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LETTERS

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Category D: unknown whether ill treatment is cause

I would like to congratulate Southall and colleagues for their very important paper, which represents extremely well the clinical and practical reality of the spectrum of child abuse.[1]

However, I have one fundamental concern relating to unexplained subdural haematomas. The authors state that "sometimes the parent is ignorant about the extent of damage that the impulsive act may cause. For example, in some societies it is not generally known that shaking of a young infant can tear veins around the brain." It is suggested that because of widespread publicity in the UK about shaken baby syndrome such injuries might be best classified under category A. Although perhaps unfashionable, there is an increasing acknowledgement that we understand very little about the mechanism of this often catastrophic event.[2–7] If the purported mechanism of injury (violent shaking of the inconsolable infant) is correct, then it would be logical to predict that the peak incidence would be in infants aged under three months. Yet the actual peak of unexplained subdurals is around eight months.[8] Until we have a much better understanding of this condition, I suggest that the proposed classification should also have a Category D (unknown whether ill treatment is cause of the injury). This category would enable appropriate classification of those previously well cared for infants who have no other signs of injury but present with subdural haematoma, retinal haemorrhages and "no adequate history of injury". Even in those infants that have rib fractures we need to consider whether there may be a less sinister explanation in some cases.[9–11]

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Gentamicin usage in newborns: an audit

This is in response to the letter by Grant and Macdonald on medicines for children and gentamicin toxicity in *Archives of Disease in Childhood*.[1]

Recently we audited our gentamicin regimen (2.5 mg/kg/dose; 24 hourly for < 29 week postconceptional age (PCA); 18 hourly for 29–35 week PCA; 12 hourly for > 35 week PCA) because of concerns that it resulted in too many subtherapeutic peak levels. We prospectively audited 50 sets of levels. Trough levels were determined just before and peak levels one hour after the third dose. Desired levels were trough < 2 µg/ml and peak 5–10 µg/ml. Most of the peak levels (92%; 46/50) were < 5 µg/ml. Trough levels were < 2 µg/ml in 98% (49/50). During the study period, 108 sets of levels were analysed by the microbiology department. A peak level of < 5 µg/ml was noted in 100/108, and a trough level of < 2 µg/ml in 107/108.

We changed our gentamicin regimen (4 mg/kg/dose; 36 hourly for < 28 week PCA; 24 hourly for ≥28 week PCA; trough levels determined before the third dose and peak levels not determined routinely), guided by current evidence,[2–5] and prospectively re-audited 60 levels. We randomly determined 20 peak levels; these were in the range 5.8–7.5 µg/ml in 16/20. Trough levels were < 2 µg/ml in 97% (58/60). During this study period, 100 trough levels were analysed in total, and only 4/100 were ≥2 µg/ml, the highest being 2.3 µg/ml.

We are happy with our new gentamicin regimen as it is practical and easy to remember. It achieves therapeutic levels without any added risk of toxicity. We have stopped routinely determining peak levels, resulting in less trauma and blood sampling for delicate newborns and the saving of laboratory time. The decision to not determine peak levels routinely is based on current evidence[2–5] that a dose of 4 mg/kg is highly likely to give peak levels in the desired range. Discretion, however, will have to be used in clinically septic newborns. In the long run, it should result in significant cost savings, as analysing the gentamicin levels has been reported to represent 75% of the cost of using this relatively inexpensive drug.[4]

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Kasabach-Merritt syndrome and interferon alpha: still a controversial issue

We read with interest the paper by Akyuz and colleagues,[1] which described a 2 year old patient with a Kasabach-Merritt syndrome (KMS) secondary to an infiltrating angiolipoma, who was successfully treated with interferon alpha 2a (IFN-alpha).

The authors did not emphasise the increasing body of concerns associated with the use of IFN-alpha in children affected by KMS. Indeed, several authors have recently warned about potential adverse effects related to the use of this drug, the most worrisome being spastic diplegia.[2–4]

Although IFN-alpha has been shown to be an effective therapy for patients with KMS,[5] it may cause transient or permanent neurological disabilities.[6] Furthermore, neurotoxicity of IFN-alpha, the pathogenesis of which remains unclear, is usually detected late during the course of treatment, and early diagnosis may result very challenging particularly in young children, who appear to be at higher risk. Although neurological complications may spontaneously reverse after discontinuation of IFN-alpha, some patients may experience permanent disabilities.

Unfortunately, no predictive risk factors regarding either the onset of symptoms or the reversibility of neurological deficits have been identified. This precludes a proper counselling about the actual risk of neurological deficits associated with long term treatment with IFN-alpha.

Further controlled studies are urgently needed in order to answer these questions.

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Table 1 Children with CF and renal failure associated with antibiotic treatment

Patient	Age	Weight (kg)	Other related problems	Pre-antibiotic plasma Cr (µmol/l)	Antibiotics used (mg/kg/day)	Gentamicin concentration* (mg/l)	Dose adjusted to (mg/kg/day)	Day first raised Cr noted	Biopsy findings	Peak Cr (µmol/l)	Treatment	Outcome at 3 months; Cr (µmol/l)
1	9 y	23	Liver disease Constipation	64	Gentamicin 10 Cefuroxime 200	13.9 peak (D8)	Stopped	9	ATN	862 (D16)	Conservative	43
2	4 mth	5.6	Diarrhoea	49	Gentamicin 8.5 Ceftazidime 130	16.2 random (D18)	Course already completed	17	Not done	494 (D19)	PD for 5 days	55
3	7 y	22	Pseudo Bartter syndrome	NR	Gentamicin 12 Ceftazidime 150	10 peak 2.1 trough (D2)	11	16	ATN	776 (D22)	HD for 4 days	60

*Only out of range/inappropriate gentamicin levels tabulated.
Cr, creatinine; ATN, acute tubular necrosis; NR, not recorded; PD, peritoneal dialysis; HD, haemodialysis; D, day.

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Authors' reply

Dr Biban states that we did not adequately emphasise the neurologic side effects of interferon treatment. Although it has been reported that interferon alpha has been responsible for various neurologic side effects, there are no clear data indicating the frequency of these in children. Short term interferon therapy has been safely used at our department in treating various different conditions, particularly in the complex hemangiomas for many years. No side effects of interferon therapy except mild fever, malaise, leukopenia, and elevation of liver transaminases have been observed. These were reversible by stopping therapy for a short period. In one patient who received long term interferon therapy, peripheral neuropathy developed during the treatment.

This patient was a 15 year old boy with Hodgkin's disease who received interferon as an adjuvant immunotherapy post autologous stem cell transplant. Peripheral neuropathy developed 20 months after IFN treatment.[1] A large cumulative dose combined with the prolonged treatment may have had an important role in this complication in our case. We concluded that the use of interferon in children affected by KSM or in children with various benign tumours containing vascular elements is still a good therapeutic alternative. If the duration of treatment and the cumulative doses of interferon are closely monitorised, severe neurologic side effects during IFN therapy would not be an important problem. As the use of interferons in various conditions gradually expands, the data related to the adverse neurologic side effects will increase and be better understood.

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Acute renal failure and cystic fibrosis

It is surprising that there are few reports of acute renal failure (ARF) in children with cystic fibrosis (CF) given the large number of antibiotic courses prescribed and the possibility of either direct toxicity from aminoglycosides or the occurrence of interstitial nephritis. The registry of our regional paediatric renal unit shows no cases of ARF in a CF patient between 1985 and 1998, but three cases between 1999 and 2001, all of whom had received gentamicin and ceftazidine.[1]

Over the past nine months we have been referred three additional CF patients who had been treated with a combination of gentamicin and ceftazidine/cefuroxime (table 1). The initial doses of antibiotics used to treat the patient were within UK guidelines,[2] but the gentamicin levels were raised. All six children had received a number of other medications including, in some instances, other antibiotics prior to the gentamicin and cephalosporin combination. Only one of the four biopsy specimens revealed interstitial nephritis in addition to the acute tubular necrosis (ATN) changes found in all four. All six children have made a good renal recovery with normal blood pressures and creatinine levels at three months.

A recent e-mail survey of members of the British Association for Paediatric Nephrology revealed four other cases of ARF with combination antibiotic therapy in CF patients (three of four with ceftazidine and gentamicin). The increased incidence points to the need for increased vigilance when gentamicin and cephalosporin combinations are used to treat exacerbations, particularly if there is a potentially dehydrating state or pre-existing renal anomaly. The cases have been reported to the Committee for the Safety of Medicines and we suggest a national monitoring programme should be instigated.

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Fatal iatrogenic hyponatraemia

We recently cared for a 13 month old girl admitted to hospital following a short history of diarrhoea and vomiting. Clinical examination revealed lethargy and moderate dehydration. Initial serum sodium was 137 mmol/l and she was commenced on intravenous fluids using 4% dextrose/0.18% saline.

Twelve hours after admission the child suffered a generalised tonic-clonic seizure at which time the serum sodium was found to be 120 mmol/l. Unfortunately, the child went on to have a respiratory arrest, developed fixed dilated pupils, and died despite full intensive care. An extensive postmortem examination revealed only diffuse cerebral swelling with necrosis of the cerebellar tonsils.

It is well recognised that symptomatic hyponatraemia can result in significant morbidity and mortality in previously healthy children[1 2] and adults.[3] The administration of hypotonic intravenous fluids to children can be fatal and the reasons for this have been well documented for several years. Many physiological stimuli encountered during acute illness result in the non-osmotic release of antidiuretic hormone; these include pyrexia, nausea, pain, reduced circulating volume, and the postoperative state. The administration of hypotonic intravenous fluids in

these circumstances results in the excretion of hypertonic urine, the retention of free water, and the development of hyponatraemia.[4]

Despite clear and repeated warnings over the past few years,[5–7] the routine administration of 4% dextrose/0.18% saline remains standard practice in many paediatric units. This practice is based on formulas developed for calculating maintenance fluid and electrolytes in healthy children over 40 years ago and there seems little understanding of the potential risks associated with their use during acute illness.

A global change of clinical practice is required to prevent these needless deaths. This is a challenge that the RCPCH should face up to, together with the Medicines Control Agency and the National Patient Safety Agency. A useful first step would be to label bags of 4% dextrose/0.18% saline with the warning that severe hyponatraemia may be associated with its use.

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Thyroid screening in Down's syndrome: current patterns in the UK

Children and adults with Down's syndrome are at increased risk of developing thyroid dysfunction, and screening for thyroid dysfunction is recommended as part of their health surveillance.[1] Clinical history and examination are known to be unreliable indicators of thyroid dysfunction in Down's syndrome. Venous blood for thyroid stimulating hormone (TSH) assay remains the gold standard. Capillary blood spot on filter paper TSH has been proposed as a simpler and more convenient alternative screening method for hypothyroidism in these children.[2]

To establish current screening practices, we under took a postal questionnaire of community paediatricians registered with the British Association for Community Child Health (BACCH). Community paediatricians are the group mostly likely to see children with Down's syndrome for health surveillance. Paediatricians were asked whether they routinely screened children with Down's syndrome for thyroid dysfunction. They were asked at what age of child they began screening, how often they screened, and which method they used.

The questionnaire response rate was 64% (209/325). All the paediatricians who returned completed questionnaires routinely looked after children with Down's syndrome. As expected, almost all of respondents, 93% (194/209), were screening routinely. Most paediatricians began screening before 5 years of age, and screened every two years (table 1). Venous blood TSH was the most frequently used method of screening (83%, 174/209). Only a small number have begun using capillary blood spot on filter paper TSH (7%, 15/209). A few paediatricians were relying on clinical suspicion alone. Those paediatricians not routinely screening for thyroid dysfunction, were either measuring TSH opportunistically or were undertaking biochemical screening only when symptoms or signs raised suspicion.

The Down's Syndrome Medical Interest Group (DSMIG) has recommended biochemical screening for thyroid dysfunction at least every two years after the first year of life.[1] Most paediatricians' practice is consistent with this recommendation. Capillary blood sampling has practical advantages over venous sampling, with regard to patient acceptability, particularly in adolescents with Down's syndrome and with regard to cost. There is growing evidence that capillary blood spot TSH is a reliable screening tool for thyroid dysfunction in children with Down's syndrome.[2–4] Capillary blood spot TSH may, in the future, come to replace venous TSH sampling in children with Down's syndrome.

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Changes in serum sodium levels during treatment of hyperglycaemia

Carlotti *et al*[1] state that fluid and electrolyte management might contribute to the development of cerebral oedema in hyperglycaemia. There is a simple rule of thumb, formulated by Katz, which may help calculate water and electrolyte deficits and predict the changes in sodium levels which accompany changes in glucose levels,[2] namely that a decrease of 0.29 mmol/l in serum sodium may be expected for every 1.0 mmol/l increment in serum glucose.

This may be explained as follows: hyperglycaemia causes an osmotic movement of water out of the cells, which leads to hyponatraemia by dilution. Thus, at presentation, the patient is usually severely dehydrated intracellularly. However, the serum sodium is lower than would be expected because of this dilution of the extracellular fluid. When the patient is treated with insulin, glucose enters the cells, taking water with it, leading to a relative concentration of the extracellular fluid, and thereby a rise in serum sodium. This rise may be predicted and calculated using Katz's formula.[2]

Carlotti *et al* also comment on the report of Glaser *et al* that the chance of cerebral oedema during treatment is increased in children who present with high initial serum urea levels and when there is a lack of an increase in serum sodium levels during treatment.[3] This increased risk may be explained by the fact that the urea level rises in proportion to the degree of dehydration. Urea contributes to serum osmolality and if the fall in urea is not taken into account the serum osmolality may be allowed to drop too rapidly, thereby increasing the risk of cerebral oedema. Carlotti *et al* do not take this into account in their formula for calculation of osmolality. The calculation of serum osmolality as twice the sum of sodium and potassium plus the urea and glucose levels (all in mmol/l) corresponds better with the formally measured osmolality.[4]

By treating hyperglycaemia using hypotonic solutions or glucose alone, the serum osmolality will fall rapidly and thereby increase the risk of cerebral oedema.

Serum osmolality must be monitored frequently, either by direct measurement or calculation from the sodium, potassium,

Table 1 Results of completed questionnaires (n=209)

Age screening initiated (y)	No. (%)	Screening frequency	No. (%)	Screening method	No. (%)
<5	167 (80%)	Yearly	35 (17%)	Venous TSH	174 (83%)
5–10	28 (13.5%)	Two yearly	115 (55%)	Capillary blood spot TSH	15 (7%)
>10	1 (0.5%)	Three yearly	20 (10%)	Both venous and capillary blood spot TSH	4 (2%)
No data	13 (6%)	Opportunistically	17 (8%)	Clinical history and examination only	3 (1.5%)
		Other	10 (4.5%)	No data	13 (6.5%)
		No data	12 (5.5%)		

TSH, thyroid stimulating hormone.

glucose, and urea levels. In this way, the effects of falling urea and glucose levels on the serum osmolality will be compensated to a large extent by the accompanying rise in sodium. Thus the osmolality falls slowly and in a controlled fashion at a rate of 1–2 mosmol/kg/hour thereby, reducing the risk of cerebral oedema.

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Author's reply

We thank Dr Oudesluys-Murphy for her letter in response to our article. In essence, two points were raised:

1. Can one estimate the deficits of Na^+ and water if one applies the formula proposed by Katz?[1]

This calculation makes the presumption that one can predict the change in plasma sodium concentration (P_{Na}) when water is drawn out of cells by hyperglycaemia. This assumption is not correct for a number of reasons.[2]

- Glucose must be added as a pure solute. Glucose will be retained in the ECF compartment (normal 10 L in a 50kg person with 30 L of total body water). With the net retention of 600 mmol of glucose without water in the ECF compartment, the P_{Glu} will rise by close to 57 mM if we assume that glucose distribution is only in the ECF compartment because water will shift from cells to the ECF. In more detail, the total number of osmoles in the body was 8550 milliosmoles (285×30 L) before the addition of glucose and 9150 milliosmoles after the addition of glucose (8550 + 600). Therefore the new P_{osm} will be 305 mOsm/kg H_2O (9150/30 L). The new ECF volume is equal to the total ECF osmoles (2850 + 600) divided by the new osmolality of 305 mOsm/L, or 11.3 L. Therefore 1.3 L of water will be drawn out of cells due to the high P_{Glu}. Bottom line: The new P_{Glu} is 57.5 mM, the new P_{Na} is 124 mM, and the new ECFV is 11.3 L.

- Addition of isosmotic glucose (285 mM) to raise the P_{Glu} by close to 50 mM with all the same assumptions: No water is drawn out of or enters cells because an iso-osmotic solution of glucose was added to the ECF compartment and all added glucose remains in the ECF compartment. When 2.3 L of this glucose solution is in the ECF compartment, the new P_{Glu} is 57 mM, the new P_{Na} is 114 mM because water was retained in the ECF compartment without Na^+, and the new ECF volume is 12.3 L. Bottom line: The new P_{Glu} is 57 mM, the

new P_{Na} is 114 mM, and the new ECFV is 12.3 L. Overall, because the ECF volume was expanded by different amounts in calculations A and B above yet the rise in the P_{Glu} was virtually identical, there is no constant relationship between the P_{Glu} and the ECF volume. Moreover, there was no change in the content of Na in the ECF compartment in these two examples. In contrast, patients presenting with DKA have a contracted ECF volume and a deficit of Na^+ when their P_{Glu} is 57 mM. Conclusion: If you do not know what the ECF volume is in quantitative terms, you cannot deduce the ECF Na^+ content from the P_{Glu}. Accordingly, much as we would like to agree with the suggestion of Dr Oudesluys-Murphy, the facts do not support that view.

- Potassium: Part of the deficit of K^+ reflects the shift of K^+ out of cells in a 1:1 relationship with a cation (Na^+ and H^+) of unpredictable amounts. The other major part of K^+ loss from the ICF reflects the catabolic state (primarily a loss of K^+ with organic phosphate (e.g. from RNA)). Since both of these components are not known with certainty, one cannot use the relationship described by Katz[1] to help in this context.

- Error in the assumption of Katz[1]: The volume of distribution of glucose is larger than the ECF volume even if there is a lack of insulin actions. Our reasoning is that, in cells that do not require insulin for glucose transport such as liver cells, cells of the proximal convoluted tubule, and red blood cells, the concentration of glucose is likely to be equal in their ICF and ECF compartments.[3]

2. Urea should be included in calculations of effective osmolality.

Urea is not an effective osmole across cell membranes when the change in the plasma urea concentration (P_{urea}) is not abrupt.[4] Given the time course for the fall in P_{urea} in patients with DKA, we did not include urea in our calculation of effective osmolality. Therefore we believe that it is more prudent to keep the $P_{Glu} + 2 (P_{Na} + P_K)$ relatively constant in the first 12–18 hours of therapy. A gradual decline in the effective P_{osm} should occur with time thereafter.

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Assessing immune responses to pneumococcal vaccines

The recent article and letter[1][2] discussing recommendations for use of heptavalent

pneumococcal conjugate vaccine (Prevenar) for at risk children is timely and interesting. We concur with the authors that further immunogenicity studies are necessary in various high risk groups to demonstrate the best protective schedule.

Children older than 2 years with recurrent infections and normal humoral immunity assessed by serum immunoglobulin levels and specific antibody responses to protein antigens, but repeated poor responses (less than 4-fold rise in antibody titers) to 23 valent pneumococcal polysaccharide vaccine (Pneumovax) using standard pneumovax based ELISA[3] are labelled as "specific pneumococcal polysaccharide antibody" deficiency.[4]

We looked at the immunogenicity of the heptavalent conjugate pneumococcal vaccine (Prevenar) in five children aged 4–12 years with specific polysaccharide antibody deficiency by the above definition. Blood was collected before and 4 weeks after immunisation with the heptavalent conjugate pneumococcal vaccine. Serum was analysed using the standard ELISA (using Pneumovax as the antigen)[4] and by the newer serotype specific antibody assay.

Results are shown in table 1 and 2. The standard assay showed 4-fold response in only one child. However 5/5 children showed 4-fold responses to at least four of the seven serotypes using the serotype specific antibody assay.

Protection is assumed at a serotype specific antibody level of 0.2 ug/ml or greater.[5] It is interesting to note that 4 out of 5 children had achieved such protective levels to four or more serotypes after immunisation with pneumovax as suggested by the serotype specific assay on the pre-prevnar sera.

Clinicians may be tempted to use the more easily available standard ELISA to assess responses to the conjugate vaccine in high risk children. These findings suggest that whilst assessing such responses it is important to use the serotype specific assay to get true measure of adequacy of response (Balmer P *et al*. Measurement and interpretation of pneumococcal IgG levels for clinical management. *Clin Exp Immun* (submitted)). The study also suggests that there may be a group of children in whom an immune response to Pneumovax is detectable only by the serotype specific assay and who may be labelled as specific antibody deficient by the standard assay.

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Tables 1 and 2 can be viewed on the *ADC* website (www.archdischild.com/cgi/eletters/archdischild;88/2/176#432)

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Partial splenectomy in CF patients with hypersplenism

Our recently published article on partial splenectomy in cystic fibrosis (CF) patients with hypersplenism[1] appeared with a commentary by colleagues from the Birmingham Children's Hospital.[2]

The authors of this commentary rightfully point out that liver disease in CF may have a widely varying symptomatology ranging from portal hypertension, bleeding oesophageal varices, ascites, to splenomegaly with hypersplenism. While the quoted clinical experience of 200 patients with CF liver disease might be considered as substantial, it nevertheless appears unjustified to rush from that experience to the statement that severe hypersplenism, requiring a specific surgical approach, is not a feature of the discussed disease spectrum. In the equally substantial clinical experience of our CF centre such severe hypersplenism occurred in only those three patients described in our paper. In these rare cases, however, we found the respiratory and haematological complications caused by the massively enlarged spleens to be impressive and to range from severe impairment of respiratory compliance to life threatening thrombocytopenia. It is for those rare patients that we consider the described surgical intervention to be of potential value. We strongly believe that such patients deserve a therapeutic intervention before their splenomegaly has effected a potentially irreversible deterioration of their chronic lung disease or their hypersplenism has caused a life threatening haemorrhage via thrombocytopenia or infection via leucopenia. The validity of this approach is illustrated by the three reported cases: white blood cell and platelet counts significantly and persistently improved (follow up time is now four to eight years), oesophageal varices disappeared in two and significantly regressed in one patient (reported in our paper and not omitted as postulated in the commentary), and respiratory function improved in all three patients. While the authors of this commentary state that they did not encounter "severe hypersplenism requiring such aggressive management" in their CF patients, one wonders whether early liver transplantation in patients with relatively less severe hypersplenism might not be considered as equally or even more "aggressive".[3]

The reason why white blood cell and platelet counts were not given in our paper was due to the editor's decision to shorten the manuscript.

In contrast to the authors of the commentary we see no reason to believe that liver transplantation and partial splenectomy are surgical interventions that are mutually exclusive. On one side, there are reports of excessive portal hypertension or hypersplenism necessitating splenectomy (or partial splenic embolisation) after liver transplantation,[4–7] on the other side, our surgical colleagues do not see any reason to believe that partial splenectomy actually increases the technical difficulties of a later transplant operation. Furthermore, in admittedly small reported series of partial splenectomies performed in children with a variety of diseases, no major complications have been observed.[8–10]

We agree with the authors of this commentary that liver transplantation, oesophageal band ligation, and transjugular intrahepatic portosystemic stent shunting are important therapeutic options for children with advanced CF liver disease. In contrast to them, however, we welcome partial splenectomy as an additional therapeutic strategy that particularly addresses the problem of splenomegaly and hypersplenism. Ultimately, this difference of perspectives on the same issue might relate to the almost philosophical question whether one welcomes such a new treatment strategy as a potentially promising addition to one's therapeutic quiver, or, alternatively, tends to reject such interesting new possibilities off hand.

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Partial splenectomy—worth the risk

In 1993, we published the results of our first cystic fibrosis (CF) patients who had complications of liver disease and portal hypertension (PHT), and had been operated on by partial splenectomy (PS). We also presented the results of 11 patients at the Jerusalem CF meeting in 1996. Since 1982, we have operated on and followed up 21 patients (aged 8 to 22 years). All patients had a large spleen measuring 15–28 cm in length, oesophageal varices graded 2 to 4 by endoscopy, hypersplenism with a platelet account below 50 000, and a well documented liver disease treated with UDCA.

Surgical procedure consisted of PS with conservation of the upper lobe of the spleen, terminal haemostasis, and suturation of parenchymentous vessels. The whole procedure lasts 3–4 hours. The only postoperative complications consisted of scar rupture in three cases and a painful episode of a few days in two cases. No pulmonary exacerbation occurred after surgery. A speedy normalisation of the haematological profile was observed. Normal function of the remaining upper lobe of the spleen was registered by scintiscan. An important improvement of oesophageal varices was noticed in nine cases out of 11 and a stable condition observed in two cases. The size of the remaining spleen remained stable in nine patients out of the first group who presented in 1996. No deterioration, and even some improvement of hepatic function was observed.

In conclusion, we believe that the risk of PS is worth taking since it appears to be a good option for the treatment of oesophageal varices, which is the main concern, and also might be the cure for hypersplenism. Partial splenectomy is an important alternative to all other procedures in the treatment of PHT. Moreover, it allows a delay of hepatic transplantation and it may even be avoided altogether.

We intend to present our global results in a near future.

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