REGULATION 28: REPORT TO PREVENT FUTURE DEATHS (1)

	REGULATION 28 REPORT TO PREVENT FUTURE DEATHS **THIS REPORT IS BEING SENT TO:** **1. [redacted], Secretary of State for Health and Social Care, House of Commons, London SW1A 0AA** **2. [redacted], NHS Regional Director for London, NHS England London, 133-135 Wellington Road, London, SE1 8UG** **3. [redacted], Interim Chief Executive, Care Quality Commission, 2 Redman Place London E20 1JQ** **4. [redacted], Chief Executive, Medicines, and Healthcare Products Regulatory Agency (MHRA), 10 South Colonnade, Canary Wharf, London E14 4PU**
1	**CORONER** I am Dr Julian Morris, senior coroner, for the coroner area of London Inner South
2	**CORONER'S LEGAL POWERS** I make this report under paragraph 7, Schedule 5, of the Coroners and Justice Act 2009 and Regulations 28 and 29 of the Coroners (Investigations) Regulations 2013.
3	**INVESTIGATION and INQUEST** In 2014 an investigation was commenced into the death of Yousef Al-Kharboush (born 23rd May 2014, died 1st June 2014, aged 8 days), Oscar Barker (born 27 May 2014, died 29 June 2014, aged 1 Month) and Aviva Otte (born 10 October 2013, died 2 January 2014, aged 2 months). The investigation concluded at the end of the inquest on 23 October 2023. The conclusions of all 3 inquests were a narrative with each of the causes of death being: 1. Aviva Otte: Narrative Conclusion Aviva was the second twin, her twin sister surviving to this day, born extremely preterm at 24+2 as a result of spontaneous onset of preterm labour at 02.03 hrs on 10.10.2013. She was described as being born in moderate condition with HR >60 and good colour, but with no spontaneous breathing, that initially being assisted by positive pressure breaths and then intubation by 22 minutes of age. She was treated with surfactant and anti-biotics. Conditions for which she received treatment from then until 31.12.2013 included: Patent ductus ateriosis, high glucose levels, a large (right sided) intraventricular haemorrhage (which in the opinion of the expert, would not have caused/ contributed to death), intestinal distension and perforation, (resulting in a laparotomy 30.10.2013 showing ileal perforation secondary to necrotising enterocolitis) with resultant stoma formation. Parenteral nutrition restarted on 6.12.2013. Remaining stable until the day of planned surgery for closure of stomas – 31.12.2013 (day 83 of life) At that operation, the surgeons found multiple adhesions, which were carefully divided and succeeded in re-aligning the two segments of bowel despite the size and operational difficulties. The plan, as far as ant-biotic cover was concerned, was to continue with iv anti-biotics for 2 days post-operatively. She was settled in/around 08.45 on the morning of 1.1.2014. By approximately 10 am, she had developed irritability, which was initially interpreted as pain, but Aviva did not settle. Further investigations revealing a developing metabolic acidosis and acute anaemia raising the possibility of blood loss from somewhere. In additional the previous irritability was considered to be increasing with the development and signs of an abnormal brain function;

bedside ultrasound revealed a catastrophic intra-cranial haemorrhage or series of haemorrhages. Despite medical supportive efforts she continued to deteriorate and sadly died the following day, 2.1.2014.

Cause of death:
I (a) Intracranial Haemorrhage (b) Bacillus cereus (Bc.38) (c) Extreme prematurity at 24+2 weeks gestation and extreme low birth weight II Necrotising Enterocolitis Conclusion

2. Oscar Barker: Narrative Conclusion

Oscar was born at the Rosie Hospital, Addenbrooke's, Cambridge on 27 May 2014 at 28 weeks gestation by C-section. He was one of twins, his antenatal period being complicated by Intra uterine growth retardation and poor foetal doppler measures, suggesting that he was compromised as a foetus chronically and was noted to have a VSD antenatally. Intubated at birth, given surfactant, treated for low glucose and had a long line inserted, but by 13 hours was extubated and receiving CPAP together with empirical anti-biotics given his earlier breathing problems.
At day 3 of life, he developed a slightly raised CRP which increased the concern about possible infection. Blood tests taken earlier on had also shown low platelet and white cell counts which, although common and as a result of prematurity, could also have been linked to the signs of developing infection; as such he received additional anti-biotic treatment. Echocardiography also revealing than in addition to his VSD, Oscar was also suffering from congenital malformation of the great vessels which would have required surgery at some point in the future but treated at the time by medical infusion to maintain foetal circulation.
On day 7 (3.6.2014) he developed increasing amounts of desaturation and apnoeas and was found to have developed a spontaneous perforation of his bowel and taken to theatre for its repair and stoma formation. Post-operatively, he was critically ill receiving medications through his long line to support his circulation, platelet and red blood cell transfusions.
On day 8 he developed pulmonary haemorrhage, received a further transfusion and an additional anti-biotic, then renal impairment (ultrasound scan was unable to locate a left sided kidney at this stage but it was not known whether it had ever been present).
By day 20 (16 June), following the ceasing of anti-biotics 2 days earlier there was a progressive deterioration with increased oxygen requirements, bradycardias and abdominal distension, he was re-intubated. He was very sick at this stage with multi-organ failure. Given the septic diagnosis, his long line in-situ was removed and replaced the following day.
Oscar had blood cultures taken on 16 and 18 June, together with the tip of the long line being sent off on 16 June. The former were negative, the latter was confirmed as having Bacillus, later identified as Bc.44. Upon commencement of the septic screen, he was also started on anti-biotics and an anti-fungal agent. Despite this and additional medical management, Oscar continued to deteriorate with excess fluid and deteriorating renal function.
By day 33 he was really unwell: unstable, acidotic and with severe reduction in urine output with a resultant metabolic acidosis from, not only the infection but also the renal failure. The medical team feared Oscar would not survive and he sadly died on that day – 29 June 2014.

Cause of death:
I (a) Multi Organ Failure (b) Bacillus cereus (Bc.44) sepsis

	3. Yousef Al-Kharboush: Narrative Conclusion Yousef was described as being born moderately premature at 32 weeks (with his twin) on 23 May 2014 but with very low birth weight, an extra factor mitigating against health, respiratory distress syndrome, patent ductus arteriosus and jaundice. He spent most of the first week being fairly unremarkable until the morning of 30 May when he started to show signs of infection (unstable temp, blood sugars were high, CRP was high) with an ultrasound showing the presence of quite severe abnormalities - indicative of brain abscesses. He had been given total parenteral nutrition on 27th and 28th. At the time of his hand over on 30th, he had an infection of unknown cause for which investigations had been commenced and for which he had been started on empirical anti-biotics. Over the night he required increasing levels of support (transfusion and platelets). The following morning, the microbiology team confirmed the positive growth of Bacillus (24-hrs after being taken) and his anti-biotics were changed accordingly. By this time, he was showing signs of multi-organ derangement; he was a very sick and unstable, small baby. His downward trend continued with a re-addressing of care aims on the Sunday: he subsequently died at 18.00 that Sunday evening, 1 June 2014. Cause of death: I (a) Sepsis – Bacillus cereus (Bc.44) (b) IUGR II Twin Pregnancy
4	**CIRCUMSTANCES OF THE DEATH** Aviva's death (January 2014) was in hospital where she had received TPN provided and compounded by the NHS establishment under a section 10 exemption. That TPN had, on balance, been contaminated by Bacillus cereus (subsequently identified as type BC.38). The Trust undertook a root cause analysis together with involving the UKHSA and its own infection and microbiological teams, but no definitive source for the outbreak was found. In June 2014 Oscar Barker and Yousef Al-Kharboush received TPN, compounded by a commercial provider, which it turned out was also contaminated by Bacillus cereus (subsequently typed as Bc.44). The compounder having positive finger dab testing for the Bacillus within its laboratory/environmental testing. This outbreak also affected other babies in other Trusts. Bacillus cereus is resistant (because it is spore forming) to the spray and wipe cleaning methods used (with alcohol) and sporocides are required to decontaminate the outside of, for example, ampoules containing one of the constituents. This was information and a conclusion that the Trust had reached in early 2014 and therefore prior to the outbreak in May/June 2014. It had not passed on those findings either within other section 10 units compounding TPN or the wider market. Subsequently, the MHRA brought in further advice for the use of sporocides in 2015.
5	**CORONER'S CONCERNS** During the course of the inquest the evidence revealed matters giving rise to concern. In my opinion there is a risk that future deaths could occur unless action is taken. In the circumstances it is my statutory duty to report to you.

	The **MATTERS OF CONCERN** are as follows. – (1) There is no requirement for a section 10 exempt entity to report any of its findings to the MHRA or indeed to other Trusts or the industry in general if an adverse event occurs. (2) The current reporting structures (for a section 10 entity) involve reporting to NHSE and the CQC but the threshold or necessity for such reporting appears unclear and, in essence, up to the Trust. (3) There may be times when section 10 entities reach conclusions which would assist the wider industry and help to assist both other Trusts and commercial organisations in assessing their own risks and improving the provision of highly specific medication to a group of vulnerable patients. (4) the same may also be true of commercial organisations but they have the power of the MHRA controlling and effecting recalls and actions and the wider dissemination of information.
6	**ACTION SHOULD BE TAKEN** In my opinion action should be taken to prevent future deaths and I believe you have the power to take such action.
7	**YOUR RESPONSE** You are under a duty to respond to this report within 56 days of the date of this report, namely by Wednesday 8th January. I, the coroner, may extend the period. Your response must contain details of action taken or proposed to be taken, setting out the timetable for action. Otherwise, you must explain why no action is proposed.
8	**COPIES and PUBLICATION** I have sent a copy of my report to the Chief Coroner and to the following Interested Persons: - Yousef Al-Kharboush family Oscar Barker's mother: [redacted] Aviva Otte: [redacted] ITH Pharma: [redacted] & [redacted] of Hickman & Rose GSTT: [redacted] of DAC Beachcroft Cambridge University Hospital: [redacted] of Kennedy's Law MHRA; [redacted] of Government Legal UKHSA/ PHE: [redacted] of Kennedy's Law Fresenius Kabi: [redacted] of DWF Law [and to the LOCAL SAFEGUARDING BOARD (where the deceased was under 18)]. I have also sent it to who may find it useful or of interest. I am also under a duty to send the Chief Coroner a copy of your response. The Chief Coroner may publish either or both in a complete or redacted or summary form. He may send a copy of this report to any person who he believes may find it useful or of interest. You may make representations to me, the coroner, at the time of your response, about the release or the publication of your response by the Chief Coroner.
9	**[DATE]** **[SIGNED BY CORONER]** **15th November 2024** **Senior Coroner Dr Julian Morris**