Australian and New Zealand Association of Paediatric Surgeons
Formerly the Australasian Association of Paediatric Surgeons

DECEMBER 2008

President's Report

There are endless enquiries into medicine and its delivery, and endless demands for statistics about health care and its delivery. Indeed, the cynic would say that if the money spent on enquiries and the gathering of statistics was redirected to patient care, many of our shortages would be over. Be that as it may, we live in a world where there is more intrusion into our profession than ever before.

Much of the measuring that is being done really relates to process, not outcome. The waiting list is a classical example, and the cynical manipulation of such indicators reached a zenith in NZ when all patients waiting more than 6 months were deemed to be no longer waiting but in need of review by their GP, and re-referral if necessary. Another great example is hospital inspection by the ACHS. This is an extremely expensive exercise for the hospital. There is a warm glow of pride when one's hospital passes inspection. There is, however, to the best of my knowledge, no evidence that patient outcomes are improved by it. Staff may know better where the fire extinguishers are, but patient outcomes are not measured, nor are they known to be better.

And that is the real point. Measuring outcomes of health care is difficult. It is even more difficult for our specialty than most because very few of our patients die, death being the most easily definable and measurable end point, and those patients of ours that do die generally succumb for reasons other than their surgery.

Did someone say "clinical indicators"? In theory, these should be the answer. The practical problem is to get ones that are meaningful. We know and are grateful for the enormous amount of work that Tat Hin Ong put into these in the early days of their development. We know that the published work of Spencer Beasley has shown that they can be used to assess whether a Unit that does small amounts of paediatric surgery has results that are comparable with those of major centres, and hence where the patients should receive care. But we are still struggling to find CIs that have meaning within the framework of major tertiary centres where most of us work. Much of our difficult work is only measurable in the long term – how many of us know how the neonates we operated on for major abnormalities are in adult life, 20 or 30 years later? What are the results of orchiopexy as far as

fertility is concerned compared with norms, or compared with unoperated patients?

Even if that information was available, it is likely that techniques would have changed (hopefully for the better) so it would be of little relevance.
Even more difficult is getting any idea of patient (and family) satisfaction. We know that the emotional aspects of care have a strong influence on recovery. As well, the family that had a positive, happy encounter is, I suspect, more likely to present to medical care early in the course of a disease if one of them develops a symptom, and so make the outcome of subsequent illnesses better. Having lots of families thank us is no gauge: surveys by medical indemnity companies show that the approval rating of the average surgeon is 93%, so if only 8 out of 10 of the families that you deal with are happy, you are relatively unpopular, more likely to have legal action or a formal complaint made, and need to lift your game.

So this is, to me, one of our major challenges. How can we measure our outcomes in a meaningful way so that we can assess our own efforts, address jurisdictions about the facilities that we have, and genuinely gauge the effect of changes that we make in an effort to improve our results?

Audit is not an answer because it also lacks sensitive measures of outcome. It is, however, a very powerful tool for detecting major deviations from a norm. Sometimes deviations detected by an audit have a valid reason that may be related to casemix. For example a referral hospital that offers ECHMO may have much worse figures for CDH than one that does not offer ECHMO, or one that drains obstetric units that have a more aggressive policy of termination for CDH. But it is likely to flag significant deviation from the norm so that such deviations can be looked at soon after they start. The whole sorry affair of Dr Kossman may never have occurred if The Alfred had had an effective audit program in which he participated, and both Patel and Reeves would almost certainly have been detected much earlier if their work had been subject to audit.

For audit to be effective, there must be a mechanism of dealing with outliers. This should include an initial examination to ensure that there is not a perfectly valid reason for the apparent anomalies, and then a mechanism of helping the surgeon or department to improve results. If an individual persistently refuses to co-operate with attempts to rectify problems that are identified, there has to be a method of sanctioning that person eventually. This may be to notify the hospital or credentialing committee where he/she works, or even the registration body of that jurisdiction.

It may seem as if I do nothing but write about the negative aspects of our life, but every "Butcher of Bundaberg", "Butcher of Bega", and Dr Kossman that hits the news makes politicians and bureaucrats even more determine to regulate and control us.

PRESIDENT'S REPORT

They hide behind the "good of the public" mantra. The College has a major role in providing balance to these moves, and trying to ensure that they do not impose ridiculous restrictions on surgeons. Getting our own house in order is the best defence against bureaucratic interference. So if you have thoughts about these matters or, even better, some ways of solving the problems, please let me know.

On a happier note, I want to let you all know that the Rowan Nicks Scholarships scheme also includes graduates from the UK and Ireland. Details are obtainable from the web. So if you know of a young surgeon in those countries who might benefit from time spent in Australia or New Zealand, do please look into the criteria and see if that person might apply for a Rowan Nicks Scholarship.

To mention some topics that have appeared here before. The electronic log book is much closer to being in general use. The problem of bariatric surgery is being moved along although slowly. Supervisors of training have greatly increased work, so it is important that all surgeons who have trainees in their hospital share the load of in-training assessments. Happily, the possibility of some financial recompense for their efforts, and some administrative support is getting closer, as these two matters are now in the accreditation criteria for inspecting training posts. Finally, another appeal to all of you to participate to the maximum in teaching outside your own hospital, such as ASSET, CRISP, and EMST courses. While they take time, they are very rewarding, and essential for producing the surgeons of the future. Didn't you get taught that way?

Don't forget to put our ANZAPS meeting in Fiji in your diary - or even better, tell the family that it is on so they nag you to go – July 12 to 15.

I hope all of you have some time off over the festive season to spend with family and friends, and prepare yourselves for another year. May 2009 be a good year for our patients, for us, and for our profession.

Mr Hugh Martin, AM FRACS
President

Congratulations to Spencer and Christie Beasley and family on the safe arrival of Peter Beasley during December.

BOARD OF PAEDIATRIC SURGERY

"It is a privilege to have a trainee." During a recent hospital inspection a colleague made this statement as part of a longer discussion. It gave us pause. At the risk of sinking into a sticky Hallmark mess of sentiment, we do need to be reminded that the privilege of guiding, teaching and inspiring the next generation of paediatric surgeons should be equally stimulating, challenging and a reminder of the altruism that led us into our careers. We are provoked by the trainees' questions. Why do we approach clinical problems so? We are challenged to find the time to teach amongst the never ending stresses of public commitments and underfunding. We did enter paediatric surgery stimulated by the breadth of expertise required, the delicacy of the surgery, the interactions with the families and children and surprisingly shamefully the desire to do good. So we are all privileged to have trainees.

Which lofty talk leads to discussion of the Board hospital post inspections. We are there to ensure the training experience is the best possible for the trainees. Is there the caseload and the commitment to teaching? Nowadays that also means safe work hours and reasonable study and on-call facilities. But we approach the inspections in the collegial manner befitting a speciality with small numbers. We would prefer to hear about problems that need our help to negotiate solutions with the jurisdictions than to be adversarial. Different posts will offer different experiences or strengths, not all posts are equal. That is why we balance the training by having trainees shift between posts. We understand some posts have vey individual support from consultants but maybe no burns or advanced oncology. Some are better for academic meetings and some are stronger for responsibility and leadership. So welcome the differences and the inspection teams for what we can do to help the trainees.

Directed On-line Group Study learning tasks have been recently decreased from three to two a year to acknowledge the work load required for the trainees and the markers. Remember both D.O.G.S. and Critical Appraisal Tasks are learning and study tools. They are supposed to encourage critical learning and ability to process information, formulation of diagnostic and management strategies, foster debate amongst peers and supervisors and promote trainees time management. They are not to be onerous time consuming manuscripts or test the ability to cut and paste texts.

SET One and Two years continue to be problematical. There is no doubt we are under pressure to provide training completely within paediatric surgery units for our trainees, without the need for the general surgeons or other specialities. The Board has debated and defended at length on this subject. I have previously expounded on our generalist speciality – the antenatal to the adolescent, and all the pathology and systems that entails. We have to train the ability to surgical approach all the physiological systems in a finite time. The only way we currently have to maximise this is to put our trainees in busy General Surgery posts with broad caseloads that will give them intense exposure for example in colorectal and biliary. We are keen to hear your opinions to approaching these aspects of training.

We are often asked about private hospital and overseas posts. At the moment these posts would have to provide the same access to formal teaching sessions, supervision, ward rounds and progressive hands on experience in operative surgery and clinical management decision making (in other words diagnosis and management) as current ANZ public posts. What is required by an advanced trainee is different from an FRACS Fellow or a surgical assistant. Bearing that in mind the Board is continually open to discussion of future training solutions. Remember training is not just about numbers of operations: we teach the responsibilities

of care, the decision to not operate, perioperative management and investigation, team work and approach to the family, judgement, insight, self assessment, strategies for continuing education, management of health resources and ability for community health leadership to name a few "nontechnical" competencies.
Congratulations to Celine Hamid, Peter Ferguson and Gordon Thomas on their recent success in the Fellowship examinations.
Thank you to all who help train – the consultants and the supervisors. Thanks to my fellow Board members and particularly to Mrs Rebecca Letson for their guidance and hard work. For light reading over the break check our college website with the training and selection regulations which have dominated and explained the Board!
Merry Christmas and Best Wishes for 2009!

Assoc. Prof. Deborah Bailey F.R.A.C.S.(Paed.) M.B.B.S.
Chair Board of Paediatric Surgery

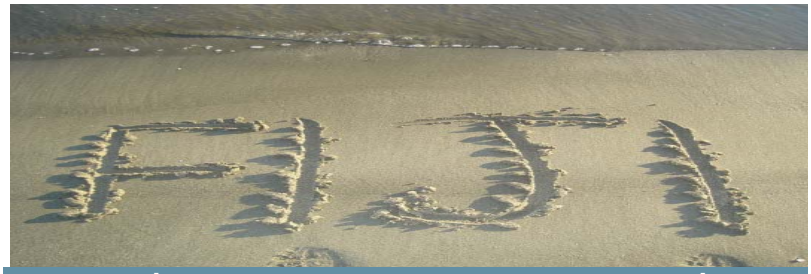
The Board of Paediatric Surgery and SET Program in Paediatric Surgery appreciates the support of the Karl Storz, Q-Med (Deflux) and Johnson & Johnson Medical for the 2008 Registrar Annual Training Seminar.

MEETINGS AND COURSES

2009 Annual Scientific Meetings of the Australian and New Zealand Association of Paediatric Surgeons and the Australian and New Zealand Society for Paediatric Radiology

Communication Combining Cultures Collaboration

The Westin
Denarau Island, Nadi, Fiji
12 to 15 July 2009



Enjoy the opportunity to meet and discuss latest developments within Paediatric Surgery and Paediatric Imaging in magnificent surroundings. The Scientific Program will be supplemented with a Family and Social Program to capitalise on the timing of the meeting for most Australian and New Zealand school holidays.

Please direct all inquiries to:
Ms Rebecca Fielding
Queensland Conferencing & Events
E-mail: rfielding@qce.net.au
Telephone: +61 (0)7 3356 5804

Bula!

Phil Morreau has been working extremely hard with the organising committee to put together an 2009 Annual Scientific Meeting that is sure to appeal on all fronts - scientific component, social and culture component and of course the beautiful location of Fiji.

Scientific Visitor Steve Fishman will be presenting on topics including Classification and Diagnosis of Vascular Anomalies and Liver Haemangioma - A New Paradigm. Paul Babyn is the Radiology Scientific Visitor (from Toronto) and will also be presenting, with topics including Investigation of an Abdominal Mass in Children and Radiology for the Paediatric Trauma Patient.

Joint sessions with Paediatric Radiology have been planned along with stand alone surgery sessions.

The Westin (meeting venue), Sheraton Villas and Sheraton Resort (part of the Starwood Group) are each within a leisurely five minute stroll along the beach and will offer a variety of accommodation options suiting those travelling solo to those attending the meeting with family in tow. The resorts feature a Kids Club, Golf and Tennis Club , Day Spa and Tours Desk.

The Organising Committee will be calling for abstracts and e-mailing registration details to all ANZAPS members early in 2009.

Thanks also to Kiki Maoate and Jitoko Cama who are representing the Association on the Organising Committee.

MEETINGS AND COURSES

2009 International Surgical Week and WOFAPS Meeting - Adelaide
Monday 7 September to Thursday 10 September

An extensive and interesting program has been put together by Tony Sparnon in conjunction with surgeons from Australia, New Zealand and around the world. Sessions include Trauma: skill maintenance in an era of non-operation; Thyroid carcinoma in children and adolescents; Vascular anomalies and tumours; What's new in NEC and neonatal conditions; Minimally invasive surgery - still at the forefront or now mainstream; Recent changes in undescended testis and hypospadia.

The conference dinner will be Tuesday 8 September at the magnificent Adelaide Art Gallery.

Further details including registration details will be forwarded to ANZAPS members during 2009.

2010 Scientific Visitor - Call for Nominations

Further to a request in the August 2008 newsletter, the Association Executive Committee would like to call for further nominations for the 2010 Scientific Visitor. A nomination has been received to invite Dr Robert C Shamberger MD, Chief Department of Surgery, The Children's Hospital Boston. Dr Shamberger has a special interest in surgical oncology and is involved in many services and programs including Hodgkin's Lymphoma Program, Solid Tumor Program and Pediatric Melanoma and Pigmented Lesion Program.

Members are invited to submit further nominations to college.anzaps@surgeons.org by 1 March 2009.

CRITICAL APPRAISAL TASK

SET Trainees in Paediatric Surgery are required to complete two Critical Appraisal Tasks (CATs) as part of their program requirements. Below is an extract of the Necrotising Enterocolitis CAT judged the best submission by the markers. This extract is from SET4 Trainee Dr Amiria Lynch.

Question 1: What are the currently accepted etiologic factors of NEC and the evidence supporting their relevance?

Necrotising enterocolitis (NEC) was first described in 1965 and multiple theories have been described to explain this disease[1]. NEC is a disease affecting almost exclusively preterm infants who have had enteral feeding (>90%).[2] Both the incidence and related mortality is inversely proportional to gestational age and birth weight[2, 3]. Pathogenesis theories have attempted to identify what it is about the premature infant that makes it susceptible to this disease. The role of enteral feeding has also been extensively investigated, and the histological findings of coagulative necrosis can give some clues as to the aetiology of this disease. It is likely that there is interplay between hypoxia, feeding, sepsis, ischaemia-reperfusion, vulnerable gut, inflammatory cascade and immature immune systems to create NEC.

Kosloske described the interaction of three critical events in the pathogenesis of NEC: intestinal ischaemia, colonisation of pathologic bacteria, and excess protein substrate in intestinal lumen[4]. Essentially the initiating event is now felt to be damage to immature intestine which leads to bacterial invasion thus initiation of the inflammatory cascade causing vasoconstriction and further destruction and perforation.[5, 6]

●Enteral feeds.
Over 90% of infants with NEC have been enterally fed, providing the substrate for bacterial proliferation [1]. The timing of feeds and the speed of advancement are contributing factors but there is no consensus on the safest way to proceed.[3] Advancement of feeds faster than 20kcal/kg/day are not recommended by some but found to have no impact by others including a Cochrane review[1]. Early trophic feeds are considered to be preventative at best, and do not increase the incidence at worst[1, 7]. Breast milk is protective with an incidence of 1.2% compared with 7.2% formula only and 2.5% in those on combination[7]. It is felt that this is because of multiple immunoprotective and antiinflammatory factors present in breast milk that are not in formula[3, 8]. This is especially true as bacterial colonisation occurs from the mother at birth, and specific protective factors to those organisms are passed on in her milk[8]. This specificity may explain why donor milk is not as effective as maternal, while still being better than formula[1].

●Immature intestinal barrier
The key structure in the intestinal wall that allows selective passage of molecule is the tight junctions between epithelial cells, these develop by the tenth week of gestation[2]. In the newborn they are immature, and easily damaged by bacterial products and macromolecules leading to increased permeability.[5] In preterm infants the composition of the protective mucin layer is deficient in protein, and decreased mucous production predisposes to damage from bacteria and macromolecules.[2, 5] There is low intraluminal gastric acid and proteolytic activity in the preterm infant, thus increased vulnerability to bacterial overgrowth and translocation.[3, 5, 7] Epidermal growth factor (EGF) has trophic and maturational effects on intestinal mucosa, and it is absent in formula but present in breast milk. It is deficient in preterm infants and this may predispose to NEC.[1, 4, 9] This is supported by administration in rat model leads to lowered incidence of NEC.

●Immature intestine motility and digestion
Gastrointestinal motility begins in the second trimester and matures though the third trimester and after birth[2]. Immaturity leads to increased transit time and stasis in the premature intestine which provides a medium for abnormal bacterial colonisation[2] The maturation of the motility in a premature infant is accelerated by enteral feeds. The digestion capabilities of the premature intestine are also immature leading to incomplete digestion especially of carbohydrates. These are though to provide a substrate for bacteria, with resulting production of reducing substances, organic acids and hydrogen gas, as well as causing direct damage to the intestinal barrier [1, 2].

●Immature intestinal circulatory regulation
The original theory of aetiology proposed for NEC was hypoxic-ischaemic injury as being the initiating event. This was based on the diving reflex where systemic circulation is diverted away from the intestine to supply brain, heart and kidneys in times of birth stress as described by Lloyd in 1969[6]. This has now been essentially discounted as the diving reflex is seen perinataly, and there has been no correlation with the incidence of NEC and birth, or postnatal hypoxic events.[4] Sustained adrenergic stimulus in infants does not lead to intestinal decreased blood flow or tissue hypoxia.[6] There is however, likely to be contributing factors from the way that the premature infants intestinal blood flow is regulated. The vascular resistance to the bowel, thus controlling blood flow, is from the submucosal arterioles. This is managed by a balance

between vasoconstricting endothelin 1 (ET-1) and vasodilation from endothelial derived nitric oxide (NO); an imbalance leads to tissue injury. In the infant the baseline tends to be in favour of vasodilatation to allow adequate flow to the rapidly growing intestine[10]. The stimulation for nitric oxide synthase (NOS) and therefore increased NO is increased blood flow and therefore vasodilatation in the newborn [6, 10]. A disruption of endothelium leads to disruption of NOS and intensifies ET-1 thereby giving unopposed vasoconstriction leading to tissue hypoxia and ischaemia.[10] There is increased expression of ET-1 in resected specimens from NEC compared to non-NEC specimens and increased in those areas with the most intestinal damage.[6] Three factors damage the endothelium: ischaemia-reperfusion injury, sustained low flow perfusion, and inflammatory mediators[10].

●Ischaemia- reperfusion injury
Ischaemia is caused by accumulation of oxygen-free radicals formed from the conversion of xanthine dehydrogenase to xanthine oxidase, these contribute to intestinal injury.[4] Reperfusion then has xanthine oxidase converted to large amounts of superoxides that cause severe tissue damage. Nitric oxide (NO) and superoxide dysmutase provide resistance to this reperfusion injury.[4] The mechanism and timing of ischaemia has not been clearly identified. No thromboembolic event is supported on the histological data.

●Inflammatory mediators
Immature intestinal cells have a higher concentration of pro-inflammatory cytokines[4]. Platelet activating factor (PAF) is an endogenous phospholipid mediator produced by inflammatory cells, platelets and endothelial cells, and is regulated by PAF-acetylhydrolase. PAF - acetylhydrolase activity is decreased in neonates, and is present in breast milk. PAF is increased in enterally fed infants, has a cytotoxic effect and plays a major role in the initiation of the inflammatory cascade.[4, 11] Its role has been confirmed in animal and human studies[11].
Tumour necrosis factor α (TNF-α) causes damage when injected with lipopolysaccharides and induces PAF and is likely to contribute [11].
Nitric oxide (NO) is a protective modulator of intestinal mucosa, produced by nitric oxide synthase (NOS). In the presence of inflammation NO production can be induced and in combination with superoxide can actually contribute to intestinal injury, and increase apoptosis of enterocytes[12]. A decreased level of epidermal growth factor, an important factor in regeneration of enterocytes has also been implicated.[9]

●Infection
Bacteria undoubtedly contribute to the aetiology of NEC but the inability to isolate a common organism makes it unlikely to be the initiating factor. Two mechanisms are involved, fermentation of carbohydrate substrates leads to production of hydrogen gas, leading to intramural gas and bowel distension. Secondly the translocation of bacteria activates immune response.[3] Abnormal colonisation of the intestine is common in NICU babies due to antibiotic use, presence of feeding tubes and exposure to hospital flora. Enterobacteriaceae sp and Clostridium sp are commonly isolated. The bowel is usually colonised by the second week of life from flora from the maternal vagina[3, 4]. Over time the organisms change from E coli and streptococci to lactobacilli and Bacteroides, introduction of enteral feeds alters the flora with different organisms for breast and formula milk.[3] Bacterial toxins are likely to play only minor role, if any, as there have been few studies able to identify these[3].

●Immature innate immunity
In the newborn antigen degradation, processing and presentation to antigen presenting cells is less effective, therefore there is less ability to detect and respond to antigens. [5] They also have fewer and less responsive T and B cells leading to a dampened proliferative response[5]. Defensins, antimicrobial products produced in Paneth cells are also decreased in premature infants[5]. Perhaps most importantly there is decreased secretory IgA, the main inhibitor of bacterial and viral attachment in the intestinal lumen, this doesn't peak until 4 years of age. [4, 5]

●Medications
Pre and post natal steroid administration seems to be protective most likely by enhancing the speed of intestinal maturation[1]. Caffeine, erythromycin and indomethacin have all been implicated in cause but have not been verified in more vigorous studies.[4]

In summary, a subclinical insult with brief hypoxia or ischaemia causes damage to the immature intestinal barrier. In the presence colonisation of the intestine, bacteria bind to injured mucosa leading to an inflammatory response. Abnormal counter-regulatory mechanism leads to increased permeability of mucosal cells and bacterial translocation. Amplification of inflammatory response with activated neutrophils cause further mucosal injury from inflammatory mediators and reactive oxygen species. Maladaptive vasoconstriction worsens ischaemia and reperfusion injury. A cycle of feedback mechanisms leads to coagulative necrosis and perforation.

DIRECTED ONLINE GROUP STUDY

SET Trainees in Paediatric Surgery are required to complete two Directed Online Group Studies as part of their program requirements. Below is an extract of the Lung Lesions DOGS judged the best submission by the markers. This extract is from SET4 Trainee Dr Torey Lawrence.

Question 1: Outline the Differential Diagnosis

On 1/09/2008 22:36, **Torey** said:

This is a chest x-ray (CXR) and chest CT scan of a 17 year old patient.
The patient is from Sudan with limited normal investigations and history of a CXR 2 years prior which demonstrated a right lower lobe mass.

The CXR demonstrates an intra-parenchymal right lower lobe mass. The mass overlies the diaphragm shadow and on the lateral CXR is seen to be in the right lower lobe. The lesion is well circumscribed and does not appear calcified on the x-ray. The right hilum appears more prominent than the left.

The CT scan is a contrast CT with and without lung windows. This CT demonstrates and well circumscribed large mass in the right lower lobe (?size, scale not present). The mass is non-enhancing and the surrounding wall is also non-enhancing. It is homogenous. There are no Hounsfield units however the lesion appears a similar density to the liver and may be solid or a cystic structure containing fluid. There are no obvious septae or calcification. The lesion is displacing the vessels but is not obviously being supplied by an aberrant vessel in the images provided. There is also some associated air trapping.

Based on the history and scans available I believe the most likely differentials are: Hydatid cyst (most likely) and then bronchogenic cyst.

1. Hydatid cyst - caused by a parasitic tape worm in the larval stage (Ecchinococcus granulosus or Echinococcus multilocularis). The lung is the second most common location of hematogenous HC spread in adults and it is probably the most common site in children. Often seen in the right posterior lower lobe as in this case (8). Lewall et al have described a pathology-based classification for this disease. This case is likely to be a Type I HC, which appears as a well-defined, round or oval cystic mass/opacity with an attenuation density near that of water (3-30 HU) that can resemble a neoplasm. Calcification (0.7% of cases) and daughter cysts are rarely seen in lung HD. Bronchiolar erosion can lead to air between the endocyst and pericyst can produce a "crescent or inverse crescent sign" which I cannot identify clearly in this case. A "water lilly" sign develops if air continues to enter the cyst cavity, (an endocyst membrane floating in the most dependent part of the pericyst cavity) also not seen in this case.

2. Bronchogenic cyst- are one type of anomaly that develops from abnormal ventral foregut budding and airway branching (1). This lesion may be a bronchogenic cyst as they often have associated air trapping and present later in childhood. Imaging shows a well-defined spherical mass and CT attenuation commonly ranges from -10 to 10 Hounsfield units depending on the protein content of the fluid. Furthermore, More common in the lower lobes. However, they are more commonly mediastinal or perihilar rather than intra-parenchymal (however results are variable and some reports have found the up to 70% of BGCs are located within the lung parenchyma (5)) and 2/3rds will be aerated and communicate with the bronchial tree (2).

There are many other possible differentials including:

Congenital:

1. Bronchocoele associated with atresia
2. CCAM - in particular Type III. Can present as a homogenous mass. Although there are individual small cysts that connect to the airway this cannot be seen on imaging (2).
3. Sequestration- most commonly seen in the lower lobes (1) and can present as a homogenous soft tissue mass, although no aberrant vessel identified on the CT images available. Lesion often becomes non-homogenous post contrast (4).

Acquired:

1. Infective
a. Fungal - pulmonary mycoses may present with similar features to TB and be nodular in nature.

b. TB- even with negative sputum, may be primary TB.
3. Pseudotumours- more common than neoplastic
a. Round pneumonia of childhood
b. Plasma cell granuloma (inflammatory or postinflammatory pseudotumour). Most common primary lung mass in children. Usually solid and well demarcated. Normally peripheral and non-enhancing, +/- calcification. However, normally the Hounsfield number is higher.
4. Neoplastic
a. Malignant lesions should be considered however, commonly the nodules/mass enhances post contrast unlike this case.
i. Primary - rare. Bronchial adenoma (or bronchial carcinoid, mucoepidermoid carcinoma, adenoid cystic carcinoma). They do frequently have associated air trapping and are well defined as in this case but are usually lobulated and elongated. This is unlikely to be bronchogenic carcinoma as they are usually a central mass with bronchial obstruction. Mesenchymal tumours (sarcomas) may be large solid or cystic lesions as seen on this CT. The most common in this group are pleuropulmonary blastomas, which may appear like benign cyst however, usually show a solid rim of tissue sometimes in a whorl-like pattern and area of central necrosis (1).
ii. Secondary - More common. Can be solitary. Most are round, sharply defined and homogenous with soft tissue density and is therefore a possible differential in this case
iii. Lymphoma - lung not commonly involved and more commonly present as mass like lesions of consolidation with small nodules (1).
b. Benign
i. Hamartoma - the lungs normal elements in abnormal proportions. This lesion may be a hamartoma as the usual radiological appearance is one of a smooth round or oval, sharply defined mass. These lesions also only occasionally calcify and are usually seen in adults or older children (1).
ii. Chondroma - can be a solitary nodule but unlikely in this case as they are usually calcified (1)
iii. Haemanigioma - unlikely, would expect this to enhance on the CT

References
1. Ed. Kuhn, Slovis and Haller (2003) Caffey's Pediatric Diagnostic Imaging. Tenth Ed.
2. Daltro et al. (2004) CT of congenital lung lesions in pediatric patients. Pictorial essay. AJR 183: 1497-1506.
3. Yuksel et al. (2007) Hydatid disease in rare locations a pictorial essay. Korean Journal of Radiology. 8(6): 531-540.
4. Mata et al (1990) CT of Congenital Malformations of the lung. Radiographics. 10: 651-674.
5. Oldham et al. Eds. (2005) Principles and Practice of Pediatric Surgery.
8. Gottstein and Reichen (2002) Hydatid lung disease (echinococcosis/hydatidosis). Clin Chest Med 23: 397-408.

Professional Practice Case Raised by Dr Torey Lawrence

Man's best friend!

On 15/09/2008 22:33, **Torey** said:
This is an interesting case that presented to our hospital. A 12-year-old boy from Orange presented with a 4-week history of a cough, urticarial rash and fever. He had been commenced on Rulide with no improvement and subsequently had a CXR which showed a cystic structure / lung abscess. He was noted to be coughing up solid material. He had a CT scan, which showed 2 cysts in the right lung and a liver cyst in segment 7/8 of the right lobe. The sputum sample showed a laminated membrane and hooklets suggestive of hydatid disease. Serology was positive for hydatid disease. Interestingly the father was a sheep farmer and their dog had been witnessed eating the entrails of dead sheep and was also known to go into the child's room and lick the patient on the face while he was in bed.

How would you manage this case? Does having a ruptured lung cyst alter the management? What would your long-term follow up, and management, be for this patient?

The best submissions for CATs and DOGS are available on the Royal Australasian College of Surgeons website www.surgeons.org in the Education & Trainees > Training > Paediatric Surgery > Forms area.

OTHER NEWS

CONGRATULATIONS TO SET PROGRAM TRAINEE DR TAMARA BONNEY WHO WAS AWARDED THE FOUNDATION FOR SURGERY JOHN LOEWENTHAL RESEARCH FELLOWSHIP FOR HER TOPIC "THE ROLE OF ANDROGEN IN TESTICULAR DESCENT". DR BONNEY'S SUPERVISOR IS PROFESSOR JOHN HUTSON.

You are invited to
Dr John Pitkin's Farewell

At
Lorimer Dods Lecture Theatre
Level 3 Education Centre
The Children's Hospital at Westmead
Sydney New South Wales
Corner of Hawkesbury Rd & Hainsworth St
Westmead NSW 2145

Wednesday, 11 February 2009
The meeting will commence at 0930 am

The provisional programme is
0930 Session 1 – Day Stay Surgery
Session 2 – Trauma
Lunch
Session 3 – Thoracic Surgery
Session 4 – Laparoscopic Surgery

Contributions to the ANZAPS Newsletter are encouraged. Please send through news of your seminars, events, personal achievements, surgical positions within your hospital to college.aaps@surgeons.org.

WARM WISHES FOR A SAFE AND MERRY FESTIVE SEASON!

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