SUPPLEMENTARY REPORT TO THE INQUIRY RE: LUCY CRAWFORD BY DR RODERICK MACFAUL

Date : 24 June 2013

In response to the report I provided to the Inquiry on 25 April 2013 a number of points have been raised by organisations or individuals mentioned and further documentation has been provided to me through the Inquiry. This supplementary report addresses these and includes proposed alterations to parts of my report.

Firstly I wish to clarify that in when I have used using the term *"it is not evident"* throughout my report I mean it was not evident to me from documentation provided at the time of producing my report.

1] <u>RBHSC Clinical Audit : Papers provided on behalf of Dr Robert Taylor.</u>

In my comment on RBHSC audit processes In Para 718 I stated

> **"……**……..When examining causes of death it is important to record if any issues relating to care were raised which should be logged and any necessary action taken. The purpose of this would be to identify a need for a change in practice immediately or, to log similar incidents as a proportion of similar cases. The latter is a form of monitoring the effect of change in practice with a view to either improving implementation of previous guidelines or changing guidance or protocols for care. This forms part of the "audit cycle" which I explain in Annex F. It is not evident how this process was managed in RBHSC and in the Clinical Directorate which had a responsibility to supervise audit processes**.** …….. It may be helpful to seek audit reports from the Trust as examples used by trainees in assembling a portfolio or by consultants when completing their annual continuing professional development (CPD) portfolios.

And In Para 721…

> "It is not evident whether an annual (or other frequency) audit report was made to the clinical directorate nor whether any reports were sent to the clinical director or whether the minutes were shared with him or her or with any oversight Audit committee in the Trust."

In an enclosure to correspondence 324-006-001 dated 12th of June 2013 such evidence has now been provided.

In summary it is now apparent to me that there was an overall Trust clinical audit committee structure and process to which monthly minutes were submitted by the directorates. And there was no querying of the established policy whereby the mortality section of the meeting was unminuted. Documents submitted include:

- The *Directory of National Audits 1999* which shows that RBHSC was involved in a number of national audits including for paediatrics: asthma admissions, end- stage renal failure, solid tumour and leukaemia, growth hormone audit and diabetic audit, organ transplant, BPSU and neonatal surgical audit as well as NCPOD, CESDI and newborn bloodspot audit. 324-006a-(027-042).

- A *Worksheet of Audit Activity* dated 15th of March 2000 has been provided to inform the Inquiry of the range of clinical audits undertaken throughout the Trust and in the paediatric directorate which outlines the activity of Dr Taylor as paediatric audit facilitator. 324-006a-(003-018).

- *The Organisational Plan for the Development of Clinical Audit 1999-2000 (Action Plan/Progress Report).* This demonstrates a structured evaluation of the audit activity in the Royal group of hospitals with documentation of a forward programme for the following year. For paediatrics the list includes : case note review, re-audit of paediatric junior medical staff, cystic fibrosis, use of care pathway for first febrile convulsion , care pathway in fractured radius and child protection education for staff.324-006a-(019-026)

- *Monthly Reports on Medical/Clinical Audit Activities February 2001 324-006a-(003-018)*

- A letter of 14th of February 2000 from Dr Mulholland, Director of Clinical Audit the Royal hospitals to Dr R Taylor which states "*more often than not the morbidity, mortality section will involve all disciplines and many of the audit reports should have a bearing on problems which all of us face from time to time. In addition the cross fertilisation of ideas from those who were not on precisely the same track as ourselves can be very fruitful…….. The wide-ranging multi-professional discussion was very worthwhile*." Dr Taylor's personal contribution in paediatric audit and beyond was commended and the letter states his *"leadership has produced a very high standard of activity".* (324-006a-002)

- *Implementing Clinical and Social care Governance February 2000*. Which shows the management structure in the Royal Hospitals and the place of the Clinical Audit Committee.

Comment: the information provided on behalf Dr Taylor is most helpful and shows a good quality of audit activity within the Trust coordinated by him. It is appropriate for me also to acknowledge here that Dr Taylor played a significant part in the recognition in Northern Ireland of hyponatraemia risk in children. In the light of the documentation received I wish to modify sections of my report to read as follows.

> **Para xvii** : The audit process for examination of Lucy's death in Belfast appears to have a number of shortcomings and were not in keeping with RBHSC practice at the time. The mortality meeting was regarded as a separate process from clinical audit generally and the latter appears to be comprehensive and up to standard although IT support for the process was limited. The process for reporting audit activity and it's monitoring within the Trust appears satisfactory. On the other hand, in my opinion, the mortality meetings

were not adequately minuted, so that significant outcomes of discussion were not recorded and there does not appear to be a process of aggregating and analysing trends on issues raised during discussion of deaths. It appears that the mortality meeting for Lucy was not attended by the intensivists or consultant paediatric neurologist involved in her care nor the pathologist.

Para 40: The audit processes in place in Belfast for consideration of deaths appear to have a number of shortcomings. The mortality meetings were, in my opinion, not adequately minuted although the anonymity of such discussions in respect of individual comments or patient identification was in keeping with practice for the time. The shortcomings reduce the opportunity to identify significant outcomes of discussion and thus limit any process of aggregating and analysing trends. The IT support for the process was limited. For example, it appears that Dr Taylor was not made aware of the full functionality of the Patient Administration(PAS)/Hospital Information system in respect of its recording of clinical conditions and diagnoses although this should have been known to the Trust administrative staff supporting audit and other processes. In my annex F on Audit I provide a review of standards and practice in clinical audit in the NHS at the time and detailed commentary on RBHSC audit in Para 680-735.

Para 721: Save for the comments I make on the mortality section, in my opinion, the structure and process for annual and regular monthly reporting of audit activity was up to standard for the time within RBHSC.

Comment: In my opinion, the mortality section of the audit meeting, although considered separately as an entity by RBHSC, was a form of audit and from Dr Mulholland's letter it appears its value as a multidisciplinary forum was clearly established and valued within the Trust. It was good practice to carry out mortality reviews within the audit meetings but it would also have been good practice, within the principles of the guidance applying at the time as a form of audit, to have made a log of the cause of each death and any clinical issues which had been raised (or not) in respect of each death (for example whether the clinical management been in keeping with current practice or guidance) in order to aggregate any repeating matters arising or to recognise trends or increased incidence with a view to addressing deficiencies (as it appears was the case in management of meningococcal disease referred to in my report para 699).

2] <u>RBHSC Clinical Audit : PICU database information provided</u>

In my report Para 713 I commented :

It would be helpful to know whether the codes which appeared on the PICU coding form were entered into the PICU audit database and, if they were, why it was not possible for Dr Taylor to identify Lucy as one of the children with hyponatraemia when enquiring of the system in 2001.

In correspondence from DLS dated 5 May 2013 [319-067f] information from the PICU database is now provided showing that for Lucy hyponatraemia is entered in a field [319-067e-003] although it was not included in the Royal's PAS clinical coding (see Para 711 of my report) although it had been for Claire Roberts.

3] Dr Kelly.

In correspondence to the Inquiry dated 23rd of May 2013 it was requested that my attention be drawn to the decision of the General Medical Council in respect of Dr Kelly in October 2012 was that a complaint is no longer outstanding and that the GMC concluded that Dr Kelly did not fall below the standard expected of a reasonably competent medical director when the time of the incident is considered. The GMC was informed by the opinion of an experienced medical director from another Trust and on behalf of Dr Kelly it has been pointed out that I have no experience of ever having held such a position.

Comment : While it is the case that I have not ever been medical director in a Trust I have worked in senior medical management within a medium-size Trust at the next layer down within the medical management structure as clinical coordinator for all non-surgical specialties as in my cv. And, in the early 1980s to late 1990s, before the post of medical director was established for the Trust, I acted as the medical member of the three-person management team of a large district general hospital and, as chair of the hospital medical committee representing all consultants within the Trust, interacted with management on their behalf for a number of years before the medical director post was established. These activities entailed frequent informal and regular formal meetings with the chief executive and other senior managers of the Trust on management and professional issues.

4] Points raised by the Royal Hospital respecting possible changes in serum sodium related to the IV normal saline infused in Lucy in Erne hospital

In a communication to the Inquiry of 27 May 2013 the Royal Hospital has asked me to respond to 2 points in my report. This comment aims to do so but first I refer to the relevant limitations of my knowledge and expertise as set out in Para 651 of my report

> *First I wish to state that I may not have understood Prof Kirkham's position correctly and am only basing any comment on this exchange of views in the hearing as they bear on Lucy. These are significant qualifications because I am neither an academic nor a clinical scientist nor am I familiar with the research papers in this area other than those relevant to clinical practice in the late 90s and early 2000s and with publications relating to hyponatraemia associated with use of hypotonic intravenous fluid in children in clinical studies in the 2000s to which I refer.*

TRUST CONCERNS

Point 1] The Trust wrote as follows:

The extent to which the administration of normal saline would have raised the sodium levels from a level significantly lower than 127 mmol/l prior to that result being obtained.

At **paragraph 541** of his report Dr.MacFaul indicates the following

" Comment : Dr Crean did not take account of the effect of a rapid infusion of a large volume of normal saline on the blood sodium which might conceal a much lower level at the time of Lucy's collapse and indicates that Lucy's death was unexplained in April 2000. But the second blood sample was obtained during the respiratory resuscitation and the case notes do not identify the sample time nor who obtained it or whether it was taken before or following the start of the normal saline infusion. But Dr O' Donohoe reported to the Trust review that it was taken by him and thus after the saline was running in his report to the review."

The Trust has significant concerns that this suggestion (that there was as much lower sodium level not identified as a result of saline infusion) is not correct. There are two aspects to this question:

The exact volume of 0.9% sodium chloride received by Lucy prior to the blood sample being taken is unclear. However, this is likely to have been no more than 250 mls (absolute maximum 500 mls).

The effect of **1000 mls** of infused fluid on serum sodium can be calculated. The most commonly used formula is the Adrogue´-Madias, published in the New England Journal of Medicine in 2000 (Table 2, page 1855: attached):

Change in Na concentration = (infusate Na level - serum Na level) / (total body water + 1)

In this case the formula can be applied as follows:

Change in Na concentration = (154 - 125) / (5.0 + 1) = 4.83 mmol/l

(This assumes that the initial serum sodium was 125 mmol/l and total body water was 5.0 l. These are estimates, based on the information that is available, but the Trust considers that they are reasonable estimates.)

On this basis **250 mls** of 0.9% sodium chloride would have raised serum sodium by **just over 1.2 mmol/l**, and **500 mls** by **approximately 2.5 mmol/l**. In other words, infusion

> of 0.9% sodium chloride would have had only minor impact on the serum sodium level prior to the 127 mmol/l result being obtained. Would it be possible for Dr MacFaul to produce a calculation to show the impact of 250 or 500 mls of 0.9% sodium chloride on Lucy's serum sodium level?
>
> There is recent evidence that this formula underestimates the increase in sodium in some cases, particularly when the initial serum sodium is <120, but this would not have been known prior to 2007.

MY RESPONSE TO POINT 1]

As a general paediatrician I make no claim to any expertise in how to quantify electrolyte changes resulting from infusions of normal saline as calculated on behalf of the Royal Trust. I would defer to clinical chemistry or intensive care specialists in this respect as it is beyond my expertise. Thus I can make no comment on this calculation other than as follows. It does appear the Trust acknowledge the possibility that a volume of 250-500ml normal saline infused rapidly could have led to a lower level of blood sodium being reached in Lucy than the one measured in her at Erne but from the calculation above the Trust's position is that this might have been a marginal change.

My intention in referring to this possibility was to draw attention to it and to the point that it did not appear that Dr Crean had taken this into account when commenting on the fluid regime used in Lucy nor that *at the time* consideration was given in RBHSC to the possible effect on the blood sodium result of the volume of normal saline given to Lucy which was unusually high and beyond her requirements. The clinicians at RBHSC did not know the precise timing of the blood sample and its relationship to the volume of saline infused as this detail is not in the clinical notes which they had received. If the possible presence of a lower level than measured (even if marginal) had been considered this could have led to greater attention being paid to the potential contribution of the changes in blood sodium and /or the volumes of fluid infused in Lucy in Erne on the development of brain oedema.

Trust Point 2] The Trust wrote as follows:

> At paragraph 67 of his report Dr.MacFaul states "It is probable that Lucy developed SIADH….". If he is correct in this assumption, then the question arises of whether an infusion of 0.9% sodium chloride would have had any impact on the serum sodium. It is now generally accepted that in SIADH the infusion of 0.9% saline has little or no impact on serum sodium (see highlighting on attached references; Verbalis et al Hyponatremia Treatment Guidelines 2007, pS12; Zietse et al, 2010). Dr MacFaul should be asked to comment the impact of 0.9% sodium chloride in serum sodium in SIADH.

MY RESPONSE TO POINT 2]

I did not consider whether the presence of SIADH might influence any possible change in the serum sodium resulting from the infused high volume of normal saline before the second blood test. This point lies outside my expertise and I am unable to comment further on the effect of IV saline in SIADH other than the papers to which I referred in my report as below.

In my opinion it can be argued that Lucy was a candidate for SIADH although there were other potential causes in her for development of hyponatraemia : water overload from hypotonic IV fluid given in inappropriately large volume and, loss of sodium in the diarrhoea which developed during her IV therapy. The argument in support of this opinion is that mild (and usually well tolerated) hyponatraemia is not uncommon in acute childhood illness even in those disorders in which, before serum sodium is measured, no excess of water has been given or electrolyte loss occurred: such as febrile convulsion, pneumonia or acute neurological disease. A number of sources suggest that this can be a result of SIADH in some children and I accept this view and have usually interpreted the finding in my patients in this context. In my report (paras 643 & 644) I referred to publications which report both low serum sodium measurements and elevated ADH to non physiological levels in children with gastroenteritis. I also refer to studies reporting that IV saline when given can produce a fall in ADH levels.

Consultant Paediatrician

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