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Solicitor to the Inquiry
into Hyponatraemia deaths
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Dear Ms Dillon

Thank you for your letter of 9 June 2010 requesting some further responses to specific issues raised by the panel of experts at your enquiry. I have attempted to respond in detail as requested.

ADAM STRAIN: HYPONATRAEMIA RELATED DEATH

The donor was a 16 year old, heart beating donor with normal renal function at the time of donation. The donor passed 200mls of urine in the hour prior to donation. The cold storage was approximately 34 hours. Questions have been raised about the viability of this kidney prior to implantation. There is very little doubt in my mind about its viability. A 16 year old, heart beating donor, is an extremely favourable donor with a very high chance of success, despite prolonged cold storage time. Average cold storage times in the UK are about 20 hours, but from my experience a young donor can tolerate cold storage times considerably in excess of this period, and I have personally been involved in transplanting organs from older donors with cold storage times greater than 48 hours with a great deal of success. It is impossible for anyone to be certain that these organs were viable at the time of implantation, but the likelihood is that they were. The information that the paired kidney also did not function, does not prove that this kidney was not viable. The observation that the kidney appeared pink and well perfused, after the initial clamp release confirms in my view that it was a viable organ. The fact that this kidney did not look quite so well perfused in the later stages of the operation, can be explained either by the fact that the patients blood pressure was low, or there was some technical error in the arterial or venous anastomosis which caused an impairment in the circulation of the kidney which was not obvious immediately. The further alternative is that the apparent reduction in circulation, was due to the kidney developing acute tubular necrosis. With a cold storage time of approximately 34 hours, acute tubular necrosis is very likely. The incidence of acute tubular necrosis, described from heart beating donors in the UK, is in the order of 20 to 30%. This means that the kidney does not function immediately and function may be delayed for periods of up to 6 weeks or more. The older the donor, the more likely there is to be tubular necrosis. The longer the cold storage time, the more likely there is to be acute tubular necrosis. Acute tubular necrosis is usually recoverable.

I do not believe it is reasonable for the pathologist to say that this kidney was infarcted before or during the operation. It is clear from the description by the surgeon that the kidney appeared viable. It is likely that the kidney thrombosed in the early post operative period, once the wound was closed. This is a not uncommon phenomenon and because of this, in my unit at Guy's, we always scan the kidney in recovery so that we can return to theatre should there be evidence of poor perfusion of the kidney. I cannot be certain that there was not a technical error in the performance of the arterial or venous anastomosis, or in the positioning of the kidney before closure. It is possible that the infarction process began towards the end of the operation, but it is more likely that it has occurred after the operation had been completed.

The implication of the apparent poor perfusion during the operation is that there would have been a discussion between the surgeons and anaesthetists about central venous pressure in order to provide the best circulation to the kidney. It is very common for the surgeon to request more fluid to be given to the patient to promote better circulation. The use of fluid and inotropes to improve the renal circulation depends on clinical judgement at the time and will also depend upon the central venous pressure and blood pressure measurements, the cardiac fitness of the patient and the serum electrolytes, particularly the serum sodium.

It is my strong view, that the low sodium in this child was related to the use of high dose Dioralate pre-operatively and the use of high dose 5th normal intravenous saline during the early stages of the operation. The first measured serum sodium was 123 and the result towards the end of the operation showed serum sodium of 119. These are **dangerously low levels** and seem to indicate that the function or non function of the renal transplant at this stage was an irrelevancy.

It is interesting that Adam had a history of occasional hyponatraemia. This merely reinforces my view that pre-operative sodium was absolutely vital to inform anaesthetists and surgeons as to the safety of subsequent intravenous electrolyte administration. Once the 5th normal saline had been administered intravenously, and the sodium found to be a low as 123, it is almost certain that the brain swelling had commenced and was almost certainly irreversible.

In summary I think it is more instructive to concentrate on the administration of intragastric and intravenous fluids and monitoring of serum electrolytes pre and peri operatively rather than focus on the perceived viability of the kidney.

I am happy to provide any further opinions if required.

With best wishes

Yours sincerely

Mr Geoff Koffman FRCS
Consultant Transplant Surgeon , Clinical Director of Renal, Transplantation and Urology