# LETTER TO THE EDITOR

# Attempted Control of Serum Sodium Concentration During Liver Transplantation in Severely Hyponatraemic Patients

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**Keywords:** Hyponatraemia, liver transplant, frusemide, serum sodium, osmolality, osmotic demyelination syndrome.

Dear Editor,

Hyponatraemia (serum sodium <135 mmol/L) is very common in patients with end stage liver disease. Between 1993 and 2005, 32.4% of all liver recipients demonstrated a serum sodium concentration <134 $mmol^{-L}$ (10.5% <130 $mmol^{-L}$) across the UK and Ireland [1]. Peri-operative [Na] is an important and reliable indicator of severity of illness as patients demonstrate a higher calculated MELD† (22.3+/-9.3). A MELD-Na score which can be as much as 13 points higher has also been proposed [2]. Hyponatraemia is also a good predictor of waiting list mortality, longer surgical times, higher intra-operative transfusion requirements, poor post-operative outcomes and reduced 90 day survival - 84% versus 95% [1-4]. Major morbidity includes sepsis and multi-organ failure (64.2%), neurological problems (10-30%) and graft failure (8.6%) [1]. Central pontine myelinolysis (CPM) is the most serious neurological sequela and tends to develop up to the 11th post-operative day [1, 4-6]. It is caused by the osmotic demyelination of neurons as intra-operative serum sodium concentration and osmolality change rapidly [7,8]. Symptoms include lethargy, seizure, coma, permanent brain damage, brainstem herniation, respiratory arrest and death [4].

To date, the anaesthetic management of intra-operative [Na] in this patient group remains an on-going challenge with a consensus for how to manage serum sodium in the acute surgical setting still lacking. Yet, it is clearly needed when considering the sodium content of commonly used infusion fluids; Blood and blood products (140-165 $mmol^{-L}$), crystalloids and colloids (30-154 $mmol^{-L}$) and human albumin solutions (140-160 $mmol^{-L}$) – all of which are widely administered during liver-related surgery. In 2005, 84% of all consultant transplant anaesthetists across the UK and Ireland responded to a survey of their intra-operative practice during orthotopic liver transplantation (Appendix I). Results showed that as first-line colloid, 72% routinely used gelofusine, 23% used a starch-based product and 3% used albumin. As first-line crystalloid, 31% used Hartmann's solution, 21% used 5% dextrose, 10% used 0.9% saline and 7% used 4% dextrose-0.18% saline. 84% routinely used a saline-based cell salvage system, and for transducer lines 74% used saline and 26% used heparinised saline as primer fluid. Other drugs commonly used during OLT also have a high osmolality - 8.4% sodium bicarbonate (2000 $mOsmol^{-L}$) and 20% mannitol (1050 $mOsmol^{-L}$).

Current recommendations are for slow, ward-based correction using saline therapy, haemodialysis/filtration over 50-60 hours with low sodium substitution fluids and drug therapies such as oral urea, lithium or the newer parenteral vasopressin II antagonists [9,10] administered pre-operatively and then avoiding VVB intra-operatively (which requires high volume priming with sodium-rich fluids) [5]. Urea and lithium are unpleasant oral preparations which produce unpredictable effects and vasopressin II antagonists [lixivitan, tolvaptan, satavaptan) promote *aquaresis* - electrolyte-free water excretion, promoting sodium and potassium preservation; clearly undesirable when sodium administration is unavoidable and potassium administration is likely as it is during OLT. Tromethamine (THAM) is also being investigated as a drug with potential use in intra-operative sodium control in the USA‡. However, *pre*-operative correction of sodium appears only to improve 21-day mortality with no change in other outcomes [8]. It also risks surgical delay and deterioration of encephalopathy.

Attempting to control or avoid wide fluctuations intra-operatively is the way forward. Haemofiltration is available intra-operatively, but may require additional large bore cannulation, circuit anticoagulation and may not achieve sodium control adequate enough to keep pace with high volume transfusions.

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†MELD - model for end-stage liver disease – scoring system (range 6-40) calculated using creatinine, bilirubin and INR and initially used to predict 3 months survival post-TIPSS. Now, reliably predicts need for liver transplantation >24.

‡*Hyponatraemia in patients with Chronic Liver Disease Undergoing Liver Transplantation*, David R. Wright, Oral Presentation for 'Hyponatraemia' at the ILTS at the ASA Peri-operative Care in Liver Transplantation one day symposium, 2007, San Francisco, California, USA.

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The following two cases describe the acute management of two patients from different centres undergoing OLT with severe hyponatraemia. The aim was to induce a supranormal diuresis (>1mL$^{-kg-hr}$) and natriuresis (urinary [Na] >50mmol$^{-L}$) using intravenous frusemide, whilst judiciously meeting the patient's transfusion needs (sodium load) and other electrolyte requirements. The discussion then examines the data obtained and whether the results could form the basis for development of a reproducible management strategy. It raises pertinent questions and postulates how further investigation could genuinely expand the currently available data pool, providing a valuable contribution to current practice that could positively affect patient outcomes.

## Case Report

Patient A - (*centre 1*[¶]) – was a 124kg, 58 year old female with non-alcoholic steatohepatitis (NASH) who developed hepatorenal syndrome (HRS), resistant ascites and intermittent encephalopathy grade 1-2 with flap. She had undergone unsuccessful TIPSS and had developed chronic hyponatraemia which became increasingly severe over the previous 4 months. She had a standard course of treatment for HRS (20% HAS, terlipressin and octreotide) which improved, but did not normalise, her renal function. She was responding to treatment for clostridium difficile and right leg cellulitis.

On the day of surgery, she had a calculated MELD of 26 and renal impairment - serum sodium was 116 mmol$^{-L}$, creatinine 132 μmol$^{-L}$ and urea 19.1 mmol$^{-L}$. Albumin was 30g$^{-dl}$ and bilirubin was 107 μmol$^{-L}$. She was anaemic (Hb 7.1g$^{-dl}$) and coagulopathic (platelet count 47x10$^9$, PT 19.0s, APTT 92.0s). Following standard induction and venous cannulation, she had raised pulmonary artery pressures (66/28 mmHg), which was thought to have a large hypervolemic component (PCWP 18 mmHg) and was partially controlled with high end-tidal isoflurane (1.2 MAC) and GTN infusion. During 6 hours and 30 minutes' anaesthetic time, she underwent 4 hours of surgery and 3500ml ascites was measured at incision. She received approximately 9000ml fluid as 6000ml blood/products, 2000ml 0.9% saline and 1000ml cell saver return (Fig. **1**). She received aprotinin as a 2 MU bolus followed by 0.5MU$^{-hr}$ infusion until the anhepatic phase began. Veno-venous bypass of 2 hours' duration was used and the anhepatic time was 35 minutes. The final estimated blood loss was 4000ml. Intra-operatively, she received a total of 120mg frusemide as three equal boluses (40mg at 0.5, 1.5 and 6 hours) and was monitored *via* laboratory and arterial blood gas assay according to centre protocol. She produced a total of 1045ml urine – a mean of 174.2 ml$^{-hr}$ or 1.45ml$^{-kg-hr}$ - with a well controlled [Na] range of 116-123mmol$^{-L}$ which tended to follow an upward trend (Table **1**). All other measured electrolytes were well-controlled ($Mg^{2+}$ 0.77-0.99 mmol$^{-L}$ and $iCa^{2+}$ 1.0-1.26 mmol$^{-L}$). Boluses of magnesium sulphate [total 2gm at 2.5 hours) and calcium chloride [titrated infusion of 1.15gm plus 5.5gm bolused in 0.5gm aliquots) were administered as needed. Haemodynamically, she was stable with minimal support.

Patient A was extubated at 24 hours and received a further 72 hours of HDU care. Her [creatinine] rose to a peak of 187 μmol$^{-L}$ on day 3 and then steadily improved. Though still hyponatraemic, it was less severe - [Na] 127 mmol$^{-L}$ and 124 mmol$^{-L}$ on day 1 and 2, respectively. She was discharged to the ward on day 4 and her [Na] fluctuated between 128-132 μmol$^{-L}$ until day 20 when it normalized along with her [creatinine]. Full anti-rejection therapy was delayed, but she did not require renal replacement therapy and maintained a good urine output, despite a recurrence of clostridium dificile infection between day 9 and 14. Her encephalopathy did not recur and she did not develop surgical or overt neurological morbidity. She was discharged from the hospital on day 22 and remains alive and well over 2 years on.

Patient B (*centre 2*[*]) – was a 70kg, 64 year old man with hepatitis C cirrhosis. He also had resistant ascites, persistent hyponatraemia over the previous 3 months and encephalopathy grade 1-2 with intermittent flap. He had suffered intermittent encephalopathy since 2001, partially attributed to dementia and was found to have atrophic changes and small vessel disease on pre-operative MRI brain scan.

On the day of surgery, patient B had a calculated MELD of 16, serum sodium of 124 mmol$^{-L}$, urea 9.1 mmol$^{-L}$, creatinine 76 μmol$^{-L}$. Albumin was 35g$^{-dl}$ and bilirubin 82 μmol$^{-L}$. He was anaemic (Hb 6.7 g$^{-dl}$) and coagulopathic (platelet count 29x10$^9$, PT 16.6s, APTT 53.0s). During 10 hours of anaesthesia, he underwent 7 hours and 45 minutes of surgery and had 5100ml ascites measured at incision. He received approximately 17,500ml fluid as 7300ml blood/products, 3100ml crystalloid ($^{1/5}$dextrose-saline 500ml, 0.9% saline 1600ml, Hartmann's solution 1000ml), 5000ml gelofusine® and 1200ml cell saver return (Fig. **2**). He received a bolus of 30mg$^{-kg}$ tranexamic acid, followed by an infusion of 10mg$^{-kg-hr}$ until 2 hours post-reperfusion. Anhepatic time was 1 hour and 17 minutes, during which, he received 1g$^{-kg}$ 20% mannitol (at 4 hours). Veno-venous bypass was not used. The final estimated blood loss was 11,700ml. He received 40mg frusemide as two equal boluses (20mg at 0.5 and 3 hours) and was monitored *via* arterial blood gases alone according to centre protocol. He produced a total of 2120 ml urine – a mean of 235.6 ml$^{-hr}$ or 3.65 ml$^{-kg-hr}$ - with a well-controlled [Na] range of 121-127mmol$^{-L}$, which tended to fluctuate (Table **2**). All other measured electrolytes were well-controlled ($iCa^{2+}$ 0.97 – 1.55 mmol$^{-L}$). Boluses of magnesium sulphate (total 2gm at 4 hours) and calcium chloride (titrated infusion of 8.3gm plus 4gm bolused in 0.5gm aliquots) were administered as needed. Haemodynamic stability was maintained and assisted with intermittent metaraminol, but he was transferred to ICU free of any circulatory support.

Patient B was extubated at 25 hours. His hyponatraemia persisted but improved and normalised on day 7 (129-135 mmol$^{-L}$). His creatinine fluctuated (97-150 μmol$^{-L}$), but normalized on day 8, having peaked on day 3 and 6. He was discharged to prolonged HDU care on day 2 until day 20 due to pneumonia and a period of tacrolimus toxicity. His encephalopathy worsened only with his temporarily increased oxygen requirements, but he did not develop major surgical or neurological morbidity. He was discharged from the hospital on day 35 and is feeling 'back to his old self' almost 8 months on.

[¶]Centre 1 is St. Vincent's University Hospital, Elm Park, Dublin 4, Ireland

[*]Centre 2 is Addenbrooke's Hospital, Cambridge University Hospitals NHS Trust, Hills Road, Cambridge, CB2 0QQ, UK.

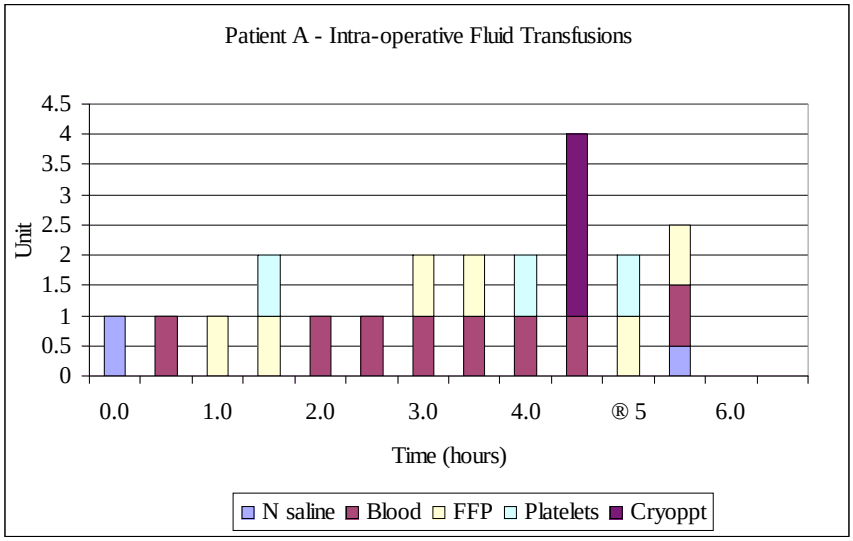


**Fig. (1).** Patient A – Intra-operative Fluid Transfusions
1 unit = ~300ml blood/blood product, 1000ml crystalloid, 500ml gelofusine

**Table 1. Patient A – Intra-Operative Trends in Electrolytes, Urine Output, Base Excess and Central Venous Pressure**

| Time (hr) | 0.0 | 1.0 | 2.0 | 3.0 | 4.0 | 5.0 | 6.0 |
|---|---|---|---|---|---|---|---|
| **Event** | IVI | | KtS | V+ | (An) | ® | EoS |
| **Sodium** /mmol$^{-L}$ | 116 | 118 | 117 | 120 | 122 | 123 | 124 |
| **Potassium** /mmol$^{-L}$ | 4.6 | 4.9 | 4.8 | 4.8 | 4.6 | 5.1 | 4.6 |
| **Creatinine** /µmol$^{-L}$ | 132 | 125 | 124 | 120 | 119 | 122 | N/A |
| **UOP** /ml | - | 200 | 170 | 170 | 270 | 125 | 110 |
| **Base Excess** | - | -11.2 | -10.1 | -10.4 | -9.4 | -10.9 | -8.5 |
| **CVP** /mmHg | 20 | 19 | 14 | 15 | 9 | 14 | 15 |

IVI intravenous induction, KtS knife to skin, V+ start of VVB, (An) start of anhepatic time, ® Reperfusion, EoS end of surgery

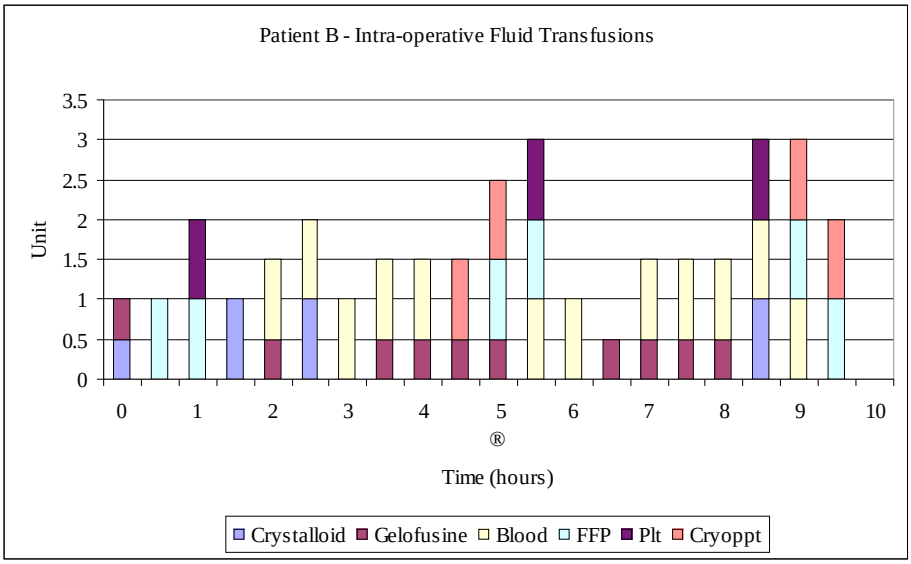


**Fig. (2).** Patient B – Intra-operative Fluid Transfusion
1 unit = ~300ml blood/blood product, 1000ml crystalloid, 500ml gelofusine.

**Table 2. Patient B – Intra-Operative Trends in Electrolytes, Urine Output, Base Excess and Central Venous Pressure**

| Time (hr) | 0.0 | 1.0 | 2.0 | 3.0 | 4.0 | 5.0 | 6.0 | 7.0 | 8.0 | 9.0 |
|---|---|---|---|---|---|---|---|---|---|---|
| **Event** | IVI | KtS | | | | | [An) | ® | | EoS |
| **Sodium** /mmol$^{-L}$ | 124 | 123 | 126 | 123 | 125 | 121 | 123 | 124 | 125 | 126 |
| **Potassium** /mmol$^{-L}$ | 3.4 | 3.2 | 3.21 | 3.59 | 3.09 | 3.36 | 4.46 | 3.92 | 3.52 | 3.1 |
| **Creatinine** /μmol$^{-L}$ | 76 | N/A | N/A | N/A | N/A | N/A | N/A | N/A | N/A | N/A |
| **UOP** /ml | - | 280 | 340 | 120 | 280 | 200 | 240 | 190 | 230 | 240 |
| **Base Excess** | | +3.2 | +0.3 | -1.5 | -3.11 | -4.3 | -8.0 | -5.8 | -5.2 | -5.8 |
| **CVP** /mmHg | - | 13 | 10 | 7 | 8 | 8 | 14 | 12 | 11 | 14 |

IVI intravenous induction, KtS knife to skin, V+ start of VVB, (An) start of anhepatic time, ® Reperfusion, EoS end of surgery.
N/A - not available - due to limits set by unit protocol at centre 2

Sodium bicarbonate was not used in either case and both patients received intra-operative antibiotics and methyl prednisolone anti-rejection therapy as per centre protocol.

## DISCUSSION

It has been established that the act of infusing sodium will cause natriuresis in normal kidneys [9] and it is known that the ability of the kidneys to excrete sodium can be impaired by factors such as hepatic dysfunction and liver transplant surgery [6]. This acute management theory proposed utilising residual renal function to produce a frusemide-induced natriuresis against active electrolyte management using judicious, flexible fluid administration and intravenous electrolyte supplementation.

Despite patient A's renal impairment, she produced a supranormal diuresis in response to 0.99 mg$^{-kg}$ total IV frusemide. Her fluid therapy was a little more cautious in light of her impairment and this may have been the cause of her upward trend in serum sodium. She demonstrated an overall rise of 7 mmol$^{-L}$over 6.5 hours and only once exceeded a 1-2 mmol$^{-L}$ rise over any single hour. Her CVP and haemodynamic stability suggest generally adequate systemic filling and her serum creatinine fell (132-122 μmol$^{-L}$). Her post-operative creatinine subsequently rose by 41%, but she did not require post-operative renal support.

Patient B had a higher starting serum sodium and normal renal function and produced a supranormal diuresis in response to 0.61 mg$^{-kg}$ total IV frusemide. His hourly output of 208.9 ml$^{-hr}$ was comparable to patient A's 174.2 ml$^{-hr}$, but based on his weight, his diuresis was doubled. His fluid was more actively managed in light of his normal renal function, utilising a wider range of crystalloid and colloid as dictated by the electrolytes on ABG. This may have been reflected in the tighter, but more variable, control of his intra-operative sodium. He demonstrated the smaller overall rise in serum sodium of 5 mmol$^{-L}$, but the range fluctuated by 1-4 mmol$^{-L}$. His CVP also suggested adequate systemic filling, but the need for metaraminol support may reflect end-organ perfusion issues. Intra-operative serum creatinine was not available, but his immediate post-operative creatinine had risen (97 μ mol$^{-L}$), suggesting an overall intra-operative rise and it continued to rise – finally by 97%. However, he also did not require renal support and continued to produce good volumes of urine.

Frusemide-induced natriuresis is not a new concept. The property is widely documented and accepted as pharmacological fact and it has been recommended as a treatment in ward-based hyponatremic patients [9,10] but it remains untested in the acute intra-operative setting. It is known to change normal urine composition by decreasing pH (6.0:6.4), increasing concentrations of sodium (140:50 mmol$^{-L}$), potassium (25:15 mmol$^{-L}$) and chloride (155:60 mmol$^{-L}$) and by increasing volume 8:1 ml$^{-min}$ [11].

Intravenous frusemide can be administered in boluses of 20-80mg (0.1-1mg$^{-kg}$) for acute conditions such as peripheral or pulmonary oedema. It has an onset time of 2 minutes - vascular effects commencing before renal effects - and a duration of action of 2 hours. As a high ceiling loop diuretic, frusemide reduces the osmotic pull around the thin descending loop of Henlé, independent of acid-base status. This inhibits the re-absorption of water, sodium, chloride, magnesium, calcium, hydrogen ions, ammonia and bicarbonate from the thick ascending loop of Henlé and has the overall effect of causing increased excretion of all ions as well as water and can produce a hypotonic urine. It is documented, along with thiazide diuretics, as being amongst the commonest drug-related cause of hyponatraemia in in-patients and out-patients [9,10,12]. This management strategy, therefore, attempted to find a reasonably adaptable balance between sodium load (fluid transfusion) versus excretion (natriuresis) using a temporally flexible agent easily accessible and commonly used by transplant anaesthetists - the 2005 survey (Appendix I) revealed that 33% consultant anaesthetists routinely (9%) or sometimes (26%) used diuretics during liver transplantation. Of these, 49% preferred frusemide to mannitol (37%), bumetanide (5%) or dopamine (2%). 72% routinely used calcium chloride bolus or infusion and 31% used magnesium sulphate bolus. Intravenous magnesium and calcium electrolyte supplementation were required in both cases, not but for potassium. However, potassium was administered as a constituent of the infusions of Hartmann's solution, warmed blood and blood products and was well-controlled in both cases.

Neither patient A or B developed ototoxicity in the post-operative period, suggesting that the diuretics were administered in an acceptable dose and fashion.

Certain assumptions have been made based on the subjective observation of these patients. There is much missing data that could more thoroughly test the hypotheses and fully answer the questions cast by their management.

NHSBT – the main source of transplant-related trends and statistics - has no information on intra-operative trends of sodium or osmolality, thus data collection is genuinely needed. It would not be difficult to implement - 86% of transplant anaesthetists routinely repeated blood tests intra-operatively; 77% hourly and 9% in a phasic manner (Appendix I). Urine output is routinely measured, thus paired serial serum and urinary sodium, creatinine and osmolality measurement during liver transplantation could demonstrate trends in relation to sodium load (fluids), routine drug interventions and surgery (including VVB). This would establish a significant database, allowing investigation of frusemide and its effects to follow along the same lines and determine its relevance for future practice.

The unrealised potential here is to discover whether anaesthetists can manipulate the fractional excretion of sodium by active intra-operative management and attempt to determine the extent to which intravenous frusemide in combination with a timely choice of fluid might play a pharmacological role and impact on that management. We might also learn to more easily identify any patient sub-group *NOT* susceptible to wide fluctuations of serum sodium, osmolality or to the development of CPM intra-operatively, and we might discover whether any non-oliguric renal dysfunction post-operatively are as expected following liver transplant in any patient or whether it may be exacerbated after natriuretic frusemide therapy and is acceptable within the limits set by the known consequences of the surgery.

Neither patient A or B exhibited serious neurological problems, but there is a 10-30% incidence of CPM on autopsy of liver transplant recipients, which indicates that sub-clinical occurrence cannot be ruled out [1]. Therefore, if unlimited resources were available, pre and post-transplant PET +/- MRI brain scans could assess and compare cerebral oedema and myelin injury and further add to the knowledge pool – both as baseline data is collected and subsequently with frusemide interventions.

Finally, we might be able to establish within what limits intra-operative control of serum sodium can be *allowed* to fluctuate. It has been recommended that correction of hyponatraemia should be a slow process of not more than 12-15 $mmol^{-L}$ per 24 hours [5] or by no more than 0.5 $mmol^{-hr}$ [10] - even less in particularly high-risk patients [9], yet both patients A and B exceeded these limits intra-operatively. Both were maintained on a volatile anaesthetic plus oxygen in air – patient A on isoflurane and patient B on desflurane. Though a slight digression, one cannot help but ponder whether a decreased susceptibility or increased tolerance might be an intra-operative phenomenon where neuronal membrane activity, possibly altered by anaesthesia or any number of factors, affords some protection from transmembraneous solute and ion exchanges and further, add to what is already postulated about how anaesthetics work.

With a co-ordinated effort between centres, the gauntlet may be taken up to broaden these simple ideas, which appear to lead to exciting possibilities within the practice of anaesthesia.

## CONCLUSION

It would be beneficial to attempt to reach a consensus amongst transplant anaesthetists for the acute intra-operative management of hyponatremic patients undergoing liver transplantation. In doing so, some much-needed data on the intra-operative handling of sodium and osmolality could have wide-reaching positive consequences and encourage further formal consideration of frusemide as a potential answer or alternative to a significant management challenge. Debate, discussion and ultimately multi-centre study within the transplant anaesthesia community and dedication of appropriate resources would provide an invaluable contribution to the on-going analysis of this patient sub-group and to the wider transplant and anaesthetic community.

## ACKNOWLEDGEMENT

I would like to thank Mr. P. Pocock at NHSBT for the information he provided.

## ABBREVIATIONS

| | | |
|---|---|---|
| ABG | = | Arterial blood gas |
| BE | = | Base excess |
| CPM | = | Central pontine myelinolysis |
| [creatinine] | = | Creatinine concentration |
| CVP | = | Central venous pressure |
| GTN | = | Glyceryl trinitrate |
| Hb | = | Haemoglobin |
| HDU | = | High dependency unit |
| MAC | = | Minimum alveolar concentration |
| MELD | = | Model for end stage liver disease |
| [Na] | = | Sodium concentration |
| NHSBT | = | National Health Service Blood and Transplant [formerly UK Transplant) |
| OLT | = | Orthotopic liver transplantation |
| PCWP | = | Pulmonary capillary wedge pressure |
| TIPSS | = | Transjugular intrahepatic portosystemic stent |
| VVB | = | Venovenous bypass |

## APPENDIX I

Conference poster; Proceedings of the Joint International Congress of ILTS, ELITA and LICAGE, 2006 May 3-6, Milan, Italy; #250275; The 60th New York State Society Post Graduate Assembly, 2006 December 8-12, New York, USA #P-9159. Reprinted with permission.

# CURRENT INTRA-OPERATIVE PRACTICE AMONGST UK AND IRISH ANAESTHETISTS DURING LIVER TRANSPLANTATION

**Authors:** DA D'Oyley MB FFARCSI, JF Boylan MB FRCPC.
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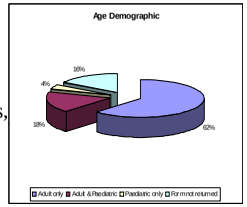
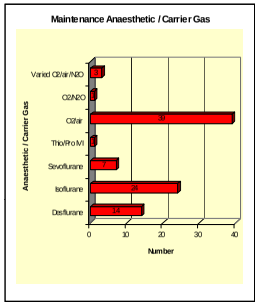
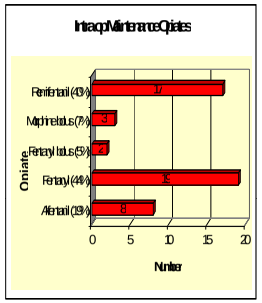
Twelve years after Carton's definitive review of peri-operative practice, we were interested to see how an increase in specialist centre numbers and experience might cause a variation in intra-operative anaesthetic practice.
**Method:** We conducted a postal survey of the 51 consultants practising liver transplant anaesthesia at the eight centres across the UK and Ireland between June and August, 2005.

We achieved an 84% response Rate within the imposed 3 month deadline period.
Questions covered demographics, Peri-operative drugs, vascular access, fluid management, blood and product transfusion, physiological monitoring and post op care.

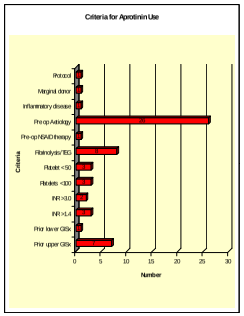


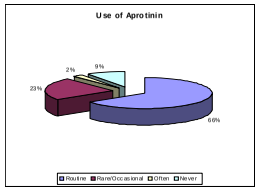

**Induction** was usually intravenous using propofol, fentanyl and atracurium. Few used thiopentone, rocuronium or suxamethonium, suggesting rare use of the rapid sequence technique.



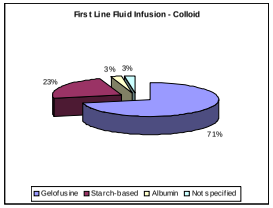

Routine intra-operative maintenance regimens varied between centres. Diuretics are not commonly used, but when indicated, frusemide is the commonest choice. Calcium chloride is commonly used and the vasopressor of choice is noradrenaline. N-acetyl cysteine is rarely used and only in fulminants.

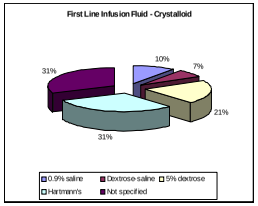


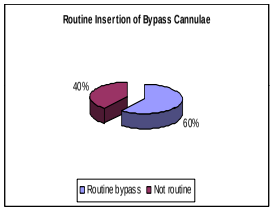

Though most practitioners Use aprotinin routinely, there is a wide range of indications. 70% Felt that a 1-5 year interval for repeat use was safe, whilst 9% would never repeat its use.

86% routinely repeat blood tests intra-operatively – 77% hourly and 9% in relation to the phase of surgery.
ABGs, Hb, glucose and electrolytes are the most commonly repeated tests. 67% use and repeat a TEG. One centre *never* uses TEG and 48-58% repeat various other clotting studies.



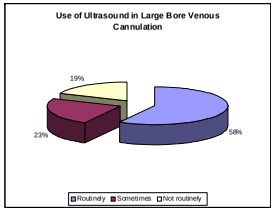



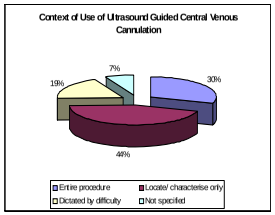

There was a wide variation and overlap in choice of fluid intra-operatively. Plain 0.9% saline was the primer fluid of choice for transducer lines, though 26% respondents still use heparinised 0.9% saline.



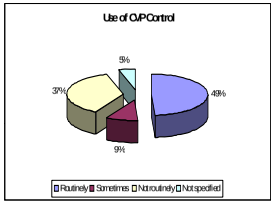

Routine insertion of bypass lines is a centre-specific practice.
3 centres do not do so.
Of the remaining 5 centres, 2 use the internal jugular approach only and 3 use the IJV with femoral vein. The subclavian vein is rarely used.

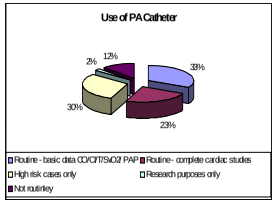
If anaesthetic bypass is *not* established, surgical access is only Routinely established 14% of the time and usually not 49% of the time



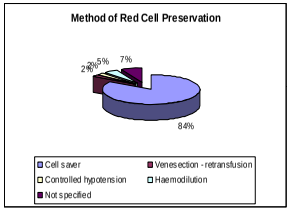



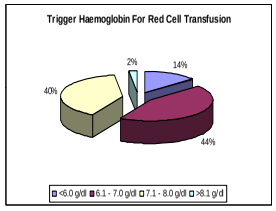

14% of respondents do not routinely site standard central vein catheters and 22% with respect to pulmonary artery catheters..
93% routinely have rapid infusion devices available. 14% at 2 centres use a centre-modified type, otherwise SIMMS Level 1, Haemonetics and FMS Belmont are the models of choice.
56% site unilateral radial arterial lines, 37% site bilateral and 7% at a single centre site left radial with right femoral.



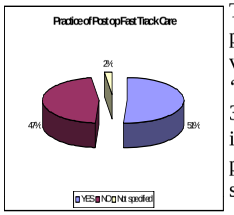




During active central venous pressure control, 42% keep to a range of 5-10mmHg, 23% allow the patient's own and 16% will vary the CVP according To the phase of surgery..
72% routinely use volume control and 23% use pressure control as the ventilation mode of choice. Pressure control is favoured in paediatric practice. 47% routinely add PEEP with 37% adding it as needed.
81% routinely use warm air convection for temperature homeostasis.
93% respondents do not routinely monitor intra-operative awareness.
Of the 5% who do so routinely, BIS was the universal method of choice.




91% cited clinical picture pf haemorrhage, 70% cited the TEG and 63% cited clotting studies/platelet count as triggers for administering blood and blood products.




Two centres unanimously report a fast track policy. Individuals from all other centres gave varying answers. Of the 51% who answered 'yes', 23%use intra-operative desflurane and 37% use remifentanil. 95% post-transplant care is delivered on an ICU. At one centre, 75% paediatric cases are extubated at the end of surgery and HDU care is delivered on the ICU.

Post op analgesia is opiate-based with 38% using infusion and 37% using PCA and infusion.
**CONCLUSION;** Practice varies and can be centre-specific. There should be more of a consensus on safety issues, such as aprotinin, Bypass cannulation, oesophageal doppler and also on fast track post-op care. Would there be a place for a Europe-wide audit database?

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