Findings of Fact

Dr. Abu Rahman, retired consultant geriatrician, was born on the 25th of December 1935. So, by November 2024, the period of which this investigation is concerned, he was aged 88 years. Like any octogenarian, he had a medical history and this included end-stage renal failure and Type II Diabetes Mellitus.

According to whose written evidence I considered carefully, he had been recently discharged from hospital, having been an inpatient treated for hospital acquired pneumonia.

On the 7th of November 2024, Dr Rahman was involved in a minor road traffic accident. The police attended and he declined medical attention on a number of occasions. Attending officers very sensibly decided that they would wait with Dr. Rahman until the arrival of a taxi, which was to take him home. This statement of says as follows: I held him up by his arms, and he had a bit of support to walk with him, and walked him slowly over to the car. At this time, I noticed how unsteady he was on his feet when the distance between the car and the wall was no more than 2 meters. So I held him up as much as possible and got him to the car door. I then reached down to the door to sit him in, and as I had stood up from doing this, I can see he's begun to stumble and fall backwards. Where I then tried to stop his fall and catch him, which was of no use as I couldn't catch him in time'

Dr. Rahman immediately identified that he had broken his hip. He was subsequently admitted to Royal Free. Upon admission, his creatinine levels were 417, which was almost identical to a previous reading of 419 a month previously, in October 2024. This is significant because it shows that the renal impairment which later became a feature of his admission had not yet commenced.

The next day, 8th November, he underwent an uncomplicated left cemented hemiarthroplasty from which he initially appeared to be recovering well. He was, in the words of his daughter from whom I heard evidence, "in great spirits". I shall refer to another medically qualified Rahman doctor, as "SR" to distinguish her from her father.

Of the course of the next few days his condition remained stable and on 9th November he mobilized with a walking frame, progress with which he was very pleased one day post-operatively.

On 10th November Dr Rahman suffered from a hallucination which SR felt was secondary to the administration and accumulation of opioids; her father had supposedly suffered a similar reaction

in 2017 in the context of bowel surgery. She asked for no more opioids to be prescribed, although she accepted in evidence that at this stage her father still had capacity. Despite being medically qualified, she did not feel that she was "heard" by treating staff, in this regard.

On 11th and 12th November Dr Rahman is still described in the medical notes as "stable" but SR described in some detail how, as matters unfolded, "each day things became gradually worse".

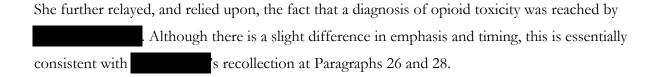
The last dose of oxycodone was given on 11th November at 1004.

On 12th November Dr Rahman was "getting confused" and SR describes being "certain" that she witnessed her father aspirate after being fed by an HCA. This too is potentially important given what later transpired and the possibility of aspiration pneumonia being implicated in Dr Rahman's death.

By 13th November, whilst his observations were stable, he was described as sleepy. His creatinine was 431, indicating that his kidney function was slightly worse. His CRP, which is a marker of infection, was 288.9. As remarks, it's difficult to determine the importance of that in the context of post-operative care, but it is nevertheless worthy of note, given what later transpired, and the possibility of hospital acquired pneumonia. A chest X-ray that day demonstrated patchy consolidation in keeping with *aspiration* pneumonia.

The 13th was the first time the putative diagnosis of "hypoactive delirium" was mooted. SR, who as I say is medically qualified, but does not give evidence in her capacity as a doctor, disputes this diagnosis and attributes the drowsiness to the continuing effect of opioids.

SR candidly accepted that she did not have the expertise to comment on or second-guess
's opinion that the recorded dosages (which were not in dispute) were "small, judicious
and reasonable". To that extent was not critical of
possible that any toxicity arose through no fault of and and indeed my attention was
drawn to medical literature to the effect that just such a phenomenon has been noted in post
operative patients in renal failure (Conway et al, 2006). Interestingly Naloxone – a therapy to
reverse opioid toxicity - was given on this date. Also of interest is that SR's husband,
, who was present on this date, is recorded in
Rahman as "cognitively sharp, but unable to stay awake for long periods". There was therefore
no suggestion that he was "not himself".



On 14th SR recalls seeing myoclonic jerks (a sign of opioid toxicity) as well as hearing explaining the "classical signs of opioid poisoning" (or words to that effect) to one of his students. Again, Naloxone was given.

SR's recollection is that in response to Naloxone, Dr Rahman's condition improved. She recounts that by 16th November (a date not covered in statement, because he was not working that day) he was "over the worst" and "recovering from surgery".

Her evidence is that a decision was unilaterally taken by a junior doctor to stop Naloxone, and then re-prescribed on 18th. Nevertheless, her evidence was that there were often logistical difficulties, with treating staff frequently running out of Naloxone and having to visit the pharmacy to obtain more. This happened on a number of occasions, meaning that there were "hours and hours" when Dr Rahman did not have any Naloxone.

It is common ground that Dr Rahman then deteriorated over the course of 19th and 20th November. His kidney function on the 19th of November had deteriorated further with a creatinine of 613. SR described how he was "re-aspirating each time he lost consciousness".

A chest X-ray on the 20th November, however, showed increased ground glass changes with multifocal nodular air space opacities within the left lung and the right mid to lower zone in keeping with underlying *infective* process.

He was reviewed by and was found to have severe metabolic acidosis, secondary to renal failure. agreed with the family that the patient was for palliative care. Dr Rahman sadly passed away, and he was confirmed deceased at 1639.

Dr Noimark produced a proposed MCCD of:

1a Hospital-acquired pneumonia

1b Fractured Neck of Femur

1c Traumatic Fall

2 End stage renal failure, and Type II Diabetes Mellitus.

SR maintains that the pneumonia was aspiration pneumonia, as opposed to hospital-acquired/infective. There is radiological support for both propositions. It is difficult to distinguish the two, as himself acknowledges, and in my judgment I (a) do not need to do so to fulfil the statutory purpose of an inquest and (b) in any event lack the evidence to do so and cannot indulge in speculation.

I consider that I am required, however, to resolve the issue of whether or not Dr Rahman suffered opioid toxicity (a) at all and (b) as opposed to hypoactive delirium.

In this regard I find in favour of SR that Dr Rahman indeed suffer from opioid toxicity, and that he did not suffer from hypoactive delirium. I do so for the following reasons:

- (1) Naloxone was prescribed, which is drug whose sole purpose is to reverse opioid toxicity.
- (2) Specifically Naloxone was prescribed by that he diagnosed opioid toxicity and was pointing out its classical features to his student. I find that the latter detail also has the 'ring of truth' to it, and indeed there is and could be no proper suggestion that SR is anything other than a truthful witness doing her best to assist the court.
- (3) The presence of myoclonic jerks.
- (4) At the onset on the so-called hypoactive delirium, Dr Rahman was described as "cognitively sharp, but unable to stay awake for long periods". There was therefore no suggestion that he was "not himself". This fits better with a picture of opioid toxicity.
- (5) No plausible cause for the hypoactive delirium has been proposed, especially in circumstances where Dr Rahman initially appeared to be recovering well from surgery.

For the avoidance of any doubt, in making this finding I do not intend any criticism of

As to whether the opioid toxicity was implicated in the death, I remind myself of R (Tainton) v HM Coroner for Preston and West Lancashire [2016] EWHC 1396(Admin)where it was said that for

causation of death to be established, the threshold is "whether on the balance of probabilities, the event or conduct more than minimally, negligibly or trivially contributed to the death" [para.41]. The relevant event "must make an actual and material contribution to the death of the deceased" [para.62].

I note that Dr Rahman's condition had, on SR's evidence, significantly improved by the 16th November to the extent that he was said to be recovering well. This was five days after his last dose, and following the administration of Naloxone, which continued albeit intermittently. I also note that I do not have any medical or expert evidence to the effect that opioid toxicity was implicated, save for serious save for serious supplemental statement that "opioids may have contributed to drowsiness and increased risk of pneumonia".

Against that background, and given the breadth of Dr Rahman's co-morbidities, I find that I lack the evidence to make this positive finding.

I therefore accept so proposed MCCD, save for the words "hospital acquired" in 1a:

As to the issue of PFD, I have considered both Conway et al and taken judicial notice of the British Renal Society's *Kidney Patient Safety Committee: Safe prescribing of opiates in kidney disease* which describes how:

15% of the population has chronic kidney disease and acute kidney injury is seen in up to 20% patients who are admitted to hospital for emergency care. Pain affects more than 50% of patients with advanced chronic kidney disease, with more than half describing this as moderate to severe. Despite its high prevalence, the recognition of pain and its management in patients with renal impairment is complicated and poorly understood. Prescribers' awareness of the choice of opioids and methods of assessment of renal impairment shows great variability and often there is limited knowledge in care settings where specialist renal input is not available

This very much tallies with the oral evidence of SR' about a lack of recognition and thus, whilst the matter is finely poised I am on balance persuaded to return a PFD Report. The Report will deal with the issue of (a) raising awareness of opioid toxicity in patients with renal impairment and (b) the administrative delays in securing medication from the pharmacy. A copy will be provided to interested persons.

In summary:

- (1) I lack the evidence to determine whether the pneumonia was Hospital-Acquired or Aspiration-based.
- (2) I do however find that there was opioid toxicity and no hypoactive delirium;
- (3) I am not in a position to find that this caused or contributed to the death;
- (4) I am issuing a PFD Report.