The Sheku Bayoh Public Inquiry

Witness Statement

Dr Martin Clark

Provided by witness
on Thursday 17 February 2022

Witness Details

1. My full name is Dr Martin [redacted] Clark. My date of birth is in 1972. I work at [redacted]. My contact details are known to the Inquiry.

Qualifications and experience

2. I qualified as a doctor in MBChB BMSC (Hons) in 1996 at the University of [redacted].

3. My qualifications are a Fellow Royal College of Anaesthetists 2001 UK, Foundation Fellow Faculty Intensive Care Medicine UK, Diploma Intensive Care medicine 2005 UK and Dual CCT Anaesthesia and Intensive Care Medicine 2006 UK.

Statement to PIRC 3 May 2015

4. I have read the PIRC-00103 statement. I think I was called at about 7:30 as opposed to 8:30 and I was first on call for anaesthesia, as I think the shift

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finished at 08:00 and I think this was the case in 2015 as well but I cannot be sure. Otherwise I agree the statement is accurate.

5. I cannot recollect for the anaesthesia shift. At the arrest from the notes Dr David Hall (daytime shift anaesthetist, who took over airway / anaesthetic role from me when he arrived), Surinder Panpher EM consusltant (in charge overall), Dr R Anderson who I presume is the A+E reg who was running the arrest but I cannot be certain of this.

6. Recollections are quite fragmented and not continuous. I only remember certain events during arrest. Recollection of event was definitely better when I gave my statement to PIRC. I have been shown a handwritten copy of my PIRC statement and can confirm my signature is at the bottom of each page.

7. Things I recall: Deciding to intubate due to leaking LMA providing suboptimal ventilation. Deciding to do inline stabilisation due to history of struggle. Thinking Mr Bayoh looked a fit individual (prior to the events of the day) who probably took good care of himself and worked out as he had good musculature and thinking "why has your heart stopped and why is it not starting"?

8. Intubation and presence of CO2 on capnograph, also asking another Dr to check air entry was present and equal as I was busy either holding the tube / tying the tube in place or bagging Mr Bayoh. Recall ventilator struggling to cope with thumper compressions at one point and changing over to manual ventilation. Recall discussions re should we do bilateral needle. Compressions with Surinder and A+E Reg and we decided against it.

9. Thinking story of behaviour pre arrest, persistent cardiac arrest and wildly abnormal arterial blood gases suggested drugs as a likely contributing factor, possibly a serotonergic type reaction plus pulmonary oedema / heart failure.

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10. Recall policeman in normal clothing on his phone all the time and pacing around. Don't recall discussion around stopping CPR. Rest is a blur.

Sheku Bayoh's arrival at hospital

11. I confirm I made and signed a note relating to Sheku Bayoh (PIRC-01069) and that it is my signature on page 14. My note is a description of my actions with regard to Mr Bayoh's care.

12. A+E resus bay. Bay in A+E resus. Has a patient trolley, resus and airway trolley and some kit on back wall. Curtains can be pulled round the area. Waterproof flooring.

13. Patient arrived by Ambulance service from notes.

14. I did not know him, did not recognise him. Can't recall any obvious injuries but I note from the A+E report he had been hit on head. Good musculature, no real body fat, looked like he worked out. Airway leak around LMA.

15. Some police in A+E but not near resus bay, unsure if any in uniform, unsure of the number, do recall one man in normal clothing on his phone and looking stressed.

Assessment and treatment of Sheku Bayoh

16. I was called down to A+E to help with airway as (I think but cannot be definite) A+E said they were struggling with it. Think arrest had been going on for some time (10mins maybe longer before they called?) before I was called but cannot be sure of this. Main role was airway and ventilation, left running of arrest to A+E staff, as arrest progressed at some point another anaesthetist (I presume Dr Hall but cannot be sure) took over the anaesthetic role. I presume at this point was when I scanned the chest and heart and got

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involved with discussions around continuing resus and other management decisions but cannot be sure.

17. Unsure of information I was given but I must have been informed of history of potential trauma/ struggle as I decided we needed to do inline stabilisation of C-spine for intubation and place patient in a hard collar. Presume I was given information by A+E registrar as this would normally be the person to give me the patient's history.

Restraints

18. I am not aware of patient restraints.

19. I can't recall Mr Bayoh being handcuffed. If he had been, it can cause difficulty with iv access and arterial lines being sited as the handcuffs get in the way.

Respiratory arrest and cardiac arrest

20. In a respiratory arrest breathing stops, in a cardiac arrest circulation ceases. You can have one without the other but generally respiratory will eventually lead to cardiac arrest as the heart becomes hypoxic and cardiac arrest will lead to respiration ceasing due to hypoxia of the respiratory centre of the brain.

21. Airway obstruction (soft tissues, blood, mucus, vomitus, foreign body, vocal cord spasm or oedema, supraglottic oedema/ inflammation/infection, tumour, trauma) decreased respiratory effort (drugs such as opiates, and sedatives such as barbitautes and/or alcohol, severe metabolic derangement or severe low bloodflow states can also depress respiratory effort due to CNS depression) respiratory muscle weakness (respiratory muscle fatigue due to prolonged hyperventilation and or hypoxaemia, neuromuscular causes

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includes cervical spinal cord injury as well as others that would not be relevant here.

22. Signs: absent or severely decreased respiratory effort / chest movement. Treatment is to breath for the patient, so obtain a patent airway and bag the patient.

23. You can virtually always breath for the patient and usually that improves matters but it does depend on what the cause is. For example severe low cardiac output states will be partially helped by adequate ventilation but the basic problem will still be present.

24. I wasn't there at the point of Mr Bayoh's respiratory arrest so I can't comment further.

25. Cardiac arrests result from toxicological: sodium channel blockers, beta blockers, ca channel blockers, sympathomimetic effects may be caused by cocaine / amphetamines, neuromuscular effects from serotonin syndrome may be caused by cocaine, MDMA plus SSRI. Ischaemic causes: acute coronary syndromes, myocardial infarction, Primary cardiac causes: cardiomyopathy, congenital heart disease, myocarditis, Electrocution.

26. Reversible causes: Hypovolaemia, Hypoxia, hypothermia, electrolyte abnormalities hypo/hyperkalaemia, hypomagnesaemia. Tension, Tamponade, toxins (see above) thrombus (see above MI / ACS) signs/ symptoms: loss of consciousness and palpable pulses, followed shortly by loss of respiratory activity.

27. Some are recognised as reversible causes, specifically hypovolaemia, hypothermia, hyperkalaemia, hypoxia, Tension pneumothorax, cardiac tamponade, thrombosis (MI) and toxins (drugs). Some drugs may be waited out by cardio pulmonary bypass if that is available in your centre. This is not available in Kirkcaldy.

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28. I was not present when Mr Bayoh first went into cardiac arrest so can't comment further.

29. For respiratory arrest the treatment is to obtain an airway, ventilate the patient with 100% oxygen and rapidly assess the patient in a ABCDE manner and to treat reversible causes. If the patient progresses to cardiac arrest the treatment is as per the ALS protocol. The guidelines state if you are unsure if a cardiac output is present then start CPR. Therefore if you have a respiratory arrest and are unsure whether you also have a cardiac arrest you should start CPR until you are confident you have a cardiac output. The risks of this are potentially rib fractures vs not resuscitating an arrested heart.

Specific tests and treatment

30. I carried out an ultrasound examination. Cannot pinpoint exact time(s) of exams but from A+E notes it seems I did an US exam 3 times. My reasons for performing the scans were to rule out a cardiac tamponade and a tension / normal pneumothorax. Examination demonstrated no tamponade and minimal/no cardiac contractility (this is what I would expect to see in a cardiac arrest). It also demonstrated bilateral lung sliding signs suggesting against a significant pneumothorax. Prior to Mr Bayoh coming into hospital I had experience of performing echocardiography having worked with the echocardiographers at [redacted] Hospital one morning a week for approximately 3-4 months to learn the technique and then performing echo's on ICU patients as part of my regular practice. I had less experience of lung scanning at that point.

31. I have no recollection of Mr Bayoh being shocked but the notes state he did receive shocks and I therefore believe it to be the case that he did.

32. From the notes amiodarone, adrenaline, naloxone were administered to Mr Bayoh. Amiodarone and adrenaline as part of standard resus protocol, was

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not there prior to arrest which is when I assume the naloxone was given but it would have been to reverse the effects on any opioids that potentially may have been in Mr Bayoh's system. I note I stated Mr Bayoh was intubated without drugs.

33. I have always known it as the "thumper" it can also be called a mechanical CPR device or an automated chest compression device. It is used in prolonged CPR (i.e. hypothermic patient or beta blocker OD) or for transfer of patient in cardiac arrest although I have never done this. Its purpose is to replace chest compressions performed by healthcare staff. It encircles the patient and compresses the chest using a piston at the front of the patient. I have never set up the thumper and if we use it in the ICU we get A+E staff up to help us set it up, so I do not feel qualified to comment further.

34. A thumper was used on Mr Bayoh. From my statement the thumper was placed too low initially and then readjusted to the correct position. Unsure of how long it was used for.

Fractured ribs

35. Rib fractures are a recognised complication of chest compressions, I do not have a lot of experience of the thumper so cannot comment on whether it is more or less likely to cause this complication. If the thumper is placed too low, again, I do not have enough experience to comment. If any rib fractures were to occur, as a result of too low a placement, I would guess however they would occur in the lower ribs as these are the ones being compressed.

36. I don't think I did any chest compressions, generally the person doing chest compressions remembers if they fracture a rib if there is a cracking noise or you can feel a crunch, but you don't always get that, so it is possible to break a rib and not know.

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37. Fractured ribs (and sternum) are a hazard of chest compressions either manual or mechanical and the incidence rises with age, being very uncommon in those under 20 and quite common in the elderly. I am not a forensic pathologist so cannot comment with any authority on this subject but there is a paper published titled "cardiopulmonary resuscitation complications encountered in forensic autopsy cases" (BMC Emerg Med 2019;19:23) which looked at incidence and location of rib fractures post CPR, 26% of patients had rib fractures and most of them were located in the upper 6 ribs with the 2nd rib being the most common. They did not state how often you got an isolated first rib fracture. However in my practice I have never noticed a first rib fractures post CPR. The difference may be because rib fractures are often not visible on Chest X-ray so they are present at autopsy but I never see them on the CXR, alternatively there may be a difference between rib fracture incidence in survivors vs non survivors of CPR and I only really see the survivors in my ICU, or finally isolated first rib fractures may be very rare post CPR.

Life pronounced extinct

38. I'm presuming the decision to pronounce life extinct was a group decision from my statement and A+E notes, usually CPR continues until all team members agree to stop. In this case from my statement I agreed to stop once rhythm changed to asystole and patient had failed to make any improvement despite over an hour of CPR.

39. I do not know who called the time of death.

40. In relation to what caused this patient to die, the Post mortem will be a lot more accurate than me, I think the most likely diagnosis from the information I had at the time was drug overdose plus exertion (and possible hypoxia) from resisting arrest leading to cardiac failure, possible serotonin syndrome and possibly pulmonary oedema (which would explain the severe hypoxia) and all of the above caused the respiratory and cardiac arrest.

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41. From my statement I thought the resuscitation effort had been performed well. After the time of death I wrote up my notes and presumably finished my shift.

42. From the notes CPR of the patient was ongoing for 1 hour 14 minutes so the patient was in the hospital for slightly longer than that.

Miscellaneous

43. I vaguely remember the PIRC interview. I was just asked to make a statement, don't remember any specific questions. I do remember being asked to clarify medical terminology into laymans terms as people reading my statement would not necessarily be medically trained.

44. I still work with Dr Surinder Panpher. I had an email from Dr David Hall asking if I had access to the medical records and replied to him that I was trying to get access to them too.

45. I am aware there was controversy over a diagnosis of "excited delirium" which I had never heard of prior to this and am aware it's controversial whether it exists. Also aware that the TV reports on the case from time to time. I am not influenced by anything so far.

46. I believe the facts stated in this witness statement are true. I understand that this statement may form part of the evidence before the Inquiry and be published on the Inquiry's website.

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