (286 CD, 126 UC, 66 IBDU). Median age at diagnosis in cohort 2 (60% males) was 12.3 years, similar to cohort 1 (58% males) at 11.9 years. The adjusted incidence rate increased from 7.8/100,000/year (95%CI 7.1-8.6) in cohort 1 (2003-2008) to 10.4/100,000/year (95%CI 9.6-11.5) in cohort 2 (2009-2013) ($p<0.001$). This significant increase was also seen individually for CD (4.7/100,000/year [95%CI 4.2-5.4] compared to 6.3/100,000/year [95%CI 5.6-7.0][$p<0.0001$]) and UC (2.1/100,000/year [95%CI 1.7-2.5] compared to 2.7/100,000/year [95%CI 2.3-3.3][$p=0.009$]). There was a non-significant increase in IBDU from 1.0/100,000/year (95%CI 0.7, 1.3) in cohort 1 to 1.4/100,000/year (95%CI 1.1, 1.8) in cohort 2 ($p=0.07$).

Conclusion:
There continues to be an ongoing rise in incident PIBD (and both CD and UC) in 2009-13 in this national, population-based study compared to recent historical data, with a further significant rise of 33%. The reasons behind this continued increase remain unclear and further research is needed to elucidate potential factors in aetiopathogenesis.

References:
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Conflict of Interest: BSPGHAN trainee chair

G23

Has the rising incidence of paediatric inflammatory bowel disease in South Wales stabilised?

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Introduction:
The incidence of paediatric inflammatory bowel disease (IBD) has risen significantly across Europe in the last 20 years, although our own Welsh data had suggested that this had plateaued by 2004. We have now studied the data from the same area over the last decade and compared them with our previous published studies from South East Wales (1983-2003).

Methods:
All cases of IBD < 16 years of age residing in a defined location within South Wales (Cardiff and Vale) were prospectively recorded from January 2004 to March 2014. The incidence, age, gender and disease type were analysed and compared to our data from 1983-2003 from the same region.

Results:
Between 2004 and 2014, there were 57 new patients compared to 39 (1996-2003) and 28 (1983-1993). The overall incidence of IBD was 5.9 per 100,000 per year, Crohn's disease (CD) 3.7 per 100,000 per year and Ulcerative colitis (UC) 2.07 per 100,000 per year compared with 5.4/100,000 for 1996 to 2003. There is no statistically significant difference between the two time periods (p value of 0.675). The median age at diagnosis remains at 12 years with a male- to- female ratio of 1.7:1.

Conclusion:
The incidence of paediatric IBD in a defined geographical area within South Wales has remained similar for more than 15 years with a slight increase in the incidence of UC, suggesting that the previous exponential rise in incidence has reached a stable state.

H1

Is liver biopsy essential for diagnosis in patients with Inflammatory Bowel Disease and abnormal liver tests?

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Introduction:
Liver disease is a recognized extra-intestinal manifestation of inflammatory bowel disease (IBD).

Aims:
To describe characteristics of patients with abnormal liver tests at the time of initial diagnosis of IBD in a single tertiary centre over the last 11 years and to evaluate the role of liver biopsy.

Methods:
Patients were identified from the departmental endoscopy register and the electronic patient administration system using ICD-10 classification. Retrospective data was collected from patient notes. Patients underwent standard blood tests and autoantibodies, immunoglobulins, hepatitis screen, and alpha-1-antitrypsin deficiency. All patients underwent liver ultrasound (USS) +/- magnetic resonance cholangiopancreatography (MRCP). Some patients underwent liver biopsy.

Results:
16 patients were identified (9-females). 12 had ulcerative colitis, 3 indeterminate colitis and 1 Crohn's disease. Eleven patients were diagnosed with sclerosing cholangitis (SC). 5 patients were positive for smooth muscle antibody (SMA positive). 10/12 patients had bile duct abnormalities identified at USS and/or MRCP and were diagnosed with SC. 2/10 patients had advanced liver disease with splenomegaly and fibrosis/cirrhosis on USS. One of these 2 patients underwent liver transplant 7 years later. 8/10 patients, underwent liver biopsy confirming a histological diagnosis of SC in keeping with changes seen on imaging. One patient with normal imaging, except calculi (SMA positive) was diagnosed with SC following liver biopsy. One patient (SMA positive) was presumed to have SC, despite normal imaging and did not undergo liver biopsy initially. She was maintained on a small dose of steroid and ursodeoxycholic acid but underwent liver biopsy 6 years later despite normal LFT's because of thrombocytopaenia after restarting azathioprine and was found to have liver fibrosis. The remaining 4 patients had transient transaminitis, one had pancreatitis (SMA positive) and another was diagnosed with Coeliac disease at the same time as IBD. No other autoantibodies were positive in any of the patients.

Conclusion:
In patients with IBD, the diagnosis of SC based on abnormal imaging (USS +/- MRCP) was not altered by liver biopsy.

H10

Liver transplantation for Undifferentiated Embryonic Liver Sarcoma (UELS)

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Background:
UELS is a rare aggressive tumour which represents 5-15% of all childhood malignant liver tumours and is more common in boys. Treatment options include chemotherapy, neoadjuvant or postsurgical resection, partial hepatectomy or liver transplantation. Historically outcomes have been poor with <37.5% 5 year survival. We present a case of an 11 year old girl who was managed successfully with a liver transplant and remains well 1 year post liver transplantation.

Method:
Case study.

Results:
A previously well 11 year old girl presented to her local hospital with a 3-4 week history of progressive lethargy, anorexia, worsening nausea and abdominal pain. On clinical examination there was no abdominal distension. She had mildly abnormal LFTs (Total bilirubin 29 umol/L, conjugated bilirubin 11 umol/L, ALT 129 IU/L, GGT 187 IU/L, AST 113 IU/L, LDH 669, total protein 76 g/L, albumin 31 g/L, INR 1.3, afp and HCG negative) and her ultrasound revealed "a complex mass in the right liver lobe measuring 15cm in the max diameter. The lesion contains foci of cystic changes, but is predominantly solid and minimally vascular". Her CT showed "a large low density oval mass 14.7 by 10.9 by 10.4 cm in the right lobe with a higher density component of 6cm. Hepatic vessels are stretched round the tumour margins particularly medially. Hepatic and portal veins enhanced normally. Normal kidneys and lungs". She was therefore referred to our unit for a tertiary hepatology opinion.

Liver biopsy of the mass showed malignant mesenchymal neoplasm consistent with undifferentiated (embryonal) sarcoma. The tumour was deemed unresectable and she was started on anti Rhabdomyosarcoma chemotherapy aiming to reduce tumour size. She suffered various side effects including PRES syndrome.

Reimaging after 2 courses showed tumour shrinkage and it was thought to be resectable. On laparotomy there was infiltration of the left hepatic vein by the tumour with no clear resectable margins and tumour free liver looked steatotic. The tumour was deemed unresectable with no extra hepatic spread so she was listed for liver transplantation.

After 2 more cycles of chemotherapy and 8 months from the diagnosis the child was transplanted with a whole liver with 'piggy back' implantation. The explant histology showed an embryonal sarcoma forming a single 115 mm mass with microvascular invasion. The background liver had severe steatosis (70%) and bridging fibrosis.

She was started on standard immunosuppression (tacrolimus and prednisolone). She had 2 further courses of chemotherapy post LT. One month after the transplant she developed biliary stricture (anastomotic) which was stented. Repeat ERCP after 3 months showed good bile flow with no evidence of stricture so stent was removed.

Subsequently the child, one year after her transplant, has been very well, with normal liver function tests.

Conclusion:
Liver transplantation along with pre and post transplant chemotherapy is a viable treatment option for children presenting with unresectable UELS with no extra hepatic spread

H11

Four component (4C) body composition in hepatopulmonary syndrome (HPS).

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Background:
Weight loss, loss of muscle and hypermetabolism are predictors of worse outcomes for children awaiting liver transplant. This is the first time that body composition measured by the 4C model is reported on children with HPS. The 4C model is the gold standard for in vivo differentiation of fat and fat free mass.

Methods:
A 10 year old girl and a 5 year old boy awaiting liver transplant had their body composition measured. The girl had neonatal sclerosing cholangitis and the boy had biliary atresia. Both had HPS. The 4C model uses the following equations:

FM= (2.747 X BV) – (0.710 X TBW) + (1.460 X BMC) – (2.050 X WT)

FFM= WT- FM

FM is fat mass, BV is body volume measured by BOD POD, TBW is total body water measured by deuterium stable isotope, BMC is body mineral content measured by DXA scan, WT is weight and FFM is fat free mass.

The children had their resting energy expenditure (REE) measured by indirect calorimetry and compared to the predicted REE estimated by the FAO/WHO/UNU 1985 equations. Hypermetabolism was defined as measured REE ? 120% predicted REE.

Results:
Both children had portal hypertension with splenomegaly and hypersplenism and no ascites on ultrasound. Both had synthetic failure (F: albumin 35 g/L, INR of 1.38, M: albumin of 29 g/L, INR of 1.43) and mild jaundice (F: total bilirubin 71 umol/L, M: 29umol/L). The girl's shunt with a macroaggregated albumin scan was 45% total (18% without kidneys) whilst the boy's was 11% (5%). The girl had been diagnosed with HPS 14 months prior to the assessment, whilst the boy had only just been diagnosed.

The girl's BW was 31.8kg and Ht was 1.55m. BMI was 13.25 (0.4th centile). FM was 4.7kg (9.2th), FFM 27.1kg (50th-74.8Th). FMI was 1.95 (2.3rd) and FFMI 11.3 (2.3rd-9.2nd).

The boy's BW was 21.7kg and Ht was 1.12m. BMI was 17.36 (75th-91st). FM was 6kg (90.8th) and FFM 15.7 (25.2nd). FMI was 4.8 (25.2nd) and FFMI (9.2nd-25.2nd).

Table 1 Resting energy expenditure (REE) and respiratory quotient (RQ).

	Measured REE (Kcal)/predicted REE (Kcal)	Wt (Kcal/kg)	REE/FFM (Kcal/Kg)	RQ
Female	16625 (1215)	51.1	60	0.72
Male	1179 (988)	54.3	75.1	0.75

Conclusion:
Liver disease is frequently accompanied by organomegaly and fluid retention making basic anthropometric measurements meaningless. The girl has a very low BMI and the 4C model confirms she has a very low FM with a relatively preserved FFM. She is hypermetabolic with an RQ indicating lipid oxidation. The boy has a misleadingly reassuring BMI. REE/FFM shows a significant increase to his Kcal/Kg expenditure that would otherwise not be realised and may indicate he is at risk for future accelerated weight loss. Whereas more detailed body composition studies are required in these patients, these 2 cases indicate the need for early nutritional intervention.

N8

Creatinine as a measure of lean body mass in children with chronic disease in a tertiary hospital population.

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Introduction and Aims:
Creatinine may be used in the paediatric clinical setting as a proxy for lean mass (LM) and as a measure of protein malnutrition. There is limited data to support this in practise, with only two paediatric studies identified, both of children with oncological diagnoses [1, 2]. The aim of this study was to identify any association between serum creatinine measurements and weight (kg) and LM measured by dual energy x-ray absorptiometry (DXA) scans. The term LM here includes both lean and bone mineral content from DXA.

Methods:
109 children were identified who were involved in a broader study of body composition in a tertiary hospitalised population. Patient notes were used to identify serum creatinine measurements at admission. Using Pearson's correlations the creatinine measurement was then correlated with body mass and body composition variables (LM and fat mass (FM)).

Results:
A total of 78 children were included: 43 (55%) female and 35 (45%) male. The mean age was 10.7 years (5 – 15.8 years). 31 patients were excluded (5: renal diagnosis, 5: no creatinine result available, 21: no DXA result).

Correlation with creatinine	Pearson coefficient	P value
Weight (kg)	0.57	<0.001
Lean body mass (Kg)	0.59	<0.001
Lean body mass (kg) adjusted for age and sex	0.26	<0.05
Fat mass (kg)	0.45	<0.001
Fat mass (kg) adjusted for age and sex	0.11	NS
Lean mass (kg) adjusted for FM, age and sex	0.24	<0.05

Conclusions:
Serum creatinine concentration was seen to correlate with weight, LM and FM. There was a stronger correlation with LM than FM, and this remained significant after adjusting for age and sex, although the correlation was weaker. The correlation between creatinine and LM also remained significant after adjusting for FM.

These data suggest that creatinine is more strongly correlated with LM than FM. However, after taking into account age and sex, the correlation is weak. Caution is

therefore required when using creatinine to monitor a child's nutritional status/lean mass, although it may be a useful adjunct to the clinical assessment.

References:
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[2] Rayar M, et. al., Sarcopenia in children with acute lymphoblastic leukemia. J Pediatr Hematol Oncol. 2013 Mar;35(2):98-102.

BSPGHAN 2015 Annual Meeting

THURSDAY 29TH JANUARY
Poster abstracts

Team 2 Poster Walk

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G8

Upper Gastrointestinal Bleeding in Children: A Single Institution Study on Presentation, Aetiology and Management

Nasher, Omar ; Devadason, David; Stewart, Richard
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Background:
Acute upper gastrointestinal bleeding (UGIB) in children is uncommon and poses a clinical dilemma as therapeutic endoscopy is limited out of hours. In order to improve our service we reviewed our recent clinical practice with regard to the aetiology, presentation and management of these patients.

Methods:
A retrospective single-institution study on children (<16years) who presented with acute upper GI bleeding over a period of 5 years (May 2009 - May 2014) using known ICD codes.

Results:
A total of 32 children (17 males, 15 females) were identified during the study period with a median age of 6 years.

There were 32 patients with a total of 33 UGIB episodes. 19/32 presented with haematemesis, 9/32 with melaena, 3/32 with both melaena and haematemesis. One patient presented with fresh rectal bleeding.

On admission the mean haemoglobin level in patients who presented with haematemesis was 12.1g/dL (5.1-16.2) and only two required a blood transfusion. On the other hand, patients who presented with melaena +/-haematemesis had a mean haemoglobin of 9.2g/dL (5.1-13.1) and 4 required a blood transfusion within 24 hours of presentation (Table 1).

Based on clinical decision, 19 patients underwent upper GI endoscopy of whom 5 required therapeutic endoscopic intervention (variceal banding, ulcer clipping and Argon plasma coagulation). Following negative endoscopy, one patient required surgical intervention due to continuous decrease in haemoglobin and was found to have an intussusception and intestinal metastatic deposits. A further patient underwent laparotomy and was found to have a bleeding duodenal ulcer (Table 2).

The most frequent diagnoses at upper endoscopy were varices (5/18) and oesophagitis (4/18). On patients who did not undergo endoscopy the presumed aetiology included Mallory–Weiss tears (4), ingestion of foreign body (1), gastritis (3), and viral illness (1), known oesophageal varices (1) and unknown (3).

Table 1: Presenting symptoms of children with UGIB	Transfusion required	No transfusion required
Isolated haematemesis	2	17
Melaena +/- haematemesis	4	8

Table 2: Findings at upper GI endoscopy

	Number of patients	Therapeutic intervention
Varices	5	Banding (3/5)
Gastric ulcer	1	Clipping (1/1)
Gastric vascular malformation	1	Argon therapy (1/1)
Gastritis and duodenitis	3	None
Ingestion of foreign body	1	None
Oesophagitis	4	None
Duodenal ulcer	2	None
No abnormality detected	2	Laparotomy - small bowel resection (1/2) Laparotomy – duodenal ulcer undersewn (1/2)

Conclusion:
Whilst UGIB is uncommon in children, the morbidity associated with it is very significant. The presence of melaena in children may predict cardiovascular instability or the need for transfusion. Successful management of these patients requires both endoscopic and surgical expertise.

G42

Natural history of paediatric IBD around transition to adult services: a regional cohort study

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Background:
Effective transition of young people with paediatric-onset IBD (PIBD) is essential, but paucity of data exists in this area. We aimed to describe the transition of PIBD patients in South East Scotland (SES) with regard to natural history (disease activity, therapy escalation and service utilisation) both at the point of transfer and post-transition.

Methods:
A prospective PIBD database identified a cohort of all patients discharged from our regional service since 01/01/09. A retrospective study of patients leaving as a result of transition (graduation from paediatric to adult IBD services through a transition process, a transition event (single joint clinic) or transfer) until 30/09/13 was conducted, with data at a minimum of 1 year follow-up (FU) post-transition collected. Disease severity scoring was by Montreal Classification and Physician Global Assessment (PGA).

Results:
83 patients had transition; 59 Crohn's disease (CD), 13 ulcerative colitis (UC) and 10 IBD-unclassified (IBDU). Median age at transition was 18.0yrs (IQR 17.6, 18.4). 82% (68/83) of patients were in steroid-free remission (SFR) at time of transfer, and 5% (4/83) had moderate-severe disease. 73% (43/59) of CD patients had ileocolonic involvement (L3) and 56% (33/59) pan-enteric disease (L3+L4); 74% (17/23) of UC/IBDU patients had extensive disease (E3). 78% (65/83) of patients had been exposed to thiopurines, 47% (39/83) to methotrexate (MTX) and 25% (21/83) to anti-TNF? therapy in paediatric services; only 19% (16/83) patients had never had immunosuppression. 10% (8/83) had major IBD-related surgery prior to transfer and 3 patients (4%) had pan-treatment refractory IBD (primary non-response, complete loss of response or non-recoverable intolerance to all of thiopurines, MTX, infliximab and adalimumab). Median follow-up post transition was 2.7yrs (IQR 1.6-4.0). At last adult FU 6 patients had transferred out of SES and 3 had defaulted from clinic. 85% (63/74) of those remaining were in SFR; 8% (6/74) had moderate-severe disease. The rates of ileocolonic CD (L3) and pan-enteric CD (L3+L4) had already increased to 75% (40/53) and 60% (32/53) respectively; 67% (14/21) of UC/IBDU patients had extensive disease (E3). 13% (2/16) patients had their first thiopurine exposure, 3% (1/37) their first MTX exposure and 19% (10/54) their first anti-TNF? exposure in adult services. 12% (9/74) had major IBD-related surgery in adult services and the pan-treatment refractory IBD rate increased to 14% (10/73). One patient died of metastatic cholangiocarcinoma 3.5 years after transition.

Conclusion:
PIBD patients have significant disease at transfer to adult services with 25% having required anti-TNF? therapy and 81% at least one immunosuppressant. Progression of disease severity continues; 19% of patients required their first anti-TNF agent in adult services and 81% at least one immunosuppressant. Progression of disease severity continues; 19% of patients required their first anti-TNF agent in adult services and the rate of pan-treatment refractory IBD more than trebled to 14%.

H4

Animal models for paediatric (type 2) non-alcoholic steatohepatitis: an extensive systematic review

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Background:
Animal models are widely used in the study of non-alcoholic fatty liver disease (NAFLD). A range of models have been produced to reflect the spectrum of disease, including non-alcoholic steatohepatitis (NASH) and fibrosis. Most children with NASH have evidence of zone 1 (portal) inflammation, known as type 2 NASH, rather than the centrolobular inflammation of adult, type 1 NASH. We performed a comprehensive systematic review to identify whether any animal models reflect type 2 NASH.

Methods:
MEDLINE search for all articles (in English) including "animal model" and "non-alcoholic steatohepatitis" or "non-alcoholic fatty liver disease". Only primary research papers with animal models were included. Models were described by animal, genetic modifications, diet, and if toxic insults were used. Models were assessed for concordance with features of NAFLD: obesity, insulin resistance, steatosis, portal steatohepatitis, centrolobular steatohepatitis, and development of hepatocellular carcinoma (HCC).

Results:
MEDLINE search identified 951 articles, 472 were excluded (230 not relevant, 132 reviews or comments, 70 papers could not be obtained, and 39 had no animal model).

Data was extracted from 479 studies, which used 208 different animal models of NAFLD: 72 dietary, 35 genetic, 11 toxic, 4 offspring, and 86 combination models. The most frequently used models were: high fat diet (HFD) in mice (83/479), HFD in rats (64/479), and methionine-, choline-deficient (MCD) diet in mice (64/479).

No studies specifically described an animal model for paediatric NASH; only 1/208 models demonstrated a predominance of portal inflammation. Mice fed an ad libitum high-fat, high-fructose diet (?4800 kcal/kg diet) for 16-weeks (used by 4/479 studies) most closely reflects paediatric type 2 NASH, with portal fat infiltration, zone 1 steatohepatitis, and portal fibrosis.

129/208 models had evidence of Type 1 ("adult") NASH, with predominance of zone 3 inflammation or panacinar steatohepatitis. 19/208 models demonstrated development of hepatocellular carcinoma.

Conclusions:
A 16-week high-fat, high-fructose diet in mice most closely reflects paediatric type 2 NASH. There are a large number of published models, with variable phenotypes and histology.

H5

Ethnic differences in paediatric non-alcoholic fatty liver disease.

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Background:
Non-alcoholic fatty liver disease (NAFLD) is now the most common cause of liver disease in children, with an estimated prevalence of 5-10%. The majority of published data has involved Caucasians. Data is lacking regarding the phenotypic differences of NAFLD in children of Asian origin.

Methods:
We performed a retrospective review of children consecutively diagnosed with NAFLD at a tertiary referral hepatology service in the UK, between 2003 and 2014. Patients had been referred from either primary care physicians or general paediatricians with incidental asymptomatic abnormal liver function tests (LFTs) and/or fatty liver on ultrasound scan (USS). Patients with inadequate data or with secondary fatty liver disease were excluded. Patients were divided by ethnicity into Asian and Caucasian. All values are (±SD), unless stated.

Results:
100 patients with primary NAFLD were identified. 59/100 were Caucasian and 41/100 were of Asian origin, of whom, 73% (30/41) were Pakistani. There was male predominance in both groups, 73% (43/59) in Caucasian and 78% (32/41) in Asians. Mean ages at presentation were 12.6±2.5 and 11.7±2.8 years in Caucasian and Asian groups, respectively.

Asian patients had a significantly lower body mass index (BMI) centile than Caucasian (91.2±9.5 vs. 96.1±5.1, $p< 0.05$). There were no differences in LFTs between Asian and Caucasian patients, ALT (87.8±60.9 vs. 94.3±84.5 IU/L) and bilirubin (9.4±7.6 vs. 9±6.5 μmol/L). 61% (25/41) Asian patients had an elevated gamma glutamyl transferase, compared to 60% (35/59) Caucasian patients. Markers of the metabolic syndrome were similar in both groups: total cholesterol 4.4±1 vs. 4.4±1 mg/L, triglycerides 2.0±1.4 vs.2.2±1.4 mg/L, and HbA1c 38.6 vs. 39.1±7.4mmol/mol.

20 patients had undergone biopsy. 33% (2/6) demonstrated moderate-severe fibrosis in Asian patients, compared to 21% (3/14) Caucasian patients ($p=0.61$).

Conclusions:
Asian children with NAFLD have similar disease severity as their Caucasian counterparts, despite a significantly lower BMI centile at presentation. Referring clinicians and risk stratification scores require a greater awareness of ethnic differences in adiposity in paediatric NAFLD.

H9

Audit of long term histological outcomes after liver transplantation in children with continuing parenteral nutrition requirements

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Background:
Isolated liver transplantation (iLTx) for intestinal failure associated liver disease is rare (IFALD), and there are only a few reports in of children with intestinal failure who develop catastrophic deterioration in liver function before they can be weaned from parenteral nutrition (PN). Although the indications and outcomes of iLTX have been described [Dell Olio; Botha], there are concerns that short bowel syndrome might contribute to long term allograft dysfunction via malabsorption of bile acids / bacterial overgrowth and that post LTx exposure to PN combined with immune-suppression may lead to chronic inflammation. We compared histology of children who had liver transplantation (LTx) for extra-hepatic biliary atresia (EHBA) with a cohort of 6 patients all of whom survived more than 10yrs after iLTX.

Subjects and Methods:
A retrospective audit of all patients transplanted between 2000-2003 identified 6 children with IFALD who had iLTx (3 = gastroschisis; 3 =necrotising enterocolitis). Consecutive age matched patients who had LTx for EHBA were identified to act as controls (n=15). The following data were obtained: age of donor; type of graft; liver histology; liver function; renal function; duration of exposure to PN after transplant. The histology slides were anonymised and scored by two pathologists using the Ishak score for fibrosis.

Results:
Age at transplant and age of donar age were similar: iLTx 9.66 mons EHBA 8.96mons; donor age was 23.34 mons and 26.52 months respectively. One patient was recovering from chickenpox in the iLTx group at the time of the 10yr review. There were no differences in frequency of rejection episodes between iLTx and EHBA and similar low scores for fibrosis and inflammation was seen in both groups. Even though duration of PN exposure post iLTx was variable (up to 4.5yrs) and iLTX patients were significantly smaller - there was no correlation between duration of PN, length of short bowel and fibrosis scores.

	iLTx10yrs after LTx		EHBA 10 yrs after LTx	
Mean height and weight z scores [sd]	-1.89[1.22]	-1.41 [1.29]	0.43 [1.93]	0.25 [1.45]
Median Bilirubin mmol/L and range ()	11 (8-203)		6 (5-34)	
Creatinine clearance mls/min mean [sd]	119 [23]		121 [30]	
PN exposure in days median & (range	428 (100-1668)		0	
Ishak inflam score (0-18) mean & [sd]	0.92 [1.16]		1.23 [1.72]	
Ishak fibrosis score (0-6) mean & [sd]	2.08 [1.16]		1.17 [1.24]	

Conclusion:
Exposure to PN after LTx does not appear to cause additional fibrosis or inflammatory changes in liver allograft and short bowel syndrome is not a risk factor for hepatic fibrosis. Isolated liver transplant is an important option that should be considered in children who develop liver failure when weaning from PN.

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H14

De novo Inflammatory Bowel Disease following Paediatric Liver Transplantation:

A Case Series of Three Patients and World Literature Review.

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Background:
Inflammatory Bowel Disease (IBD) is a T-cell driven inflammatory process due to inappropriate and enduring activation of the enteric immune system, generally treated with immunosuppressant therapy. Following solid organ transplantation (SOT), recurrent and de novo IBD have been described despite immunosuppressive therapy. The majority of cases in adult patients occurred post liver transplantation (LT) (136/175) with 96/136 cases reported having been originally transplanted for sclerosing cholangitis (SC) or autoimmune hepatitis (AIH). In paediatrics, 14 cases have been described post liver, heart and renal transplant. 9 cases have been described post LT (3/9 cases transplanted for SC/AIH). Various risk factors have been implicated in the development of post-transplant IBD including CMV, acute and chronic rejection, post LT biliary stasis, personal and family history of autoimmunity and immunosuppressant therapy. Herein, we describe 3 cases of de novo IBD post LT for causes other than SC/AIH.

Methods:
Case 1 was the index case and the other 2 patients were identified through an electronic search of the Birmingham Liver Unit Paediatric Transplant database that holds the data of 782 patients who have undergone a liver transplant between 1983 and 2014. Patient case notes were then reviewed. Patients transplanted for SC and AIH were excluded. Medline and Embase were searched for "de novo inflammatory bowel disease" and "transplantation". The search was extending by scanning reference lists of related articles and free text web search. Non-original articles and articles referring to the same study population that was originally reported elsewhere were excluded. A total of 46 articles were included in the systematic review.

Results:
3 patients (2 females) were identified with de novo IBD following LT. 2 patients were originally transplanted for a1 antitrypsin deficiency and 1 for extra-hepatic biliary atresia. Risk factors for the development of post LT IBD are shown in table 1. Mean age at the time of de novo IBD diagnosis was 7.3 years (range 3-11 years) with a mean of 4.6 years post LT. Diarrhoea was the presenting symptom in 2 patients and intermittent rectal bleeding in 1. All patients were investigated for possible IBD according to Porto criteria. The patients underwent on average 2.6 upper and/or lower GI endoscopies prior to diagnosis, while the time of presentation to diagnosis varied from 3 months to 1 year. Crohn's Disease was diagnosed in 2 patients and Indeterminate Colitis in 1. Infliximab was used in 1 patient while the other 2 were treated with 5-aminosalicylic acids. All patients are in clinical remission.

Conclusion:
De novo IBD does occur following liver transplantation in children but is rare. De novo IBD should be considered in the differential diagnosis of chronic diarrhoea post-transplant.

Table 1. Risk Factors for De Novo IBD and Immunosuppressive Rx before and after De Novo

	Case 1: Crohn's disease	Case 2: Indeterminate Colitis	Case 3: Crohn's disease
CMV mismatch (donor / recipient)	-ve/-ve	+ve / -ve	-ve / -ve
CMV infection post LTx	No	No	No
Acute / Chronic Rejection	No / Yes	No / No	No / No
Biliary stasis	No	No	No
PMHx of Autoimmunity	No	No	No
FHx of Autoimmunity	No	No	No
Immunosuppression at presentation with de novo IBD	Tacrolimus, Prednisolone, MMF	Cyclosporine	Cyclosporine
Immunosuppression after diagnosis with de novo IBD	Tacrolimus, Prednisolone, AZA	MMF, Prednisolone	MMF, Prednisolone
Other IBD Tx following diagnosis	Infliximab	Mesalazine	Mesalazine

H15

Pegylated interferon + Lamivudine for treatment of Hepatitis B Virus associated nephrotic syndrome

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Objectives and Study:
Hepatitis B virus-associated glomerulonephritis includes membranous nephropathy (MN), membranoproliferative glomerulonephritis, polyarteritis nodosa, essential mixed cryoglobulinemia, IgA nephropathy, and focal segmental glomerulonephritis. MN is the most common renal manifestation of hepatitis B. These patients often require immunosuppressive medications (steroid, cytotoxic agents) to control the renal pathology. Clear guidelines for the treatment of hepatitis B virus associated nephropathy do not exist and as a result patients are treated variably.

We report a success treatment of child presenting with steroid resistant nephrotic syndrome with chronic HBV infection with Lamivudine and Pegylated Interferon.

Methods:
A retrospective case note review

Results:
A three year old child who was born in the UK had presented to her local hospital with oedema and significant proteinuria. A provisional diagnosis of nephrotic syndrome was made and she was commenced on Prednisolone 60mg/m2 /day but there was no improvement in her proteinuria after two months so a gradual weaning of Prednisolone started and Lisinopril was commenced. During investigations she was found to have chronic HBV infection.

The child presented to our centre three months after her initial presentation and was HBeAg+, HBsAg+, HBeAb-, HBsAb-, HBV DNA >3.4 E8. Her liver biopsy showed mild hepatitis with minimal fibrosis occasional ground-glass change.

She was started on Lamivudine 3mg/kg once day as an 8 week lead-in prior to the addition of Pegylated Interferon 100?g/m2 for a further 48 weeks. She was on tapering Prednisolone dose was on 10mg on alternate days at this time.

The details of her blood results are as follows:

	Presentation at local	Presentation at our centre	Week 8 treatment with Lamivudine and Peg started	Week 31 treatment	Week 56 end of treatment	6 months post end of treatment
HBsAg	positive	positive	positive	positive	positive	negative
HBsAb		negative	negative	negative	positive	positive
HBeAg		positive	positive	positive	positive	negative
HBeAb		negative	negative	negative	positive	positive
HBV DNA		>3.4 E8	2.15 E5	6.64 E3	<160 IU/ml	<4 E1
Proteinuria	positive	positive	negative	negative	negative	negative
Prednisolone	40mg od	30 mg od	off	off	off	off
Lisinopril	5mg od	5mg od	5mg od	5mg od	5mg od	5mg od

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THURSDAY 29TH JANUARY
Poster abstracts

Team 3

Poster Walk Round

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Concurrent Autoimmune Disease in Paediatric Inflammatory Bowel Disease

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Background:
Paediatric Inflammatory Bowel Disease (PIBD) is a chronic condition seen in genetically-predisposed individuals. Genome-wide association studies have implicated over 160 genomic loci in the disease with many genes coding for proteins in key immune pathways.

Objective:
This study aimed to look at the autoimmune disease burden in paediatric patients diagnosed with IBD and to interrogate the exome data of a subset of patients.

Methods:
Patients were recruited from the Southampton Genetics of PIBD cohort. Clinical diagnosis of autoimmune disease in these individuals was ascertained from medical records. Whole exome data was interrogated for a subset of patients with concurrent diagnosis of asthma to ascertain the burden of pathogenic variants within genes implicated in asthma and IBD. Association testing was conducted between disease status and a population control using the SKAT-O test.

Results:
Forty nine (28.3%) of the 173 children with IBD studied (32 (18.49%) CD, 15 (8.6%) UC and 2 (1.15%) IBDU patients) had a concurrent clinical diagnosis of at least one other autoimmune condition. Asthma was the most prevalent additional condition affecting 16.2% of the overall PIBD cohort. Rare variant association testing between exome data from 18 children diagnosed with both IBD and asthma revealed six significant genes ($p < 0.05$) prior to Bonferroni adjustment. Three of these genes have already been implicated in both asthma and IBD (ZPBP, IL1R1 and IL18R1). ZPBP is located on the chr17q12-q21 regions which have been associated with early-onset asthma, and variants in the same linkage disequilibrium block have been associated with Crohn's disease, type 1 diabetes and primary biliary cirrhosis. IL1R1 and IL18R1 are all involved in the cytokine-mediated signaling pathway2. The remaining three genes have been found involved in the pathogenesis of asthma only (PYHIN1, IL2RB and GSTP1). PYHIN1 encodes a protein that belongs to the HIN-200 family of interferon inducible proteins, important in controlling cell cycle, differentiation and apoptosis1. GSTP1 is involved in the detoxification of a wide variety of exogenous and endogenous compounds, including reactive oxygen species.

Conclusion:
Within our modest cohort, we observe high overall incidence of asthma that is even more pronounced in CD patients. Exome analysis to identify all coding variants, followed by joint analysis of common, rare and private mutations has power to identify associations not previously described. For some patients the underlying relationship between PIBD and autoimmune disease may lie in a systemic immune dysregulation rather than organ specific immune dysfunction.

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G18

Asssessment of the safety and efficacy of Laparoscopic-assisted Endoscopic Percutaneous Jejunostomy(LAPEJ): a novel technique and retrospective case series study

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Background:
Artificial enteric nutritional support is vital in the management of patients who are unable to maintain oral nutrition. Gastric feeding may not be optimal due to severe GERD, delayed gastric emptying or antro-pyloric dysmotility(1). In some circumstances, post-pyloric feeding can be used and can avoid parenteral nutrition (2).

For delivery of long term post-pyloric feeding a jejunal feeding via a direct jejunostomy provides a more stable and secure jejunal access compared with the nasojejunal or gastrostomy with jejunal extension(3).

It has been suggested that direct percutaneous jejunostomy insertion is technically more difficult and associated with a higher risk of complications, therefore; its usage has not, to date, been widespread (4).The aim of this audit was to review the novel approach of laparoscopic assisted direct percutaneous jejunostomy(LAPEJ).

Method:
Case records of paediatric patients who underwent LAPEJ between January 2008 and September 2014 were reviewed.

With a 2 port laparoscopic technique, the DJ flexure and jejunum were identified. Simultaneously an endoscope was passed to the jejunum. Safe and optimal positioning of the jejunostomy site, close to the abdominal wall, was followed by insertion of a percutaneous needle and then guidewire is passed in to the jejunum as per standard PEG placement. The guidewire was retrieved and a 12Fr Corflo PEG Tube was then pulled in to position.

Result:
14 patients were identified (median age 6.5 years, range 2-17), 11 had significant neurological impairment and 9 had already had a fundoplication. Current median follow up is 20 months (1-60).

All LAPEJ were sited successfully, feeds commenced within 6 hours with no evidence of of leak or peritonitis. Two patients developed subsequent jejunal volvulus requiring surgical intervention. Two developed gastrointestinal volvulus (3 months and 2 years post insertion).Both patients had abnormal gastrointestinal anatomy; one with a jejunal diverticulum which has been reported as a rare cause of midgut volvulus (5),and one with a complete small bowel malrotation which was evident only on laparotomy following LAPEJ. Barium meal is therefore a wise precaution before LAPEJ.

Conclusion:
LAPEJ placement seems to be a relatively safe and successful approach for children requiring jejunal enteral feeding.

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G19

Efficacy and safety of wireless capsule endoscopy in 305 paediatric patients: a tertiary centre experience

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Background and objective:
Wireless capsule endoscopy (WCE) is a useful diagnostic tool proposed to observe small-bowel lesions undetectable by conventional endoscopy.

Aim:
assessment of diagnostic yield and safety of WCE in a large cohort of paediatric patients and to compare with magnetic resonance enteroclysis (MRE).

Method:
In a retrospective review of consecutive capsule endoscopy studies, 305 studies were performed in 265 patients with a mean age of 11.2(range 2-18) years during an 8-year period. 47(15%) patients were younger than 6 year old. 136(46%) underwent WCE for suspected or confirmed Crohn's disease (CD);13 (4%) anaemia; 55 (18%)obscure gastrointestinal bleeding; 12% (37) polyposis; 13(4%) intestinal lymphangectasia; 30(10%) recurrent and chronic abdominal pain. Safety, efficacy and MRE correlation were analysed.

Results: Safety:
34(11%) patients had delayed passage of the capsule beyond the study period but none necessitated endoscopic removal because of symptoms.

Efficacy:
152(51%) had positive findings and 170 WCE findings (57%) led to a change in management of patients.

WCE resulted in reclassification of 15 IBD from indeterminate colitis to CD and another 30 patients to likely UC. 32(64%) of 50 patients with previously diagnosed CD had more extensive small bowel disease identified leading to treatment escalation.

In polyposis:
22(59%) WCE were positive for suspected PJP, and 12(32%) had a positive finding leading to endotheraputic resection/ablation. In 9(24%) WCE for Familial Adenomatous Polyposis (FAP)/Gardner, 2 had small polyps detected in small bowel but no intervention was necessary.

For obscure GI bleed, 10 patients had had identified bleeding site during conventional endoscopy and 23(42%) patients had positive findings from WCE. All positive cases were followed by DBE/ enteroscopy with endotherapeutic intervention.

24 suspected small bowel IBD involvement had both MRE and WCE: 21(88%) cases had similar outcome for both modalities where as 2(8%) cases showed positive WCE finding of extensive luminal Crohn's and negative MRE finding. One case (4%) had positive MRE finding of small bowel stricture and negative WCE finding. 3 cases showed hold up of the capsule in some areas and correlated with MRE finding of narrowing. Hence WCE 95% sensitivity and 100% specificity versus 89% sensitivity and 100% specificity for MRE.

Conclusion:
WCE is a safe and reliable investigation with 51% diagnostic yield in our patient cohort and changed management in 57%. Its use in recurrent or chronic abdominal pain is of no diagnostic value in our cohort.

G33

Clinical implications of measuring Infliximab profile in children with inflammatory bowel disease

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Background:
Infliximab has been proven effective in the treatment of inflammatory bowel disease (IBD) in children and can lead to a dramatic improvement in the life of these patients. The development of anti-Infliximab antibodies (ADAb) may indicate the cessation of treatment and poses challenges to clinicians. We present here our experience in children with IBD who developed ADAb against Infliximab.

Material and methods:
27 paediatric patients (13M), median age 11.5 years, were treated with Infliximab for ulcerative colitis (7 patients) and Crohn's disease (20 patients) at King's College Hospital in 2013-2014. Serum Infliximab and ADAb were measured using a commercial ELISA assay (Theradiag). The clinical response, disease activity and Infliximab levels / antibodies were reviewed.

Results:
Infliximab treatment was discontinued in 4/27 patients (14.8%) due to poor response despite therapeutic serum Infliximab levels (>2 ug/mL) and in the absence of ADAb (<10 ng/mL). 5 patients (1M) (18.5%) developed ADAb. Patients' age, diagnosis and information regarding Infliximab treatment are shown in table 1. 3/5 patients developed ADAb within a year after initiation of treatment and 5/5 in less than 2 years. 2/5 patients (patients 1 and 5) were noted to have measurable serum Infliximab levels in the presence of a high ADAb. These samples were subjected to further inhibition testing which confirmed positive ADAb with sub-therapeutic levels of Infliximab (<1 ug/mL).

Patients 1 and 3 were subsequently treated with Adalimumab. Patient 2 is due to undergo endoscopic reassessment. Patient 4 underwent endoscopy that revealed mildly active chronic pancolo-proctitis, was treated with steroids and is currently off biologics. Patient 5 is in clinical remission and still on Infliximab.

Conclusion:
The development of ADAb in children with IBD can result in sub-therapeutic drug levels and subsequent loss of response which may necessitate a change in treatment strategy. Measurement of Infliximab and ADAb in combination with clinical assessment is useful in guiding clinical decision-making.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age (years)	9	16	13	6	12
Diagnosis	Perianal Crohn's	UC/AILD	Crohn's	UC	Perianal Crohn's
Duration of the disease (years)	5	5	1	1.7	1
Treatment	Azathioprine	Mesalazine, Prednisolone, Azathioprine	Azathioprine	Mesalazine, Azathioprine, Sulphasalazine (enema)	Mesalazine
Duration \9months)/ number of infusions	18/ 11	4/ 5	8/ 6	8/ 6	13/10
Dose of infliximab (mg/kg)	10	5	5	5	5
PUCAI-PCDAI/FC	12.5/1480	5/1064	7.5/904	47.5/3000	15/207
Serum Infliximab (ug/mL)	measurable	<0.1	0.2	<0.1	measurable
Anti-infliximab antibody (ng/mL)	>200	>200	111	>200	115

Table 1.
UC: ulcerative colitis, AILD: autoimmune liver disease, PUCAI: pediatric ulcerative colitis activity index, PCDAI: pediatric Crohn's disease activity index, FC: faecal calprotectin

H13

Pancreatic disease in children; a 10-year single centre experience

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Background:
Pancreatic disease is becoming recently more prevalent in children. A wide spectrum of underlying pathology is observed with diagnosis and treatment proving challenging for pediatricians. We present here our recent experience and work up in children with pancreatitis.

Material and methods:
92 children with pancreatitis were referred to our centre in 2004-2014. Demographic, clinical and laboratory data were collected.

Results:
60 patients(26M), median age 10.15yrs were diagnosed with acute pancreatitis(AP). Three presented with necrotizing and 1 with hemorrhagic pancreatitis. Surgical intervention was required in 47. Hereditary(HP) and autoimmune(AIP) pancreatitis was confirmed genetically and histologically in 5 and 2, respectively. Two were diagnosed with drug-induced pancreatitis. Radiological findings included pancreatic pseudocyst(9), peri/pancreatic collection(5), pancreatic atrophy(4), necrosis(1) and laceration(6), pancreas divisum(3), CBD stricture/ dilatation(2/8), pancreatic duct(PD) stricture/ dilatation(2/6), accessory pancreatic duct(1), long common channel(3), choledochal malformation(7), pancreatic mass(1), stones(2), gallstones(3) and NAFLD/NASH(3/3).

32 patients(17M), median age 10.2yrs were diagnosed with chronic pancreatitis(CP). The work up and findings are shown in table 1. Seven and four children were diagnosed with HP and AIP, respectively. 34 surgical procedures were performed. Pancreatic insufficiency was confirmed only in 12%. Radiological findings included pseudocyst(3), peri/pancreatic collection(2), atrophy(5), necrosis(1), pancreas divisum(2), CBD stricture/ dilatation(13), PD stricture/dilatation(6), long common channel(1), pancreatic mass(2), stones(5), and NAFLD(2).

A pancreatoblastoma and a pancreatic solid pseudopapillary tumour were diagnosed. ERCP and endoscopic ultrasound(EUS) were utilized in 43% and 87% of AP and CP, respectively. Investigations and management are listed in Table 1.The etiology of AP and CP remained unknown in 12(20%) and 5(15%) children, respectively

Table 1. Diagnostic work up, findings

	Acute pancreatitis	Chronic Pancreatitis
Work up		
US/MRCP/MRI/CT ERCP/Endoscopic US/biopsy Secretin stimulation test SPINK1+/PRSS1+/CPA1+/CFTR	30/18/7/6 23/1/2 1 2/3/0	8/14/1/1 28/1/5 0 5/3/0/1
Management		
Stent insertion/Sphincterotomy Hepaticojejunostomy/plasty Excision of choledochal malformation Cholecystectomy Whipple procedure/Puestow Laparotomy and wash out/Collection drainage Antibiotics/NG-NJ feeds/Parenteral nutrition Pancreatic enzyme supplementation	17/1 5/1 7 9 1/1 1/4 7/1/4 0	18/3 3 0 2 0/5 0/3 2/1/1 4

Conclusion:
The incidence of pancreatitis in children remains unclear in the UK. The commonest causes of pancreatitis in our series were anatomical abnormalities, HP and AIP. Endoscopic investigations and pancreatic biopsy in children are safe procedures enhancing the diagnostic pathway.

N1

Vitamin D status of gastrostomy-fed children with special needs

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Vitamin D deficiency is a major public health problem in the United Kingdom and many other parts of the world. Children with special needs are at greater risk due to factors such as decreased mobility and outdoor play, concomitant medications that increase catabolism of vitmain D, reduced nutritional intake and low body weight. Gastrostomy-fed children receiving a nutritionally complete formula may still be at risk of vitamin D deficiency due to the above factors.

The objective of this study is to assess the vitamin D status of special needs children receiving full or partial nutrition via gastrostomy.

Methods:
Thirty-two children (24 male) aged 5-16 years, from 7 special schools in Manchester receiving gastrostomy feeds took part in the study. Blood samples were obtained in March 2014 (end of winter) to evaluate serum levels of 25hydroxyvitamin D (25OHD), calcium (Ca), phosphorus (P), alkaline phosphatase (ALP) and parathyroid hormone (PTH). In addition, carers were interviewed to obtain a dietary history and assess the subjects' exposure to sunlight.

Results:
All children had complex medical conditions. 26 were non-ambulatory and 18 were taking anti-epileptic drugs. Nineteen children (59%) had a Fitzpatrick skin type score of III or higher (South Asian or Black African ethnicity). The children had little or no sunshine exposure in the 3 months prior to data collection.

Thirty results were obtained for serum 25OHD. The mean serum 25OHD was 71.1nmol/L (+/- 21.4nmol/L). One child was found to be vitamin D deficient (serum 25OHD 24.6nmol/L) and 4 children were vitamin D insufficient (serum 25OHD range 29.9-47.9nmol/L). 2/25 children (8%) had an elevated PTH (serum PTH 75ng/L and 110ng/L). All children had normal serum P, Ca and ALP.

Thirteen children (43%) received less than 10µg of vitamin D per day from their feed (range 3.5-9.2µg/day). None of the children were taking vitamin supplements.

Dietary calcium intake met the recommended intake for age and gender in all but one subject.

Conclusion:
From our results we conclude that nutritionally complete gastrostomy feeds may be protective against vitamin D deficiency in gastrostomy-fed children with special needs.

Conflict of Interest:
This study was funded by Danone UK

BSPGHAN 2015 Annual Meeting

FRIDAY 30TH JANUARY
Poster abstracts

Team 1

Poster Walk Round

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Attitudes Towards A Clinical Trial Of Growth Promoting Therapy In UK Children With Crohn's Disease And Their Parents

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Background:
Despite major improvements in the management of Crohn's disease (CD), growth retardation is still commonly encountered and may be followed by a reduction in final adult height (Ht) in approximately 20% of patients treated with contemporary treatment regimens. Considering that the abnormality may occur at multiple levels of the GH/IGF-1 axis, the possible use of other forms of growth-promoting agents such as rhIGF-1, either alone or in combination with rhGH for promoting growth, requires further investigation.

Objectives:
To survey the attitude of young people with CD and their parents toward growth hormone and insulin like growth fator-1 (IGF-1) treatment as part of a proposed randomised clinical trial where children would have standard therapy for CD, daily injection of rhGH, daily injection of rhIGF-1 or daily injections of rhGH and rhIGF-1. Methods: A feasibility survey on the attitudes and opinions of young people with CD and their parents was conducted to establish whether they would be willing to take part in a future trial of growth promoting therapies Eligibility for completion of the survey was any young person (and their parent/guardian) attending inflammatory bowel disease out patients clinics (at two tertiary paediatric hospitals) whose HtSDS was ? 1. A total of 48 completed questionnaires by young people and 46 completed parents' questionnaires were returned.

Results:
The overall response rate was 80 % (48 out of 60 questionnaires were completed by young people and 46 out of 60 questionnaires were completed by parents). The median age of young people was 14.3 years (range 7.0 to 17.7) and 35 (73 %) respondents were males. Median HtSDS at time of approach was -1.2 (-3.01, 0.23) and median mid-parental HtSDS was -0.6 (-3.14, 1.4) [p=0.003 HtSDS vs mid-parental HtSDS]. 4 (9 %) young people had been specifically treated for a growth problem before. Of 48 young people and 46 parents: 26 (54%) young people and 29 (63%) parents were not concerned about height, 19 (40%) young people and 11(24%) parents were slightly concerned about height and 3(6%) young people and 5 (11%) parents were very concerned about height. The majority of respondents, 42 (88%) young people and 40 (87%) parents, agreed that doctors should try to find a better treatment for growth in CD; 20 young people (42%) and 25 parents (54%) believed that opportunity of gaining extra height was worth a year of daily injections. Overall, 21 (44%) young people and 22 (48%) parents were willing for blinded randomised controlled trial (RCT) participation.

Conclusion:
Around 40% of children and parents surveyed would take part in an RCT of growth promoting therapy even though only a minority are very concerned about their height. Allaying fears about injections to participants would be likely to achieve higher recruitment rates.

G17

Single centre experience on outcome of anti TNF therapy in young people with Crohn's disease who have transitioned care to adult services

Belsha,Dalia[1]; Barraclough, Harriet[1], Brooks, Alenka[2]: Fieldsend,Bev: Robinson, Kerry: Forbes, Valda[1]: Donnelly, Mark: Lobo, Alan: Campbell, David[1]: Urs, Arun[1]: Thomson, Mike[1]: Rao, Prithviraj[1]: Narula, Priya[1]:
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Anti TNF (Tumor Necrosis Factor) agents have increasingly been used in the treatment of moderate to severe Crohn's disease. However, there is no clear guidance on the optimum duration of therapy and little is published on paediatric experience following withdrawal of these agents.

Aim:
To review the usage of anti TNF agents and medium term outcomes in the transitional population with Crohn's disease and Indeterminate colitis (IC).

Method:
Young people (YP) with Crohn's and IC who were on maintenance anti-TNF agents and had undergone transition and transfer of care to the local adult team in the last 6 years were identified from a database maintained prospectively in our centre. Data was collected by case note review on disease distribution at diagnosis, concomitant medications, need for surgical interventions, indication for starting anti TNF therapy, switching or stopping anti TNF agents during and after transfer of care to the local adult services.

Results:
30 YP with Crohn's and IC transferred care to the local adult services in the time period studied had received anti TNF therapy, but complete data was available for only 21. The median age at diagnosis of IBD in this cohort was 11.6 years (range 6.7–16.2 yrs), median age at most recent follow up was 19.75 years (range 18-22 yrs) and median duration of follow up in the local adult service was 2.75 years (range, 0.75-6 yrs).

All had active disease despite standard medical management or were intolerant necessitating step up treatment (20 on Azathioprine, 1 on Methotrexate) and 3 had had surgical treatment prior to starting anti TNF therapy. Infliximab was the first line anti TNF started in all YP.

Three patients had 10 mg/Kg as treatment dose of Infliximab at some point in their treatment course and 8 had 6 weekly infusions and one had 4 weekly infusions before switching to Adalimumab.

11(52%) had endoscopic reassessment after transfer to the adult service. 4 (19%) YP remained on Infliximab infusions during follow up, 5 (24%) required surgical resection following which Infliximab was discontinued. Of these one restarted anti TNF (Adalimumab) after 8 months and another after 4.5 years.

6 (28%) lost response to infliximab (median duration 3.25 yrs) and changed to Adalimumab which they remained on during their last follow up with 2 requiring weekly injections. 2(9%) had an allergic reaction and changed to Adalimumab. 1(5%) patient switched electively during transfer to Adalimumab and then discontinued it due to side effects but was restarted back on Infliximab due to relapse. 4(19%) stopped Infliximab following reassessment and only one re-started anti TNF (Adalimumab) 3 years later.

Apart from one patient who developed Tuberculosis after being on Infliximab for 4 years and 2 who had an allergic reaction whilst on Infliximab, there were no other serious side effects.

Summary and Conclusion:
Anti TNF treatment was well tolerated in our cohort of patients. At the time of last follow up in the adult services, only 19% were on the same anti TNF, 15% successfully discontinued anti TNF therapy and the remaining required surgical resection (24%), switched or restarted anti TNF therapy. In total, 15 (71%) of our patients remained on anti TNF therapy. This data may provide guidance in counselling YP and their families prior to starting anti TNF therapy.

G20

Banding of Bleeding Jejunal Varices in an 8 year old child.

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Introduction:
Ectopic varices are defined as large porto-systemic venous collaterals occurring anywhere in the abdomen except in the cardio-oesophageal region, and present with observable portal hypertension, haematochezia with/or without haematemesis, and often with a history of abdominal surgery

Case Report:
An 8 year old male patient presented with obscure but profound acute GI bleeding (AGIB).

A complex background of gastroschisis associated with duodenal and colonic atresia necessitating multiple surgical interventions and adhesions was noted.

Multiple episodes of GI bleeding over 6 years had occurred possibly secondary to a superior mesenteric vein thrombosis. These were intermittent in nature and managed conservatively, with a period of two years without an AGIB prior to this presentation, which consisted of 17 episodes of AGIB. Seven episodes involved significant haematochezia and/or large melaena, with lowest recorded haemoglobin of 22 g/l - multiple blood transfusions and octreotide were required. CT angiography suggeste possible mesenteric varices in the upper abdomen with no active bleeding source.

Upper GI endoscopy and ileo-colonoscopy revealed portal gastropathy only and wireless capsule endoscopy revealed varices in the proximal jejunum. Trans-oral double balloon enteroscopy (DBE) revealed 4 moderately large isolated jejunal varices around 40-50 cm post-pylorus. Four bands were placed successfully on the afferent and efferent ends of 2 of the larger varices via an operating gastroscope. Two weeks later endoscopy revealed the 2 banded varices to be thrombosed and sloughing within the bands was noted which were beginning to fall off the mucosa. The remaining 2 variceal vessels were then also banded. No AGIB recurred for 4 weeks following the initial banding however a third endoscopy occurred at this point and some bleeding was noted from the presumed afferent arm of one of the varices and this was banded once more. No further bleeding has occurred to date and no perforation ensued from any of these banding procedures.

Conclusion:
Endoscopic jejunal variceal banding was used successfully to achieve initial haemostasis in this child and this represents the first report of this technique in the paediatric population.

G24

Incidence of both Crohn's disease and ulcerative colitis has continued to rise over the last 10 years within the Southwest region of England.

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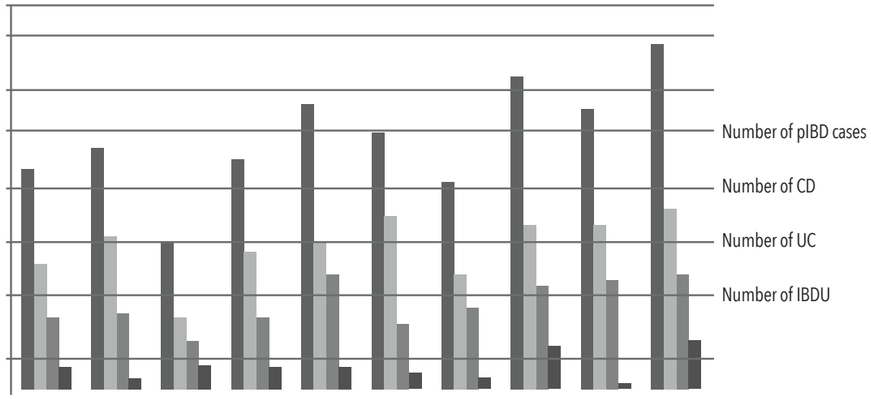
Background:
The first prospective national survey of paediatric Inflammatory Bowel Disease (pIBD) was conducted in the British Isles in 1997 and documented an incidence of 5.2/100,000 children per year1. A higher incidence was noted in the north (Scotland: 6.5/100,000) as compared to the south (England:5.2/100,000) and Ireland (4.4/100,000). The aim of this prospective study was to document any change in incidence of pIBD in Southwest of England (SWE).

Methodology:
Bristol is the single specialist paediatric gastroenterology centre for SWE to which all children suspected of having IBD from the 12 paediatric centres are referred for endoscopy. Prospective data was collected on all new pIBD cases between 2004–2013 including types of IBD, gender and postcode address.

Results:
461 new cases of pIBD were diagnosed with 40 cases in 2004 compared to 63 in 2013. The incidence of pIBD in SWE increased from 4.5 (2004) to 6.7/100,000 (2013). Male (n=269) to female (n=192) ratio was 1.4:1. Crohn's disease increased from 2.61 to 3.54, Ulcerative Colitis 1.48 to 2.25 and IBD-unclassified 0.45 to 0.96. The 5-year cumulative incidence rates for the city of Bristol were much higher than the whole SWE and increased from 9.2 (2004-2008) to 11.0 (2009-2013).

Annual incidence of pIBD and its subtypes for Southwest (2004 – 2013).



Conclusions:
Annual incidence of both Crohn's disease and ulcerative colitis in SWE has continued to increase during 2004–2013 (4.5 to 6.7) with male preponderance. This study documents significantly higher incidence in the city population (9.2 to 11.0) supporting role of environmental factors.

Refs. (1) Sawczenko A. Sandhu BK et. al. Prospective survey of childhood inflammatory bowel disease in the British Isles. Lancet. 2001 357(9262):1093-4.

G35

Patterns of colonic transit and mucosal abnormalities in children with refractory constipation

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[1]Medical University of Warsaw; [2]Great Ormond Street Hospital London

Background:
Functional constipation is a common problem in paediatric clinical practice with a worldwide prevalence of up to 29.6%. Refractory constipation, defined as failure to respond to optimum conventional therapy for a minimum of three months represents a diagnostic and therapeutic challenge. In children with refractory constipation, investigating underlying colonic disorders is required to guide medical and/or surgical therapy. Several methods were described to measure colonic transit time of which radiopaque markers are simple to use and easy to interpret.

Aims:
The aim of this study was to measure colonic transit time (CTT) using radiopaque markers and to assess the incidence of colonic mucosal abnormalities in children with refractory constipation.

Methods:
Retrospective review of clinical, pathological and radiological data of children with refractory chronic constipation who underwent colonoscopy and a radiopaque marker study in our institution between January 2010 and December 2013.

Radiopaque marker studies were performed within two weeks after colonoscopy by ingesting one capsule (Colon Transit 3*2 Radiopaque Markers (St. Marks Configuration); Pentland Medical®) per day for three successive days. Each capsule contains 10 distinctive makers. A plain abdominal radiograph was taken on day 4 on the same time of ingestion. CTT was calculated using the following formula: number of markers*2.4.

Results:
134 radiopaque studies were identified. 8 were excluded, 4 were unable to complete the studies and 4 did not fulfil the criteria of refractory constipation. Of the remaining 126, Halve were from females and halve were males. The mean age of patients (±SD) was 7.9 years (±3.6); range 0.8-17.1 years.

82 (65%) had slow transit constipation and 44 (35%) had colonic segmental delay of which 10 (8% of total) were side right, 18 (14%) left side and 16 (13%) were rectosigmoid hold up.

The mean total CTT (±SD) for all children was 52.6 hours (±17), right colon transit 14.8 (±12.4), left colon transit 19.2 (±14.9) and rectosigmoid transit was 18.7 (±13.2). We then calculated CTT for each group. Children with slow transit constipation had CTT of 57.7 (±14.8), while children with segmental dysmotility had CTT as follow: right side CTT was 21.7 (±8.9), left side CTT was 40.1 (±20.95) and children with functional outlet obstruction had rectosigmoid CTT of 30.6 (±12.38).

109 children had colonoscopy within 2 weeks before taking the radiopaque makers. In 64 children (59%) the histology was normal and in 45 (41%) mucosal abnormalities were identified.

37 (82%) had increased inflammatory cells density in the lamina propria with eosinophils and lymphocytes are the dominant inflammatory cells. 3 (7%) had prominent lymphonodular hyperplasia, 3 (7%) had melanosis coli and 2 (4%) had focal cryptitis. Children with slow transit constipation had significant histological abnormalities (pvalue 0.03) however; the histological abnormalities were not significant in children with segmental delay.

Conclusions:
More than halve of children with refractory constipation had slow transit constipation and only 13% had functional out let obstruction. Increased numbers of eosinophils and lymphocytes in mucosal biopsy was a significant finding in children with slow transit refractory constipation.

G36

Changes in patterns of colonic transit in children with functional constipation.

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[1]Medical University of Warsaw; [2]Great Ormond Street Hospital London

Background:
Functional constipation is mainly a clinical diagnosis as described by the Rome III criteria including stool frequency, fecal incontinence, painful defecation, retentive posturing, stool size and difficulty in passing a bowel movement.

In children with chronic constipation, investigating underlying colonic disorders is helpful to guide medical and/or surgical therapy. Several methods were described to measure colonic transit time (CTT) of which radiopaque markers are simple to use and easy to interpret.

Aims:
The aim of this study was to evaluate changes in patterns of constipation and CTT in children with functional constipation.

Methods:
We retrospectively reviewed clinical and radiological data of children with functional constipation who underwent more than one colonic transit measurement using radiopaque marker studies in our institution between January 2010 and December 2013.

Radiopaque marker studies were performed by ingesting one capsule (Colon Transit 3*2 Radiopaque Markers (St. Marks Configuration); Pentland Medical®) per day for three successive days. Each capsule contains 10 distinctive markers. A plain abdominal radiograph was taken on day 4 on the same time of ingestion. CTT was calculated using the following formula: number of markers*2.4.

Results:
19 patients were included in this study. The average mean age of patients (±SD) was 7.8 years (±3.0); range 2.8-13.4. M/F ratio was 15/4. Two radiopaque marker studies were performed for each patient. The average mean time between both studies (± SEM) was 1.8 years (±0.75); range 0.3-0.6.

The baseline studies showed: 12 (64%) patients had slow transit constipation (STC) and 6 (37%) had colonic segmental delay of which 1 (5%) were right side (R-SD), 5 (26%) left side (L-SD) and 1 (5%) was rectosigmoid hold up (RS-SD). The mean total CTT hour (±SD) was 58.0 (±13.9), right colon transit (RCTT) 21.1 (±18.1), left colon transit (LCTT) 16.7 (±13.5) and rectosigmoid transit was (RSTT) 20.2 (±13.3).

In the subsequent studies, 1 (5%) study was normal, 10 (53%) had STC and 8 (47%) had colonic segmental delay: 2 (11%) R-SD, 2 (11%) L-SD and 4 (20%) were functional outlet obstruction. The mean total CTT was 56.0 (±19.7), RCTT 20.3 (±20.7), LCTT 15.8 (±15.9) and RSTT 19.8 (±16.2).

There were no changes in pattern of constipation in 11 cases (8 patients had STC, 2 had L-SD and 1 had RS-SD).

The colonic transit patterns changed in 8 cases: in 1 patient from STC to normal; in 2 cases from STC to RS-SD; in 1 case from R-SD to functional outlet obstruction; in 1 from STC to R-SD; in 1 from L-SD to R-SD; in 2 cases from L-SD to STC.

However, none of the changes in total and segmental colonic transit time were significant ($p>0.05$).

Conclusions:
Measuring total and segmental colonic transit time with radiopaque markers allows the identification of types of colonic motility disorder and the appropriate choice of treatment according to the location of the delay.

In this study, 47% of children had a different type of constipation in repeat radiopaque marker study. Total and segmental transit time was not statistically different between the first and second transit measurement likely due to small sample size.

In children with chronic functional constipation who failed to response to optimum therapy, repeating colonic radiopaque marker study may provide a useful guide to further therapy.

H8

EBV associated post-transplant smooth muscle tumour in a child following isolated intestinal transplantation.

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Introduction:
Post transplant smooth muscle tumours (PTSMT) are rare neoplasms associated with Epstein Barr Virus (EBV). They have been reported previously in children; but not to our knowledge in the native colon following isolated intestinal transplantation.

We report a case of a PTSMT in the sigmoid colon of a child following isolated intestinal transplantation.

Case description:
The child was born at term following antenatal diagnosis of gastroschisis. Following resection of necrotic bowel after birth he was left with 31cm of dysmotile small bowel. He underwent isolated intestinal transplantation at 5 years due to life-threatening catheter-related blood stream infection. Immunosuppression was Basiliximab at induction, steroid taper, and then tacrolimus and prednisolone maintenance with sirolimus added at 1 month, as per the protocol of the transplant centre. He was EBV seronegative at transplant. He became positive within the first week and remained viraemic but there was no evidence of post transplant lymphoproliferative disorder (PTLD). The transplant was complicated with ongoing mild ulceration of the graft ileum and insufficient absorptive function. The ulceration was treated with infliximab with partial response.

A routine GI endoscopy at 30 months post transplant demonstrated improvement in the overall ulceration though a prominent ulcer was seen in the ileum and minor ulcers in the rectum. A polyp was noted in the sigmoid colon and was resected endoscopically. Histological analysis confirmed a lesion similar to a leiomyosarcoma[i], composed of spindle cells demonstrating a diffuse and strong nuclear staining for EBV. Following these findings, immunosuppression was decreased.

An MR enterogram showed enlarged mesenteric lymph nodes and a diagnosis of PTLD was considered. EBV DNA PCR in the blood remained high. Full thickness bowel and mesenteric lymph node biopsy were performed but did not support this diagnosis. Tacrolimus was discontinued and sirolimus level was increased and has remained as maintenance immunosuppression.

Further endoscopies have shown multiple small ulcers in the graft and a small ulcer in the colon, but no polyps; now 42 months post transplant and 12 months post diagnosis of PTSMT.

Discussion:
EBV-PTSMT is rare and there have been no previous cases described in intestinal transplant recipients without diffuse PTLD. The long term prognosis remains unclear and is difficult to quantify due to the rarity of the disease, however it appears more favourable in cases of isolated PTSMT. A case review of 14 paediatric patients with EBV-PTSMT resulted in a mortality rate of 57%, but of those that died 50% also had PTLD[ii]. A meta-analysis of 63 cases of PTSMT in adults and children found that most cases of PTSMT occurred in renal transplant patients and the commonest site was the liver. Lower survival rates were found in those with multi organ involvement (48.5%) and particularly patients with intracranial involvement[iii].

Many management options have been attempted but the most promising are complete resection of the lesion or administration of sirolimus[iv]. In contrast to the management of PTLD, reduction of immunosuppression does not appear to be effective.

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N4

Intestinal failure in neonates - Causes and outcome.

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In adult and paediatric patients, there are clear definitions around provision of PN and the diagnosis of intestinal failure. There is no definition in neonatal patients and it is unclear at what stage these infants could or should be classed as having intestinal failure.

The aim of this retrospective cohort study was to identify and describe the number of neonates who require PN for greater than 28 days in a level 3 neonatal unit and describe their outcomes for growth, mortality, and time taken to achieve enteral autonomy.

Methods:
Neonates receiving PN for greater than 28 days between January 2009 to December 2013 were included. The cases were identified from Badgernet (version 2.8.0.0). Accuracy of ascertainment was assessed using pharmacy data. Data on each eligible case was collected recording their demographics, gestation age, diagnosis, duration of use of PN and anthropometry. Statistical analysis variables were completed using Statistical Package for Social Sciences 19.0 (SPSS: An IBM Company, Chicago, IL). Differences between weight and variables of interest were analysed using Wilcox-Mann-Whitney Test to determine statistical significance.

Results:
A total of 128 cases were identified where neonates had received PN for greater than 28 days. The mean gestation age was 26 weeks with 119 cases being preterm. There were 69 males and 59 females. The indication for use of PN was congenital or acquired gut disorder in 65 cases with 63 cases needing PN because of gut immaturity associated with prematurity. Prematurity in absence of GI disease required PN for a median duration of 35 days and they were able to achieve enteral autonomy by the corrected gestation age of 29.6 weeks. No case in absence of GI disease required PN for 90 days or more. Infants with GI disease required a significantly longer duration of PN with the median being 45 days (p 0.02) .

109 cases were discharged home, 3 transferred out to another hospital and 16 died. 8 cases were on PN at the time of discharge from neonatal unit. Of these there were 3 males and 5 females. 4 cases had congenital gastrointestinal disorder with 4 requiring bowel resection because of complications associated with prematurity.

Conclusion:
Our results suggest that the use of PN for up to 90 days in extremely pre term infants is very prevalent. Failure to achieve enteral autonomy in neonates by 90 days is seen in presence of GI disease only. Nearly half of all infants in a neonatal unit require prolonged PN(>28 days) because gut immaturity associated with prematurity. All preterm infants would achieve enteral autonomy in absence of gastrointestinal disease with the median age being 30 weeks.

This data would suggest that intestinal failure in neonates would be better described as use of parenteral nutrition for greater than 90 days.

BSPGHAN 2015 Annual Meeting

FRIDAY 30TH JANUARY
Poster abstracts

Team 2

Poster Walk Round

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G3

Telephone consultation as a substitute for routine out-patient face-to-face consultation for children with inflammatory blowe disease: randomised control trial and economic evaluation

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Objectives and Study:
Crohn's disease and ulcerative colitis, collectively known as inflammatory bowel disease (IBD), are chronic bowel disorders characterised by recurrent relapses alternating with periods of remission. Traditionally, children with IBD are asked to attend regular face-to-face hospital appointments for their routine out-patient follow up. This means that, even when they are well, they have to travel to the hospital and this may involve travelling long distances. We tested the hypothesis that telephone consultation is an effective, safe, and a cost-effective alternative to face-to-face consultation for children and adolescents with IBD.

Methods:
Patients with IBD (aged 8-16 years) were randomly assigned via a computer-generated randomisation schedule to receive telephone consultation or face-to-face consultation for 24 months. The primary outcome measure was the paediatric IBD-specific IMPACT quality of life (QOL) scores. Secondary outcome measures included patient satisfaction with consultations, disease course, anthropometric measures, proportion of consultations attended, and duration of consultations. Analysis was by intention to treat.

Results:
86 patients were randomised to receive either telephone consultation (n=44) or face-to-face consultation (n=42). Baseline characteristics of the two groups were well balanced. 86% and 83% of patients completed follow-up in the telephone and face-to-face consultation groups respectively. After 12 months, there was no statistically significant difference in the primary outcome, QOL scores (estimated treatment effect 5.7, 95% confidence interval -2.9 to 14.3; P=0.188). There was a statistically significantly lower mean consultation time for telephone consultation compared to face-to-face consultation (estimated reduction 4.3 minutes (95% confidence interval 2.8 to 5.7; P<0.001). No statistically sigificant differences were seen in the other secondary outcomes. NHS costs per patient consultation was lower in the telephone group [Telephone £35.41 vs Face-to-Face £51.12, difference £15.71 (95% confidence interval 11.80 - 19.60; P=<0.001)]. Given that the confidence interval excluded a clinically significant detriment and the telephone consultation costs less, telephone consultation could be considered as a more cost effective alternative to face-to-face consultation.

Conclusion:
This study demonstrates that telephone consultation does not materially lower quality of life. It reduced consultation time and NHS costs without evidence of negative impact on other outcomes. Telephone consultation may be a cost-effective alternative to face-to-face consultation for the routine out-patient follow up of children and adolescents with IBD

G12

The use of a low FODMAP diet in paediatric functional gastrointestinal disorders

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Objectives and Study:
Irritable bowel syndrome (IBS) and functional abdominal pain (FAP) are common paediatric conditions. Symptom management includes pharmacotherapy, psychological therapy, lifestyle and dietary changes. There is sufficient evidence for the use of a low FODMAP diet (LFD) for symptom management in adults. Consequently, paediatric centres are considering using a LFD in children with IBS and FAP, despite limited evidence in children. Whilst symptom efficacy in children is important, nutritional adequacy and maintenance of growth needs to be ascertained on such a complex diet, prior to conducting large paediatric trials. The aim of this study was to evaluate the nutritional safety and feasibility of a LFD in children with IBS/FAP.

Methods:
A before-and-after quasi-experimental pilot study was conducted at a tertiary NHS trust, and national ethical approval was obtained. Five patients aged 10-16 years with IBS or FAP who commenced a LFD were included. Nutritional intake (using 4-day estimated food records), anthropometrical measurements (weight, height and mid upper arm circumference) and symptom scores (using a Likert-scale questionnaire) were measured at baseline and after 6 weeks of following a LFD. Nutritional adequacy was also compared to national dietary reference values. Practical implementation of a LFD was assessed at 6 weeks.

Results:
Data from 4 completed participants (2 male) are presented. One participant discontinued the study in view of no improvement. Overall symptom scores decreased on the LFD (121 vs 93), with no improvement in one participant. Average intake decreased on the LFD for all macronutrients and most micronutrients, with magnesium, iron, copper and zinc intakes insufficient. Average z-scores decreased for weight-for-age (0.013 vs -0.208) and BMI-for-age (-0.12 vs -0.45). Difficulties in following the diet included the lack of variety, cooking family meals, eating out, appropriate snacks, and cost.

Conclusion:
This preliminary data indicates symptoms improved in 75% of paediatric patients following a low FODMAP diet; however it may compromise dietary intakes and growth. Despite some difficulties in implementation, all families were motivated to continue the diet. Additional dietetic support may be required to improve nutritional adequacy. Further research will be required to replicate this data and to inform on the effect of a LFD on long-term nutritional adequacy, growth, efficacy compared to standard treatment, and the social impact on the child and family.

G22

Pilot study of impedance measurements and ph- monitoring in children and infants with gastroenterology, respiratory or neurology problems symptoms

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Background:
Multichannel intraluminal impedance combined with pH monitoring (MII-pH) has become the standard test for diagnosing gastro oesophageal reflux disease (GORD) in children. It is regarded as superior to isolated pH monitoring as it provides an opportunity to study bolus flow within the oesophageal body for both acid and non-acid reflux. It also provides a temporal association between symptoms and reflux.

The cut off between physiological and pathological reflux in children is not clearly defined and the current normal values are extrapolated from adult data or are indirectly produced by studying symptomatic children. In this study we aim to assess pH and impedance values in infants and children with gastrointestinal, respiratory and neurological disorders.

Methods:
Retrospective review of results of children underwent MII-pH testing (Sandhill Scientific®) at Great Ormond Street Hospital between November 2010 to October 2014. Age appropriate transnasal catheter were used and position check by X- ray to align the pH sensor at two vertebral bodies above the diaphragm.

All studies were analysed using auto scan followed by manual analysis of the whole study. Standardised reports were generated for all studies.

Infants (< 1yr) and Children (>1yr) were divided into three groups according to their background symptoms: gastrointestinal, respiratory and neurological disorders.

Results:
Data from 69 children and infants was used. 21 children and infants had abnormal impedance measurements. 20 children and infant had abnormal Reflux index. 37 children and infants presented with gastroenterology symptoms, 25 children and infants with respiratory symptoms and 8 children and infants with neurological problems.

The group with gastroenterology problems had 23 normal and 14 abnormal impedance values compared with 30 normal and 7 abnormal reflux index ph measurements.

The group with respiratory problems showed 21 normal and 4 abnormal impedance measurements compared wit 16 normal and 9 abnormal reflux index ph values.

The group with neurological background had 5 normal and 3 abnormal impedance values compared with 4 normal and 4 abnormal ph values.

To note is that depending if impedance measurement or reflux index was used several children and infants had normal values and with the other measurement abnormal values.

Conclusions:
This pilot review of impedance and ph measurements provides a range of values for normal and abnormal measurements in the paediatric population and adds to the overview of these measurements in the different subgroups of children presenting with either gastroenterology, respiratory and neurology symptoms. Further research and analysis of data is needed to analyse and identify clear patterns and trends of these measurements in the different symptom groups.

G26

A Nationwide Survey of Post-Operative Management of Crohn's disease in children in UK

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Background and aims:
Despite advances in immune-modulators and biologic therapy, the risk of surgical resection for paediatric onset Crohn's disease was 48±5% by the age of 30 years (ECCO-ESPGHAN) and disease recurrence is 80-100% within 3 years without post-operative treatment(ECCO). There is currently no proposed consensus guidelines for the management of post-operative Crohn's disease in paediatrics. Our aim was to review the current practice throughout UK for the post-operative management of paediatric Crohn's disease.

Methods:
An online survey of all tertiary paediatric gastroenterology centres across UK was undertaken by advertising on the BSPGHAN website and also by contacting units individually.

Results:
In this survey our response rate from gastroenterology centres was 18/23 (78.2%). Pre-operative steroid weaning policy: 53% continued low dose steroids, 33% had withdrawn completely and 13% continued either low dose steroids or off steroids. Risk Factors for post-operative recurrence: 33% used ECCO guidelines to define risk factors, 16% followed departmental protocol, 11% used both and 38% had no specific policy. Post-operative prophylaxis: 14% did not use prophylaxis until relapse or endoscopy, 57% used immediate prophylaxis with immune-modulators if there are risk factors, 21% used anti-TNF for high risk patients and 7% used antibiotics. Duration of post-operative prophylaxis: 23% used regular ileocolonoscopy to decide about the duration of prophylaxis, 23% used both ileocolonoscopy and CDAI score, 30% used clinical symptoms and biological markers and 7% used combination of all. Reassessment of the disease: 66% used endoscopy post-operatively only at time of relapse and 13% reassessed the disease with endoscopy at 6 months. Treatment: 61% started immune-modulators from no prophylaxis if there was endoscopic recurrence and 76% continued the same in the maintenance phase. Follow up: 43% followed their post-operative patients in IBD clinic, 25% in combined gastro-surgical clinic and 31% in both clinics.

Conclusions:
This survey demonstrates that there is significant variability in the treatment modalities of paediatric post-operative Crohn's disease in UK. It highlights the need for developing consensus guidelines/ algorithm based on risk stratification to reduce variability in practice and to deliver the optimal post-operative treatment in children.

G37

Lactobacillus paracasei bacteraemia following probiotic use in an infant with short gut syndrome – a report

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Sheffield Children's Hospital,Sheffield

Background:
Probiotics are live non-pathogenic microbial preparations that colonise the intestine and are increasingly used in patients requiring nutritional support. The commonly used strains are Lactobacillus, Bifidobacterium and Saccharomyces boulardii. Infections associated with lactobacillus are extremely rare. We report an infant with short gut syndrome developing Lactobacillus paracasei bacteraemia following use of probiotics.

Case Report:
A baby girl born at 27 weeks developed signs of enterocolitis at 10 days of life requiring resection of 16 cm of necrotic bowel including terminal ileum secondary to volvulus with ileo-colonic anastomosis. At10 weeks of age, she was unwell with peripheral blood culture growing enterococcus faecalis. A lower GI contrast suggested narrowing of small bowel in the right iliac fossa requiring adhesiolysis. At 15 weeks of age, she was treated for suspected sepsis with negative cultures. She was admitted being unwell again with possible gastroenteritis, severe dehydration, and metabolic acidosis at 20 weeks of age. The cultures for blood, urine, including stool for virology and bacteriology were negative. At discharge she was initiated on probiotics and parents have used various formulations but predominantly "Yakult" (Yakult UK, London, UK) for prevention of antibiotic-associated diarrhoea/ enteral-associated diarrhoea. She presented at 46 weeks of age with progressive abdominal distension, faltering growth, loose stools and vomiting; nutrition was optimised and bacterial decontamination with antibiotics was initiated. 2 weeks later she was acutely admitted with signs of obstruction with bilious vomiting. A laparotomy showed massively dilated small bowel loops (~5-6cm) with probable functional obstruction at the level of anus ending up with loop colostomy. Her feeds were stopped and parenteral nutrition was initiated. Initial central line cultures grew enterococcus and Klebsiella, which were sensitive to Teicoplanin. She received 2 weeks of antibiotics but on stopping spiked temperature to 390c with increased white cell count and CRP. The Echocardiogram did not indicate vegetations but showed a small thrombus at the end of the line. The central and peripheral cultures grew Lactobacillus paracasei sensitive to clindamycin and linezolid. Probiotics including breast-feeding was stopped, central line lock changed to Taurolock, and Rifaximin used for bacterial decontamination. After 2 weeks of treatment with Tazocin, no additional bacteraemia was noted. She is slowly establishing enteral feeds and trained for home parenteral nutrition.

Discussion:
The taxonomy of genus lactobacillus has been in a state of flux with Lactobacillus casei Shirota regarded as belonging to species Lactobacillus paracasei. Although no finger-printing of isolate was performed, we suspect the source of Lactobacillus might be exogenously administered probiotic. We also believe the fragility of intestine may have contributed to translocation of supplemental Lactobacillus paracasei. This case illustrates specific safety issue when potential infective agents are given to immunologically immature patients receiving enteral/parenteral nutrition, central venous catheter and disorders associated with increased bacterial translocation.

G38

Status Dystonicus: Is it a gut feeling?

Velayudhan,Manjula; Mordekar,Santosh; Narula,Priya; Rao,Prithvi; Urs,Arun; Thompson,Mike; Campbell,David
Sheffield Children's Hospital

Aim of the study:
Status dystonicus (SD) is a life-threatening movement disorder emergency. We report nine severely disabled children with SD and gut motility problems managed by the paediatric neurology and gastroenterology teams at the Sheffield Children's Hospital U.K.

Methods:
Retrospective case notes review of nine children admitted with SD at a tertiary centre in the UK looking at underlying neurological diagnoses, age of onset of dystonia, concomitant antidystonia medication, intrathecal baclofen pump with rate of daily infusion, anti reflux medication, gastroenterological investigations, use of enteral feeding (oral, gastrostomy and jejunal feeds) and parental feeding (PN).

Results:
Nine children, mean age 8.43 years (range 5 months to 15.2 years) presented with SD. Pain, pallor, sweating and Sandifer's syndrome were precipitated by enteral feeding. All children had multiple, independently observed time points where stopping feeds resulted in resolution of symptoms and recommencement of enteral feeds even at 5mls per hour lead to the return of dystonic spasms and pain. pH impedance studies and gastroscopy in all children showed no evidence of reflux disease and esophageal biopsies were not suggestive of peptic esophagitis. Six of the nine children had Nissen's fundoplication as a trial to relieve spasms and on repeat endoscopy, fundoplication wrap was intact. All children had jejunal feeding tubes (jejunal extensions to the PEG) with confirmed tip position in the first loop of the jejunum or close to the Duodenojejunal flexure by fluoroscopic placement. Barium studies and plain films ruled out intestinal obstruction or subacute obstruction. Five patients with severe feed induced pain and dystonias were treated with PN (3 long term and 2 short term).There was objective evidence of substantial pain resolution in these 5 children.

Conclusion:
Children with SD can be at risk of enteral feed induced abdominal pain and small bowel (foregut) dysmotility with some needing PN. The severity of the neurological condition seems to correlate with the severity of gastrointestinal problems raising the need for an ethical frame work and greater understanding of the neurogastroenterological issues.

H12

Infectious Complications In Biliary Atresia; A Single Centre Experience

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Background:
To evaluate incidence, timing and bacterial aetiology of cholangitis and spontaneous bacterial peritonitis (SBP) in Biliary Atresia (BA), after Kasai Porto-enterostomy (KP), prior to liver transplantation (LT).

Methods:
A single-centre retrospective analysis, comprising 78 patients who underwent KP between 2008-2010

Results:
78 patients (36M:42F) who underwent KP (BA;Type III 90%; syndromic 10%) comprised our study group. Cholangitis followed in 38/78 (48%) patients; median number of episodes 2 (1, 5). Median age for first episode was 5.6 months (2, 72.5). Six patients showed dilated biliary radicles on ultrasound. Organisms were isolated in blood cultures in 6 patients; Staphylococcus Aureus, Klebsiella, Streptococcus Pneumonia, E-coli (n=2) and Pseudomonas. 27/38 (71%) patients with cholangitis underwent LT, 10 are alive with their native liver and one died.

Ascites developed in 29/78 (37%) patients, at median age 6.5 months (3.1, 66). Ascitic taps were performed in 41% (12/29), due to respiratory distress with fever (5/12) or without fever (7/12) at a median age 7.4 months (3.2, 22.8). Timing of tap was at the onset of ascites in 6 patients and at a median time of 2 months (0.1, 4.6) from onset of ascites in the remaining patients. Four patients fulfilled criteria for SBP diagnosis; 3 culture-negative (wcc > 250mm3), one bacteri- ascites (wcc < 250mm3; mixed gram-positive cocci and gram-negative rods). No culture-positive SBP was identified. One culture-negative SBP was positive for Streptococcus Pneumoniae in blood cultures. Five patients that underwent ascitic taps previously had cholangitis. Antibiotics were already commenced in 8/12 patients pre-tap. Raised plasma wcc (>17 mm3) was identified in SBP (3/4) and non-SBP (3/8) patients. All SBP patients underwent LT at a median age 10.5 months (7.1, 16.1). Non-SBP patients underwent LT (n=4), died (n=3) and are alive with native liver (n=1).

Conclusions:
Cholangitis and SBP occurred in 48% and 5% of BA patients respectively, with cholangitis episodes presenting earlier. Few cases revealed positive bacterial cultures, in particular in ascitic fluid, which may be partially attributed to antibiotic use pre-tap. The definition of SBP in children needs to be considered cautiously to account for antibiotic use and new molecular techniques should be sought to aid diagnosis. LT is a successful outcome in cholangitis, SBP and non-SBP ascites.

BSPGHAN 2015 Annual Meeting

FRIDAY 30TH JANUARY
Poster abstracts

Team 3

Poster Walk Round

G6
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G6

Importance of D1 biopsies in the diagnosis of coeliac disease in children with tTG levels between the upper limit and 10 times the upper limit of normal

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Background:
Coeliac disease is an immune-mediated enteropathy triggered by exposure to gluten in genetically susceptible individuals. An antibody level to tissue transglutaminase enzyme (IgA tTG antibody) >10 times the upper limit of normal together with positive anti-endomysial antibodies in symptomatic, HLA DQ2/8 positive patients is now diagnostic of coeliac disease (2013 joint BSPGHAN and Coeliac UK Guidelines). This combination of findings negates the need for intestinal biopsies in children suspected of having coeliac disease.

The patchiness of coeliac disease has been well documented. More recently, a growing number of reports have shown that the histological changes of coeliac disease are more severe in the duodenal bulb (D1) when compared to the distal duodenum (D2 and beyond). Changes restricted to the duodenal bulb have also been reported, highlighting the need for both duodenal bulb and distal duodenal biopsies in the diagnosis of coeliac disease. However, the relationship between location of histological changes in the duodenum and the levels of IgA tTG antibodies has not previously been studied.

Methods:
A retrospective audit was carried out on 96 children diagnosed with coeliac disease at a tertiary paediatric gastroenterology centre between January 2011 – when a two-site biopsy policy (D1 and D2) was introduced for suspected coeliac disease – and June 2014. 85 were investigated because of symptoms. Biopsies were reported by a single histopathologist and graded using the Marsh criteria. IgA tTG antibody levels separated the symptomatic children into two groups: a "low" group with raised levels of IgA tTG antibodies <10 times the upper limit of normal as per the laboratory assay; and a "high" group with levels of IgA tTG antibodies of >10 times the upper limit of normal.

Results:
66 of the 85 symptomatic children had both D1 and D2 biopsies. Children in the "low" tTG group were more likely to have greater histological changes in D1 than D2 (changes restricted to D1 in 35.7% of children in the "low" group compared to 7.7% in the "high" group), whereas children in the "high" tTG group were more likely to have similar histological changes in D1 and D2 (76.9% in the "high" group compared to 21.4% in the "low" group)

Conclusions:
Children undergoing biopsy for the diagnosis of coeliac disease based on a tTG between the upper limit and 10 times the upper limit of normal, require biopsy of D1 in addition to D2. Our findings suggest that IgA tTG levels increase with more extensive duodenal involvement.

G9

Percutaneous Endoscopic Gastrostomy (PEG) at The Shrewsbury & Telford Hospitals NHS Trust - A Bench Marking Exercise

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Background:
The Royal Shrewsbury Hospital, Shrewsbury & The Princess Royal Hospital, Telford are 15 miles apart but form a single Trust. The Paediatric Gastroenterology Unit which is based at the Royal Shrewsbury Hospital provides a service for both hospitals. This includes the placement of PEG's and its subsequent management, with support by the Community Paediatric Nurses. The planned hospital reconfiguration, with a move to a single paediatric Inpatient site at the Princess Royal Hospital necessitated an evaluation of various services. Therefore, a benchmarking exercise of the PEG Service was undertaken and audited against the European Society for Parenteral and Enteral Nutrition (ESPEN) Guidelines.

Methods:
All children with a PEG in-situ were identified from the Community Nurses PEG database. The elctronic records of these patients were accessed for relevant data and input into a Microsoft Excel 2010 database by a single researcher. The ESPEN standards were used to audit the service in the relevant domains.

Results:
53 children with a PEG were identified. There was a clearly documented indication for a PEG in 96%, with neurologically disabled children at risk of aspiration comprising the largest group (47%). Feeding difficulties and Failure to thrive (FTT) made up 30% while other indications were Sensory Feeding Disorder in combination with other primary disorders (13%) dysmotility (4%) and metabolic causes (2%). The primary diagnosis was neurological in 55%, gastrointestinal 11%, renal 4% and cardiovascular, respiratory and endocrine 2% each. There were multiple diagnoses in 11%

Nasogastric feeds were instituted and documented prior to the PEG in 94% and may have been used in a further 2% but not documented. More then half the children had their PEG sited at the age of 13-36 months (52%), a further 15% under 13 months while 20% were after the age of 36 months. Almost one third of the children (29%) had no complications from the PEG. Localised infection was the commonest complication (28%) with granulation tissue (13%), mechanical problems (dislodgement 8%, blockage 4%), skin ulceration (10%) and leakage (8%) as other complications.

PEG feeds were successful in improving the weight centiles of these patients. Parental satisfaction with the service could not be evaluated retrospectively.

Conclusions:
The PEG Service at the Shrewsbury & Telford Hospitals NHS Trust adheres to ESPEN standards in the majority of patients and is associated with a low complication rate. Parental Satisfaction with the service should be sought prospectively.

G13

Do we adhere to Guidelines : A retrospective review of diagnostic pathway for local children presenting with suspected Coeliac Disease in whom the diagnosis was confirmed 2013-4

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Background:
In 2012, the European Society of Paediatric Gastroenterology Hepatology and Nutrition (ESPGHAN) published guidelines for the diagnosis and management of coeliac disease. For symptomatic patients with a tissue transglutaminase (tTG) greater than ten times the upper limit of normal who were endomysial antibody (EMA) positive and either HLA DQ2 or DQ8 positive, diagnosis could be confirmed without the need for a duodenal biopsy.

Methods:
All children from the Southampton district diagnosed with coeliac disease between January 2013 and March 2014 were identified from the departmental database. Data is prospectively recorded for all children with coeliac disease who are followed in our centre. For each patient, histology, tTG, EMA and HLA results; presenting symptoms and date of endoscopy (if performed) was recorded on an EXCEL spread sheet.

Results:
53 patients were included, of whom 26 were males and 27 females. Of these 30/53 (56%) patients had a tTG greater than 10 times the upper limit of normal (ULN). All were EMA and HLA DQ2/DQ8 positive. 16/30 were symptomatic and according to the ESPGHAN guidance, the diagnosis of coeliac disease could have been confirmed without biopsy. Out of these 16, 11 cases underwent an endoscopy for histological confirmation and in only 5; a diagnosis was made without duodenal biopsy.

In summary:
- All cases with a tTG greater than 10 times ULN had coeliac disease

- Out of 16 cases where histological confirmation was not required as per the new guidelines, only 5 were diagnosed without histology
- 11 out of 53 patients had an endoscopy and biopsy when it wasn't required

Conclusions:
According to the ESPGHAN guidelines, 30% of cases reviewed could have had a diagnosis of coeliac disease confirmed without undergoing endoscopy. However, in our centre only 5/16 had the diagnosis made without histological confirmation.

From discussion with colleagues this non adherence to the published guidance is widespread. This needs to be explored further by discussion of anxieties, concerns, audit of outcomes and increase in awareness to improve confidence in their use, as is intended, if practice is going to change for the benefit of patients and their families.

G21

The NICE Guidelines on Diarrhoea & Vomiting - How good are we?

Dlamini, Noni, Ayub, Naeem,
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Background:
The NICE Guidelines on the Management of Diarrhoea and Vomiting were published in April 2009. The key priorities for implementation were Diagnosis, Assessing Dehydration & Shock, Fluid Management, Nutritional Management and Advice for Parents and Carers. However, there is little information on how well this has been implemented both at Local and National levels. We therefore sought to audit our Paediatric Unit against the NICE Implementation standards to benchmark our service.

Methods:
Fifty case-notes were identified by the codes for "Gastroenteritis" and "Diarrhoea and/or Vomiting". Relevant data was extracted and input into Microsoft Excel 2010 by a single researcher.

Results:
In the first domain of "Diagnosis", stools were cultured in 13 cases. However, a clear and justifiable indication for this was only documented in 3 cases. The assessment of dehydration and shock was variable, depending upon the signs documented. This ranged from 9 (for Blood Pressure) to 46 (Pulse and Respiratory rates) out of 50 cases. Oral hydration was succesful in 43 cases although it was attempted in 49 of the 50 cases. Nasogastric rehydration was utilised in one case. Eight children required intravenous fluids. Clear indications for this intervention were documented as "Clinically unwell" in 4 cases and "Vomiting" in the other four. Nasogastric rehydration was only attempted in one of the 4 cases with vomiting.

Nutritional management following rehydration was appropriate in at least 29 cases with 25 children commenced on normal solids while 4 children received full-strength milk.

Documentation of information (verbal or written) given to parents or carers was identified in just 7 cases.

Conclusions:
Documentation overall was poor. The Implementation Standards were poorly followed in terms of Diagnosis and Clinical Assessment. However, oral rehydration was appropriately attempted, and successful in the majority.

Nasogastric rehydration was poorly utilised but intravenous fluids were generally only given when truly indicated.

Appropriate recommendations have been made as a result of these findings.

G27

Is respiratory involvement in microvillus inclusion disease a part of multisystem disease?

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Background:
Microvillus inclusion disease (MVID) is a congenital disorder with lack of microvilli and intracellular vacuole formation leading to irreversible intestinal failure. It presents with persistent life threatening watery diarrhoea. However, in recent years, improvement in parenteral nutrition (PN) and availability of Intestinal transplantation (ITx) has offered hope for long term survival.

MVID is recognised as a multisystem disease causing liver dysfunction and renal fanconi syndrome. Improved survival in these patients has offered an opportunity to study other likely systemic manifestations in MVID.

Patients who undergo small bowel (with or without liver) transplantation need significant immunosuppression and are at risk of severe graft versus host disease. They are also at greater risk of infections with respiratory pathogens due to immune-compromised state post-transplant. This is likely to predispose them to significant respiratory morbidity. At present there is no evidence in the literature that patients with MVID develop significant respiratory morbidity.

We share our experience of MVID cases referred for ITx in a regional paediatric ITx centre over 20 years (1993-2012) with emerging respiratory association probably evident due to improved survival.

Objective:
To review the respiratory complications in patients with microvillous inclusion disease with or without small bowel transplantation

Method:
It was a retrospective audit at a supra-regional referral centre for small bowel transplantation with vast experience of looking after patients with MVID who have needed intestinal transplantation. Retrospective review of case notes and radiological investigations in patients referred with MVID over 20 years (1993-2012) was done

Case notes were reviewed to look for demographic details, management of MVID and evidence of chronic respiratory signs and symptoms in these patients. The findings on radiological investigations if any were studied and confirmed with the paediatric radiologist within the trust.

Results:
Twenty cases referred (11 females and 9 males). Nineteen studied in detail (insufficient data in one). Ten underwent transplantation (3 liver and ITx, 7 isolated ITx). Median age at transplantation was 38 months (8-55). 6/10 died post-transplantation at median age of 46.5 months (12-81). 8/9 died pre-transplantation at median age of 26.5 months (3-83). One (late onset) weaned off PN at 11 years.

It is interesting that seven patients (3 transplant survivors, one late onset MVID, 2 post transplant and one pre-transplant deaths) developed chronic respiratory symptoms (cough and haemoptysis) with chest radiographs showing atelectasis and bronchial wall thickening. Three had chest Computer Tomography done which confirmed bronchiectatic changes.

G30

Patient experience with endoscopy at a single regional endoscopy unit

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[1]Sheffield Children's Hospital NHS Foundation Trust, Sheffield; [2]University of Sheffield, Sheffield

Background:
A Global Rating Scale (GRS) is used in adult UK gastroenterology endoscopy services as a quality improvement and assessment tool1. Within this quality of patient experience is measured and feedback is embedded. A paediatric endoscopy GRS in development will include evaluations of patient experience, an essential part of service evaluation. We piloted a survey to assess patient experience.

Methods:
A patient/parent survey was developed with our PALS and clinical governance teams. It was distributed to all patients on elective and emergency lists prior to the procedure and collected the same day. The study periods ran from 10 December 2013 to 31 January 2014 and 25 June till 1 August 2014. Data was analysed with Excel.

Results:
229 (109 in winter) patients underwent endoscopy during the study periods. There was a 33% (n=36) response rate during the winter period and 49% (n=59) in summer. Sex distribution of the respondents mirrored those listed. There were significantly less (ANOVAs $p<0.003$) responses from under 2s and more (ANOVAs $p<0.003$) from 13-15yrs age groups. Main procedures performed included Upper GI endoscopy, ileocolonoscopy, pH, bravo pH and impedance studies and PEG procedures.

100% of respondents felt they had opportunities to ask questions prior to the procedure. 3% of respondents stated the procedure was not explained prior to consent. 2% felt they did not understand the procedure. Information leaflets were given to 80% of respondents. 25% of respondents in winter and 17% in summer stated they were not informed of waiting times. 73% of respondents in winter and 49% in summer felt waiting times were about right. 24% in winter and 46% in summer felt it was too long. 1 in winter and 3 in summer felt it was too short! Most felt prepared based on information given (94% acceptable or above).

46% of respondents stated complications, chiefly pain (35% winter and 40% summer).

		Winter (%)	Summer (%)	Domain: Care for		Winter (%)	Summer (%)
Preparedness	Good/excellent	89	81	Dignity	Good/excellent	94	96
	Acceptable	6	12		Acceptable	7	2
	Poor/terrible	6	7		Poor/terrible	0	2
Comfort	Good/excellent	97	92	Additional needs	Good/excellent	87	50
	Acceptable	0	4		Acceptable	0	40
	Poor/terrible	3	4		Domain	14	10
Dr's sensitivity & courtesy	Good/excellent	97	94	Privacy	Good/excellent	90	92
	Acceptable	3	2		Acceptable	10	6
	Poor/terrible	0	4		Poor/terrible	0	2
Dr's sensitivity explaining findings	Good/excellent	92	87	Overall feedback	Good/excellent	97	94
	Acceptable	4	5		Acceptable	0	2
	Poor/terrible	4	8		Poor/terrible	3	4

Conclusion:
Overall patients had a good experience of endoscopy in our unit. Areas identified to improve include analgesia and procedure and waiting time explanations.

References: 1. Global rating Scale Accessed 5/11/14

H5

Prolonged Neonatal Jaundice - Is Urine Culture a Useful Test?

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Background:
Jaundice is one of the most common conditions requiring medical attention in the newborn baby. 80% of preterm babies and 60% of term babies develop jaundice in the first week of live and 10% of breast-fed babies are still jaundiced at one month.

Prolonged jaundice is defined as the persistence of jaundice beyond 14 days in a Term infant and 21 days in a Pre-term infant. Standard protocols for investigating prolonged neonatal jaundice include blood tests (Split Bilirubin, Liver Function Tests, Thyroid Function Tests, Galactosaemia screening) and Urine Culture. The latter is difficult to obtain and its value remains uncertain.

This study aims to determine the value of Urine Culture in infants with Prolonged Neonatal Jaundice at the Royal Shrewsbury Hospital, UK

Method:
All babies investigated for Prolonged Neonatal Jaundice at the Paediatric Assesment Unit of The Royal Shrewsbury Hospital from January - April 2014 were retrospectively identified from the Unit Registar and relevant data input by a single researcher into an Excel database.

Results:
56 babies were identified during this period. There was an equal distribution between males and females with an age range from 14 - 32 days. 13 babies (23%) comprising of 6 males and 7 females had a positive urine culture. Twelve of the 13 samples were clean catch specimens and one a midstream specimen. 6 of these 13 positive urine cultures were thought to be significant based on microscopy findings and pathogens isolated. A second catheter urine specimen was obtained in these babies of which 3 grew the same isolate (2 E. Coli and 1 Staph Aureus) and were succesfully treated with the appropriate antibiotic. Renal ultrasound scans were normal in all three babies.

Conclusions:
Although 23% of the babies investigated for prolonged neonatal jaundice had positive urine cultures, false-positives were common with an ultimate yield of only 3.5%. Nevertheless, the significance of a UTI cannot be ignored in a newborn baby and urine culture should continue to remain part of the investigation protocol for Prolonged Neonatal Jaundice.

N3

A Multi-Dimensional Intensive Tube Weaning Strategy: Cost Effective and Preferable Outcomes Over Surgical Solutions in Some Cases

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Background:
It is often medically necessary for children who fail to consume required nutrients and calories orally to be tube fed in either the short- or long-term. This can provide the professional and their family with the certainty that the child may maintain their growth trajectory. It also may be a stop-gap to provide necessary medical intervention or to quickly rectify an idiopathic diagnosis of growth faltering. Tube feeding itself is not a preferable long-term solution for some children and is not without its negative consequences for the child's development of the eating behaviour. Depending on the intellectual capacity, the child's age at the time when the tube was inserted and the medical experience associated with the placement of the tube, the child may become psychologically dependent on the tube; requiring nutritional support beyond the duration of their ailment. Tube feeding is also a costly enterprise, is socially debilitating for the child's family and disables the child from reaching age-appropriate milestones.

Despite the certainty that the feeding tube provides, other options beyond surgical interventions are raising in the horizon: Intensive home /community or hospital-based psychological treatments are available. Such interventions are far more cost effective, have no known future limitations for the child and end with socially and developmentally appropriate outcomes.

Methods:
The current study presents three cases where the child was scheduled for surgery within weeks to have a percutaneous endoscopy gastrostomy (PEG) placed. All three children (ages ranging from 14 months to 39 months) were offered an intensive multidimensional psychological evidence-based treatment (MPs-MDA) developed and implemented by psychologists specialists in avoidant and restrictive food intake disorder (ARFID). Each child presented with a variety of medical conditions, sensory impairments and global developmental delay. The children's weight, total caloric intake and food type (Carbohydrates, Fruit and Vegetables, Meat/Protein source, Dairy and Confectionary) was recorded. Each intensive intervention lasted for a week with a follow-up period of at least three months. All children started from a baseline of zero calories consumed orally during baseline.

Results:
Despite poor expected outcomes and the belief that only a long-term tube dependency was the appropriate management, results showed that the MPs-MDA facilitated for these children to acquire developmentally appropriate eating behaviour that allowed them to make a transition to full orally consumed diets and within a very short period of time. In addition, all negative behaviours and barriers measured (screaming, crying, tantrum, refusing to come to the table, spitting out food, refusal to try foods, etc...) diminished following the treatment. All children ate orally, met their caloric needs gained weight and most importantly PEG were avoided. These outcomes continued during the follow-up period. Costs of the cases show significant and large savings of the intensive intervention against projected surgical solutions and ongoing nutritional support.

In conclusion:
The MPs-MDA treatments for children at risk of having NG or PEG placed should be considered in some cases as it is shown to be cost-effective, provides preferable outcomes for the child's development and offers better quality of life for the child and family.

BSPGHAN Annual Meeting

PICO Presentations selection for oral presentation

Wednesday 28th January 2015

To compare outcomes in children with UC using Methotrexate or standard treatment in cases with steroid dependence or intolerance to or failure to respond to standard treatment.

PICO session

Dr Dharam Basude

BSPGHAN Annual meeting 2015

Stratford-on-Avon

Clinical and Cost Effectiveness of Methotrexate treatment in children with steroid dependent UC?

- Steroid dependent Ulcerative colitis is challenging when occurs
  - despite optimum 5ASA and Thiopurine use
  - When there is Intolerance to Thiopurine and or 5-ASA
- Therapeutic options are limited leading to
  - Prolonged steroid use
  - Infliximab – no NICE approval
  - surgery – high morbidity.
- Methotrexate treatment
  - Is easily available and cost-effective
  - Effective in a number of autoimmune and inflammatory conditions including Crohn's disease
  - Has not been adequately investigated in UC

The PICO

- P= **Child (5-16 yrs age) with steriod dependent UC**
- I= **Oral Methotrexate once weekly with Folic acid and antiemitics**
- C= **current standard treatment**
- O= **Rate of maintenance of remission, steroid usage, escalation to Infliximab, Calcinurin inhibitors and surgery.**
  Analyzed at 3, 6 , 12 months.

Outline research methods

- Multicentre RCT comparing standard treatment to Methotrexate
  - Inclusion: 5-16yrs of age with confirmed UC, steroid dependent ≥ 3 months on standard treatment or intolerance/failure to respond to standard treatment.
- Power of 0.8, sample 32-40 in each arm
- 2-3 years (depending on number of centers involved)

References

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Advances in diagnosis of paediatric gastro-oesophageal reflux disease

PICO session
BSPGHAN Annual Meeting 2015
Stratford-on-Avon

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Co-authors: Philip Woodland, Clinical Lecturer; Ian Sanderson, Prof Paediatric Gastroenterology; Daniel Sifrim, Prof GI Physiology.

What is the Clinical and Cost Effectiveness of Salivary Pepsin?

Background / rationale:

•Gastro-oesophageal reflux (GOR) is a physiological phenomenon.

•Daily regurgitation is reported in up to 2/3 of infants <6 months of age, and is described as prob-lematic in up to 1/4.

•The specificity of "GORD symptoms" and assessment of the causal relationship between reflux events and symptoms is challenging.

•Lack of a definitive test for GORD in infants has led to increasing empirical pre-scribing of PPIs for patients with symptoms suspicious of GORD.

•Salivary pepsin measurement is a novel non-invasive diagnostic tool in adult GORD.

The PICO

- **P = Patient group** (*Who will be the subject of the research?*)
  - ✓ Children 0-18 yrs of age.
- **I = Intervention** (*What will be researched?*)
  - ✓ Salivary pepsin measurement
- **C = Comparator** (*Against which the intervention will be compared*)
  - ✓ Established, invasive (endoscopy and biopsy, 24 pH-impedance measurements) methods of diagnosis of gastro-oesophageal reflux
- **O = Outcome(s)** (*Why the research will be important for patients?*)
  - ✓ Normal values of salivary pepsin in healthy children
  - ✓ Is salivary pepsin as sensitive and specific as the established methods of GORD diagnosis?

Outline research methods

- **Study 1: Salivary pepsin in a healthy paediatric population**

✓Establish normal values for salivary pepsin in a paediatric population.

✓We will recruit individuals with no gastrointestinal symptoms.

✓We aim to recruit 30 individuals in each of 3 age groups (0-18 months, 18 months to 4 years, 5 years to 16 years).

Outline research methods

- **Study 2: Comparison of diagnostic findings from endoscopy, 24 hour pH-impedance monitoring and salivary pepsin measurements in children**
- ✓ Evaluate the role of salivary pepsin in the diagnosis of Paediatric GORD.
- ✓ We will recruit children seen in the paediatric gastroenterology outpatient clinic and suspected (by the clinician) of having gastro-oesophageal reflux disease.
- ✓ We aim to recruit 45 children (15 children in each age group; 0-18months, 18months-5 years, 5-16 years) with clinically suspected GORD.
- ✓ We will only recruit children who are undergoing an upper GI endoscopy and 24-hour pH-impedance study for clinical reasons.

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Acknowledgements

- Dr. Philip Woodland, Clinical Lecturer, QMUL
- Prof. Daniel Sifrim, Prof. in GI Physiology, QMUL
- Prof. Sanderson, Prof. of Paed. Gastroenterology, QMUL

To establish the best method for weaning infants with short bowel syndrome from parenteral nutrition

PICO session
BSPGHAN Annual meeting 2015
Stratford-on-Avon

Sarah Macdonald, Susan Hill
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What is the Clinical and Cost Effectiveness?

Background / rationale

- There are no randomised trials about the best method of enteral feeding infants with intestinal failure post-surgical resection of a portion of the intestine
- It is agreed that enteral nutrition (oral/enteral liquid feed given via an artificial feeding device) is introduced at the earliest opportunity.
- There is no consensus about the most appropriate type and method of feeding

The PICO

- P = **Patient group**
- **infants with short bowel syndrome**
- I= **Intervention**
- **Introduction of hydrolysate vs elemental vs whole protein feed & to compare continuous vs bolus**
- C = **Comparator**
- **Time on parenteral nutrition for infants given the usual method of introduction of enteral feed in each individual centre during the 6 months prior to the study**
- O = **Outcome(s)**
- **Length of time patient requires parenteral nutrition**

Outline research methods

- Randomise infants to one of the three feed groups:-
- whole protein
- hydrolysate
- amino acid
- each feed to be given as bolus or continuously
- Aim for 100 cases in 4-8 centres over 18 months- 2 years

References

- Ksiazyk J et al. Hydrolyzed versus Nonhydrolyzed Protein Diet in Short Bowel Syndrome in Children Journal of Pediatric Gastroenterology and Nutrition 2002 35:615-618
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Fish-oil based intravenous lipid emulsion (Omegaven®) as a rescue in septic infants with intestinal failure and with or at risk of developing liver disease

PICO session
BSPGHAN Annual meeting 2015
Stratford-on-Avon

Dr. Huey Miin Lee (Paediatric ST5)
Dr. Ann Hickey (Consultant Neonatologist)
Dr. Jonathan Hind (Consultant Paediatric Hepatologist)

What is the Clinical and Cost Effectiveness of Omegaven®?

Background / rationale

- In infants with intestinal failure, it is known that episodes of sepsis can be accompanied by a significant deterioration in liver function.
- Recent studies have demonstrated that using Omegaven®, a fish-oil based intravenous emulsion which is high in ω-3 fatty acids, can improve or reverse IFALD. [1-4]
- Omegaven® has been increasingly used in King's College Hospital in neonates at risk of severe liver disease during episodes of sepsis as preliminary data suggests that Omegaven® protects the liver in these infants during episodes of sepsis.

The PICO

- **P** = Septic neonates at risk of severe liver disease
- **I** = Omegaven® (1g/kg/day) for short period during septic episodes, e.g. 2 weeks or 4 weeks
- **C** = SMOFlipid® (1g/kg/day) or no lipid during septic episode
- **O** = Bilirubin, triglyceride, CRP, essential fatty acid levels

Outline research methods

- **Trial design:**
  Multicentre Randomised Controlled Trial
- Strict inclusion and exclusion criteria
- **Comparing:**
  a) Omegaven® to SMOFlipid® both at 1g/kg/day
  b) SMOFlipid® at 1g/kg/day to no lipid
  c) Omegaven® at 1g/kg/day to no lipid
- **Likely number of patients needed**: 50 patients in each arm
- **Duration of study**: 2 years

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3. Diamond IR, Sterescu A, Pencharz PB, et al. J Pediatr Gastroenterol Nutr. 2009 Feb; 48(2):209-15.
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BSPGHAN Annual Meeting

PICO Proposals

Not selected for oral presentation

Wednesday 28th January 2015

Fecal inflammatory markers in infants with cow's milk protein allergy

PICO session

Dr Palittiya Sintusek

BSPGHAN Annual meeting 2015

Stratford-on-Avon

What is the Clinical and Cost Effectiveness of "Fecal inflammatory markers in infants with cow's milk protein allergy" ?

- Cow's milk protein allergy is one of the most common condition in children especially in infantile period.
- No ideal laboratory investigation nowadays is used to confirm the diagnosis.
- There is the therapeutic diagnosis by omitting cow's milk for the suspected children and many studies found that the children with diet restriction significantly have slow growth compared with the normal children.
- The definite diagnosis of cow's milk protein allergy and the timing of tolerance are crucial and should be concerned.

What is the Clinical and Cost Effectiveness of "Fecal inflammatory markers in infants with cow's milk protein allergy" ?

- Because of nonspecific symptoms of cow's milk protein allergy, it tends to over-diagnosis of cow's milk protein allegy in clinical practice.
- There are many non-invasive tests developed for this purpose such as skin prick test, serum specific IgE for cow's milk that are particular useful in IgE-mediated cow's milk protein allergy.
- However, the children suspected nonIgE-mediated cow's milk protein allergy that mostly present with gastrointestinal symptoms and skin manifestations, we find that skin prick test and serum specific IgE for cow's milk are usually negative.
- It is more difficult to differentiate cow's milk allergy in this group of patients.

What is the Clinical and Cost Effectiveness of "Fecal inflammatory markers in infants with cow's milk protein allergy" ?

- This study aims to develop the non-invasive laboratory investigations that help the paediatricians to diagnosis, follow up and management the paediatric patients suspected cow's milk protein allergy especially in nonIgE-mediated type.

The PICO

- **P = Infants suspected cow's milk protein allergy.**
- **I = Rechallenge with cow's milk**
- **C = Cow's milk restriction**
- **O = Primary→clinical response, fecal eosinophil-derived neurotoxin, fecal calprotectin**

  Secondary→skin prick test and specific IgE for cow's milk

Objective

- To study the value of fecal eosinophil-derived neurotoxin and fecal calprotectin as noninvasive markers for diagnosis cow's milk protein allergy in infant.

- To study the value of fecal eosinophil-derived neurotoxin and fecal calprotectin as noninvasive markers for follow up and reintroduced cow's milk protein to the infant with cow's milk protein allergy

The proposed study

First (day 1) - Collect all demographic data
- Investigation for skin prick test, specific IgE for cow's milk, stool for inflammatory markers
- Stool collection for inflammatory marker testing
- Treatment the infants by avoiding cow's milk (maternal restrict cow's milk if the infants receive exclusive breast feeding, changing cow's milk formula to aminoacid formula if the infant receives cow's milk before)

Second (day 14)
- Follow up and collect data about clinical response and physical examination
- Stool collection for inflammatory marker testing

Third (day 28)
- Assess clinical and physical examination
  If the infants improve, continue cow's milk avoidance
  If the infants do not improve, work up for another causes of these symptoms
- Stool collection for inflammatory marker testing

Forth (day 28 up)
- Re-challenge test
- Collect stool for inflammatory marker testing before and after challenge test (more than 2-3 hour after challenge test)

If positive challenge test —> continue cow's milk avoidance
If negative challenge test —> look for other mimic disease of cow's milk protein allergy

References

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Grapefruit extraction increases tacrolimus level in paediatric post-liver transplantation

PICO session

Dr Palittiya Sintusek
BSPGHAN Annual meeting 2015
Stratford-on-Avon

Background

- Liver transplantation is considered the most effective treatment in paediatric cirrhosis.
- However, after liver transplantation, there are short and long term complications occur especially from graft rejection.
- Immunosuppressive drugs play crucial role after liver transplantation.
- Tacrolimus and steroid are the main immunosuppressive drug used in this condition.

Background

- Tacrolimus (FK506) inhibits type I Tcell proliferation at T cell receptor. It is necessary to keep higher tacrolimus level after early period of liver transplantation.
- This drug is metabolized mainly through cytochrome P450(CYP)3A4.
- There are many drugs and foods effect the tacrolimus level; ketoconazole, methylprednisolone, dragon fruit, grapefruit etc.

Background

- Children use more tacrolimus dosage than adult in order to keep high target drug level.
- There is some concerns about side effect(from other metabolites) and cost of tacrolimus when we have to increase the dosage to keep the high target drug level.
- Using grapefruit which also metabolized through cytochrome P450(CYP)3A4 might increase tacrolimus level instead of increasing tacrolimus dosage.

The PICO

- **P = Children post-liver transplantation**
- **I = Tacrolimus and grapefruit extraction**
- **C = Tacrolimus**
- **O = Primary → target tacrolimus level**
  Secondary → safety from grapefruit extraction

Outline research methods

- Population: paediatric patients post-liver transplantation.
- Grapefruit extraction will add in the meantime of tacrolimus ingestion.
- Tacrolimus level follow-up closely.
- Monitor adverse effect of grapefruit extraction.

References

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Modulation of the gut microbiome by enteral diet

PICO session
BSPGHAN Annual Meeting 2015

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Why is this topic important? (1)

The Question: *Does enteral nutrition change the gut microbiome?*

Enteral feeds are first line therapy for children with Crohn's disease throughout the United Kingdom and in many other countries. The incidence of Crohn's disease has been on the rise over the past few decades highlighting the role of environmental factors in this disease.

One of the most important environmental factors affecting microbial composition is dietary preference.

Consumption of various nutrients affects the structure of the microbial community. Examples include differences in microbiota between breast fed versus formula fed infants or differences in microbial richness in people who consume a plant based versus a western diet, high in meat and fat.

Dietary modulation, faecal microbiome transplantation, and other methods to manipulate microbial communities may confer beneficial features of healthy microbiomes to define treatment goals.

Why is this topic important? (2)

While the effect of enteral diets on the intestinal immune system has been examined over the past 30 years, studies on how this affects the microbiome have been rare. We still do not completely understand how enteral nutrition works and what impact it has on the gut microbiome.

The aim of this project is to determine the effect of an enteral diet on the composition of the gut microbiome .

Thus far, only limited information on this topic has been gathered in humans, likely as a result of the challenge of setting up a large-scale controlled diet study. As a result, our understanding of the dynamic role of diet on the human microbiome remains incomplete.

The PICO

- P = **Children with IBS like symptoms but no inflammation in the gut**
- I = **Exclusive enteral diet therapy for 6 weeks**
- C = **no dietary manipulation (i.e. children with IBS like symptoms continuing their normal diet)**
- O = **microbiome composition**

Research methods

- Patients

Intervention group: Children with IBS like symptoms given an exclusive enteral diet for 6 weeks. Patients on antibiotics excluded.

Control group: Children with IBS like symptoms continuing their normal diet

- Samples

Stool samples collected prior to start of enteral diet. Repeat stool sample taken at 2 weeks and at the end of enteral diet therapy at 6 weeks. Stool samples at 0, 2 and 6 weeks for control group on usual diet.

- Power estimate: 30 patients in each arm of the study
- Duration of study: 6 weeks
- Methods: The faecal microbiota characterized at particular time-points by amplification and sequencing of the 16S ribosomal RNA gene V4 region using the Miseq platform.

References

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- Yatsunenko T, Rey FE, Manary MJ, et al. **Human gut microbiome viewed across age and geography.** Nature. 2012 May 9;486(7402):222-7.
- Fukuda S, Toh H, Hase K, et al. **Bifidobacteria can protect from enteropathogenic infection through production of acetate.** Nature. 2011 Jan 27;469(7331):543-7.

Use of Ferrell Valve Gastric Decompression System in Children who are gastrostomy fed

PICO session
BSPGHAN Annual meeting 2015
Stratford-on-Avon

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Background/Rationale

- Gastrostomy fed children frequently have symptoms of upper GI dysmotility (vomiting, pain, discomfort during feeds), which are difficult to control
- Adverse outcomes include multiple hospital admissions, poor carer experience and heightened anxiety, unnecessary fundoplication, progression to transpyloric feeding
- Use of this simple and cheap gastric venting system may alleviate a large proportion of the symptoms that are most distressing to carers, and mitigate against further medical intervention (often symptom driven)

The PICO

- **Patient group:** Gastrostomy fed children with symptoms of GI dysmotility
- **Intervention :** Farrell valve
- **Comparator:** "standard" practice
- **Outcome(s):** improvement in symptom score, feed tolerance; reduction in the following: hospital admissions/contact with secondary services, progression to fundoplication, progression to trans-pyloric feeding

Outline research methods

- Randomised controlled trial vs Cross over trial
- I haven't powered the study yet but it will be done in conjunction with colleagues from neurodevelopmental paediatrics.
- I envisage 6 months data collection.
- The farrell valves are quite cheap (1-2 pounds) so costs should be minimal

References

- Valve invented in 1986 but no papers exist as to its effectiveness of use
- http://www.corpakmedsystems.com/farrell2014.html

What is the natural history of paediatric fatty liver disease?

PICO session
BSPGHAN Annual meeting 2015
Stratford-on-Avon

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Prof. P. N. Newsome, NIHR Centre for Liver Research, Birmingham

What is the Clinical and Cost Effectiveness of studying the history of paediatric NAFLD?

Background and rationale

- Most common paediatric liver disease
- Few long-term prospective studies of natural history
- Likely high morbidity as (young) adults
- New treatments being trialled in adults
- Basis for additional (interventional) studies

PICO

P **Patient group** 2-17 years with USS-confirmed NAFLD (other causes excluded)

I **Intervention** Routine clinical care

C **Comparator** Age/sex-matched children

O **Outcome(s)** Liver-related mortality, cardiovascular mortality & morbidity, T2DM, sleep apnoea

Outline research methods

- Prospective cohort study
- Multi-centre, 200-300 patients (initially)
- Secure online database
- >10 years follow-up, with minimum 5-yearly measurements, bloods, & USS
- No biopsies required as part of study
- HES data, GP database data, clinic data for outcomes

References

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Gut Microbiota in Children with Biliary Atresia A Prospective Study

PICO session
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BSPGHAN Annual meeting 2015; Stratford-on-Avon

Biliary Atresia Outcome

- **Biliary Atresia (BA)**
  - Incidence 1 in 20 000
  - Progressive inflammatory destructive process of the bile ducts
- **Kasai Portoenterostomy (KP)**
  - Surgical procedure aimed at restoring bile flow
- 5- and 10-year survival rates with native liver
  - 60 and 45% respectively
- BA is commonest indication for liver transplantation in children
- Why do some children survive with native liver and others need transplantation?
- Could *Gut Microbiota* play a role in BA outcomes?

The PICO

- P = BA patients that receive liver transplantation
- I = Faecal Microbiota analysis
- C = BA patients that survive with native liver
- O = Composition of Faecal Microbiota
- T = follow up 18 months

Study (1)

- Prospective longitudinal Study
- All newly diagnosed BA patients at KCH
  - Over a 2 year period
  - In 2012; n= 18
  - In 2013; n=26
- Follow up; 18 months
- Primary Endpoints at 18 months;
  1. Alive with native liver
  2. Liver transplantation

  In 2012/2013; 50% had native liver/liver transplantation at 18 months

Study (2)

- **Sample measurements;**
  - **Faecal:**
    - Microbiota analysis by 16s rDNA sequencing
  - **Blood:**
    - Laboratory indices, immune function (immunophenotyping, cytokines..)
  - **Tissue/intra-abdominal:**
    - Duodenal and biliary epithelial cells, bile, portal venous blood
    - Microbiota analysis by 16s rDNA sequencing
- **Timing of samples;**
  - **Faecal/blood**
    - Diagnosis (KP)
    - 1, 3, 6, 12, 18 months
  - **Tissue/intrabdominal**
    - Diagnosis (KP), biliary surgery, upper GI endoscopy, liver transplantation

Outcome

- Does faecal microbiota composition play a role in BA outcome?
- Could there be a role for antibiotics/probiotics in BA?

References

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- *Am J Gastroenterol Suppl (2012) 1:9–14; doi:10.1038/ajgsup.2012.3The Intestinal Microbiota and Liver Disease Jasmohan S Bajaj MD, MS1, Phillip B Hylemon PhD2 and Zobair Younossi MD, MPH3*

Exhibitor:

PharmacoMedics

Charities:

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Crohn's and Colitis in Children

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