

## Medicines and murder

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**THE MEDICO-LEGAL SOCIETY**

**Notes of a Meeting**

**held by Zoom Meeting**

**on**

**Thursday 10 December 2020**

**The President**

**Professor Harry Zeitlin  
(in the Chair)**

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**Guest Speaker**

**PROFESSOR ROBIN FERNER**

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Transcript of the shorthand notes of Shelley Dutton  
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## **The Medico-Legal Society**

A meeting of the Society was held via Zoom Meeting on Thursday, 10 December 2020. The President, Professor Harry Zeitlin, was in the Chair.

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### **Medicines and Murder**

**Professor Robin Ferner, Honorary Consultant Physician and Clinical Pharmacologist at City Hospital Birmingham, and Honorary Consultant at the West Midlands Centre for Adverse Drug Reactions**

**The President:** It's seven o'clock and can we welcome all our listeners who have come to listen to a zoom meeting in spite of all the difficulties. We are very pleased and it is very rewarding to have quite a lot of new people, people who have come in either as new members or people who have come to have a look at our meetings to see whether they would like to become members. We say you are all very welcome, both as people who are going to be listening to our talks but also as people who might like to see how they can contribute. Welcome to you all and I have no doubt that we are going to have a very interesting evening.

Tonight, we are very fortunate in having Professor Robin Ferner. I've not met him before but I've been looking at his CV: Professor of Clinical Pharmacology at the University of Birmingham; Associate Professor at University College; Consultant Physician and Clinical Pharmacologist at City of Birmingham Hospital. I could go on, but a very experienced and certainly a person in demand. His CV says he has worked for nearly 40 years as a NHS physician and for 25 years as a Director of the West Midlands Centre for Adverse Drug Reactions. He served as Secretary for the BPS Clinical Subcommittee. So, a very experienced person and I think we are very fortunate to have somebody of this quality to help us know more about the subject. I don't know if there's anything else I should add to that and perhaps we could ask him to get underway. What we will be doing, if I may tell everybody, is that we have asked him if he can talk for 45 minutes and if people can let

Sandra know if there are questions that they would like to ask because we will have a 15-minute question period at the end.

I think we might ask him to get underway, please.

**Professor Ferner:** Thank you very much.

I am going to be talking about medicines and murder which is, if you like, a hobby. I have a declaration of interest which is that I have provided medico-legal opinions on harm from medicines and drugs in general in cases including cases of murder which are fortunately rather rare though I read in yesterday's paper that, during its 60 years on air, there have been 25 murders in *Coronation Street* and some of the murders we will come on to discuss occurred near Manchester if not actually in Manchester.

Some of the cases I am going to talk about I've been involved with but not all of them and that will become clear more or less from the start. I have half-a-dozen cases that I am proposing to discuss.

The first is the case of The Guilty Gambler. It was gambling that led to the death of a man called John Parsons Cook in the Talbot Arms in Rugeley in Staffordshire less than 30 miles from Birmingham but before my time, actually in 1855. Cook was interested in horse racing and, indeed, was quite successful. He had a horse called Polestar that won a race in Shrewsbury shortly before Cook's death. He also had a friend, Dr William Palmer, a local general practitioner, who owed Cook quite a lot of money; he owed other people much more money. He temporised by forging cheques using his mother's name – his mother was independently wealthy – but at the time that Cook died Palmer still owed a great deal of money.<sup>1 2 3</sup>

John Parsons Cook became unwell while still in Shrewsbury, probably as a result of a drink that Palmer had given him and which caused vomiting. He improved. He goes back with Palmer to Rugeley and Palmer treats him with a concoction that Cook drinks and says, "It

burns my throat”. He then becomes quite seriously ill, recovers temporarily until Palmer gives him a second draught and then he develops convulsions and dies in agony because, although convulsing, he is still conscious.

There is a post-mortem in spite of the fact that the elderly Dr Bamford said that this was cholera. The post-mortem in itself was interesting; it took place in public; Palmer was there. The operator was actually a medical student by the name of Devonshire and, as he was removing the stomach with the help of the pathologist, he was “bumped” by a man called Newton whom William Palmer had himself nudged and, as a consequence, the stomach contents were spilt. This was an unfortunate accident made more remarkable by the fact that William Palmer turned to his elderly colleague Bamford and said, “They won’t hang us yet”, a prophesy which did not, in fact, come true – as we will see.

Alfred Swaine Taylor was the pre-eminent toxicologist of the 19<sup>th</sup> century; he was a Guy’s man, so knew no wrong. He analysed the samples from Cook’s post mortem without any clear idea of the clinical story, found antimony, which is a poisonous metal, and decided that Cook had died from antimony poisoning. Antimony would be quite unable to explain the convulsions or the failure to lose consciousness. Swaine Taylor wrote a report saying that antimony was the cause but, when he came to give evidence in court, said that actually, the poison wasn’t antimony, it was strychnine. The antimony was from treatment with tartar emetic (antimony tartrate), which was widely used and which Cook was known to have taken; he could not find strychnine because it had been destroyed post-mortem.

A rival of Swaine Taylor’s, William Herapath from Bristol, pointed out that Swaine Taylor had changed his opinion, that he was unable to find the strychnine, that the clinical events could be explained by natural tetanus, which is an infection that causes precisely these symptoms and, as Herapath put it regarding the destruction of strychnine after death, “No authority can be drawn from experiments and it is supported by no eminent toxicologists but themselves”, that is the toxicologists giving evidence for the Prosecution. This sounded compelling but it was not sufficient to persuade the jury that the tetanic spasms, the convulsions that John Parsons Cook suffered, were caused by natural disease; they were

happy to believe that they were caused by strychnine and the consequence was that William Palmer was convicted and hanged at Stafford Gaol in 1856 (Figure 1).

His execution was itself notable because, as he was taken to the scaffold, he stood at the bottom of the rickety steps and he looked up at the rather ill-used apparatus, he turned to the executioner, he looked up again and he said, "Is it safe?" The answer for Palmer was "No".

Strychnine is not much used in medicine these days, but it is an interesting toxin. In the normal course of events, motor control depends on the spinal motor nerves, the anterior spinal nerves, and they send an impulse and the impulse is carried on but the nerves do not fire repeatedly, because they are 'turned off' by an inhibitory nerve transmitter substance called glycine. What strychnine does is interfere with glycine; it stops glycine having this calming effect on the nervous system and the consequence is repetitive firing and the tetanic spasm which was so evident in John Parsons Cook.

Let's pass on to the 20<sup>th</sup> century where one of the things that distinguished the gentry in Hay-on-Wye was a well-manicured lawn and there was probably no more well-manicured lawn than that of this man, Herbert Rowse Armstrong, a solicitor in the town. Major Armstrong had been away at the War for five years. He returned to his wife, who henpecked him. Some of you will know the system, though I think even the most henpecked of you will not have been playing a game of bowls with your friends to be told by your wife, "Herbert, you must come in, it's your bath night".<sup>4</sup> He endured this for many years. He was a punctilious man as you can see from the photograph (figure 2), and one of the aspects of this was that he had an intense dislike for dandelions, a dislike which he manifested by buying quite a large quantity of arsenic, which he broke down into 20 small packets, each of which was designed to kill one dandelion. He used 19 packets in precisely this way, leaving one packet in his coat pocket.

Armstrong's wife, who had spent some time in a mental hospital, had recovered sufficiently by January 1921 to go home, but then, returning to Hay-on-Wye, she became

ill again and died from neuritis and heart failure. She was buried and Armstrong carried on with his solicitor's practice.

The story might have ended there but, in his time away, another veteran, a man called Oswald Martin, had come home and set up a rival practice as a solicitor. Martin had been wounded early in the war and invalided out, so he had plenty of time to acquire clients who had previously been with Armstrong and there was no love lost between them, though they tried to maintain civilities, so much so that Armstrong pressed Martin to come to tea repeatedly. Eventually, Martin did come to tea and they had tea and scones. The slightly surprising aspect and one which probably contributed to Armstrong being hanged is that rather than hand Martin the plate, Armstrong picked up one of the scones and handed it to his solicitor colleague with the phrase, "Excuse fingers".<sup>5</sup> Martin took the scone, ate it, and was taken ill; he vomited profusely afterwards. The doctor who looked after Armstrong's wife, Dr Hincks, was called and – persuaded by Martin's father-in-law, who happened to be the local pharmacist – he organised for analyses to be made of the solicitor's urine which showed, as you might already have guessed, arsenic. So, too, when exhumed, did the corpse, the well-preserved corpse I should say, of Mrs Armstrong.

The Prosecution in this trial was led by the Attorney General though, at least according to the account of Martin Beales, a solicitor in Hay-on-Wye in the 1990s, Mr Justice Darling, the trial judge, helped greatly in the prosecution of the Crown's case by cross-examining Armstrong in great detail about the dose of arsenic in his little packets.<sup>6</sup> Mr Justice Darling pointed out was enough not just to kill a dandelion but also a man. The Prosecution maintained that the neuritis was caused by chronic arsenic poisoning. The defence was simply that while Kathleen Armstrong had died from arsenic poisoning, she had taken it herself, either for her complexion or because it was contained in the "homeopathic" remedies she took. This did not save Herbert Rowse Armstrong, who was hanged in Gloucester in 1922.

Arsenic damages cells by binding to the sulfhydryl (-SH) groups essential for your enzymes to work. The consequence is, if you take enough of it, severe, acute diarrhoea and

vomiting – if you are interested in these things, then the Seddon case is a pretty good example of acute arsenic poisoning, but we won't stray there – heart failure, kidney failure and death within a few days. Chronic arsenic poisoning however can cause nerve damage, can make you mad, which pretty much is what happened to Mrs Armstrong, can cause skin changes (arsenical keratoses), which is useful clinically, liver damage and cancer.

That was Herbert Rowse Armstrong, you will remember a Cambridge-educated solicitor.

We now move on to *A Woman Scorned* and this is a drama which plays out between Newcastle and John Bell & Croyden in London.<sup>7</sup> Margaret Vickers was a brilliant but infirm Cambridge mathematician married to a man she met when he was an undergraduate medic in Cambridge, Paul Richard Jarvis Vickers. Margaret Vickers suffered from schizophrenia. In due course, she developed a severe anaemia and bone marrow failure and was admitted to the Royal Victoria Infirmary in Newcastle. In normal bone marrow you see lots of cells; in Mrs Vickers's bone marrow there were hardly any cells: she had aplastic anaemia, that is, there were no cells making the constituents of blood, and she died. Aplastic anaemia can occur, for example, as a result of virus infections and there was no suspicion that Mrs Vickers had died from anything unnatural.

Paul Vickers had consoled himself before his wife's death with a woman, Pamela Collison, who had at one time been Mr Heseltine's special adviser I suppose you would call them now, and a former beauty queen. Vickers promised to marry Ms Collison but he didn't. This had quite important consequences. She went first to Dr Gerard Vaughan, who was Secretary of State for Health I think at that time, and said, "Gerry, what shall I do?" He advised her to go to the police, and she went to the police and said, in so many words, "I took prescriptions to John Bell & Croyden because Paul asked me to, and the prescriptions were for the anti-cancer drug CCNU". CCNU (lomustine) causes bone marrow failure and the Prosecution case was that Mr Vickers had given CCNU to his wife and murdered her so that he could prosecute his affair with Pamela Collison.



The defence was, in my view, extraordinary. It was that he was treating his wife for a brain tumour. So, first, the diagnosis was on clinical grounds alone; secondly, Vickers was a casualty surgeon and he had no experience of treating brain tumours; and thirdly, the medicine he used was quite inappropriate. He was convicted and his appeals on more than one occasion failed. Ms Collison was acquitted and went free, though what happened to her afterwards I don't know.

We now move to the 1990s and Birmingham and the Tower Ballroom. This is a couple of miles from the hospital where I used to work. In fact, on more than one occasion, the ward night out at Christmas was held at the Tower Ballroom. The events I am talking about occurred in August 1996 when the body of a young woman wrapped in a carpet was found on waste ground beside the ballroom. The murder case came to be called by the popular press "The Band of Gold Murder." At that time, there was a television series called *Band of Gold* about prostitution and a poor 16-year-old schoolgirl from Staffordshire watched the programme, thought this might be quite fun and set about earning money in the way that women in *Band of Gold* did. In the course of her work, she met two men who befriended her and who took her back to their place. There she took or was given drugs, lapsed into coma and her respiration became shallow. The two men put her to bed upstairs and 12 hours later she was dead.

On analysis, her blood contained those substances [6-monoacetylmorphine, morphine, and morphine glucuronides] that meant she had been given or had taken heroin. In fact, she probably smoked it. There was no doubt that the heroin caused her death; she was otherwise a fit, young girl. At trial, John Mitting QC, who at the moment is busy with undercover policing, established that, at any time before she died, she could have been saved by appropriate treatment. The defence was: this was not murder; there was no intent to kill her even though the malefactors had given her the heroin, so this was manslaughter by gross negligence. The two men were convicted of manslaughter and were given ten-year sentences. They also concurrently had sentences for firearms offences and drug offences.

The case is of some interest because it went to the Court of Appeal, who held that a drug dealer has no duty of care to the person he or she supplies with drugs, and so the manslaughter conviction was quashed. It didn't do the two perpetrators very much good because they were still serving sentences for other crimes, but the judgment does seem to make it more difficult to convict people for the death of persons to whom they have supplied drugs.<sup>8</sup>

The eponymous curry in what I've called The Curry Murder was an exhibit at trial. This sad story involves a love triangle and this man, Lakhvinder Singh Cheema, who was known to his friends, ironically, as Lucky. Lucky had a longstanding girlfriend; they had been seeing each other for about 12 years when Lucky bumped into a pretty, much younger girl called Gurpreet, who became his fiancée. Lucky and Gurpreet had supper one evening in 2008 and, after supper, they both became ill. Lucky became unable to walk; he developed copious vomiting; he found that his eyesight became dim; and he had tingling in his hands and around his mouth. He was still able at about 8.00 pm to ring the London Ambulance Service and say, "I think we've been poisoned".

You can see from the CCTV camera that, four hours later, his cousins came and took him in a car to hospital with Gurpreet because the London Ambulance Service had not turned up. In due course he arrived at hospital but, shortly after arriving, he had a series of convulsions and a cardiac arrest, and he died. His fiancée had only taken about half what he took and although she became very unwell with vomiting and weakness in her legs and although she developed heart rhythm disturbance, she was treated with all the treatments that people could think of and whether because of or in spite of that, I am pleased to say that she survived.

There wasn't very much doubt that she had been poisoned and that Lucky had died from poisoning, but the poison was difficult to find and a colleague of mine, Professor Robert Flanagan, suggested that it might be aconite. Aconitine is the poison in *Aconitum napellus* (monkshood), the beautiful flower which, if you are brave, you grow in your garden. Another colleague carefully analysed various samples by a very sensitive technique that

separates substances by molecular weight (or, more precisely, by the ratio of molecular weight to charge) but there was no aconitine.

I was asked what I thought, and I am not sure that I thought very much at this stage except that aconite sounded possible. I went back to the *BMJ* or actually to the predecessor to the *BMJ*, the *Medical Times and Gazette*, and, in 1882, they ran this article which said, “Poisoning by aconite in India. Last week we drew attention to the general features of poisoning by aconite” – that was because of the Lamson case – “and we then indicated that with regard to certain species at all events our Indian medical brethren were far better authorities than ourselves”. It won’t have escaped your notice that Lakhvinder Singh and, indeed, Gurpreet, were of Indian origin and I wondered a little about this. It turns out that, in India, the aconite is actually *Aconitum ferox*, the Himalayan or ferocious aconite, in which the toxin is not aconitine but a very closely related compound, pseudoaconitine, with a molecular weight slightly different, 690.<sup>9</sup> That was one contribution. A second was to suggest that the Metropolitan Police enlist the help of Professor Monique Simmons at the Royal Botanic Gardens, who runs a famous laboratory for analysis of plants and plant poisons. She and her colleague Geoff Kite were able to demonstrate pseudoaconitine at 690.348 daltons – that’s the molecular weight – in the curry sauce, in the blood of Lucky Cheema and also in a herbal powder which happened to be in a raincoat hanging on the banister of the house where Lucky Cheema’s former mistress, Lakhvir Kaur Singh, lived. She was tried at the Old Bailey, convicted, and sentenced to 23 years in prison.

The last case I wanted to talk about is a case from Stockport, just south of Manchester. It has an important general hospital with two big admission wards, A1 and A3, and treats many patients.<sup>10</sup>

All went well until 2011 when there was an outbreak of low blood glucose concentrations, technically hypoglycaemia, on the admission wards. These outbreaks were sporadic and they were unpredicted. They were unpredicted because most hypoglycaemia that one sees in hospital occurs in patients who are given anti-diabetic drugs (including insulin) and none of these patients was diabetic.

To put the hypoglycaemia in context, the normal fasting blood glucose concentration is between 4 and 7 mmol/L. Below about 2 mmol/L you become unwell and below 1 mmol/L you lose consciousness. The values in patients during the outbreak on Wards A1 and A3 were, by and large, much lower than 4 and many of them were much lower than 2. Two patients died and one man suffered permanent damage as a result of the hypoglycaemia.

What was it all about? Greater Manchester Police put a ring round the hospital and combed the place without finding out, and life continued on Wards A1 and A3 until one day a nurse went into this room, the IV preparation room, and took out a vial to make up an injection and she noticed, before she gave the injection, that the vial was wet, it was leaking. On closer inspection, she noted a tiny pinhole, actually a needle hole. She put a drop the liquid from the bottle on her hand and she said, "This smells of insulin". I had no idea that insulin smelled even though I did some research on diabetes and I've been injected with insulin in the name of science and I have given many patients insulin. So, I went to the ward fridge and, in fact, insulin smells strongly of phenol or, more precisely, ortho-cresol, so it is a very distinctive smell and the nurse proved to be right. Someone had been putting insulin in bottles and bags in the IV preparation room, not in the patients directly. Who it was only became apparent after he subsequently forged a prescription.

The business of diagnosing insulin poisoning is more straightforward than the popular imagination allows. The pancreas gland secretes insulin and it does it having made proinsulin which is a big molecule which comes in two bits, one of which is insulin itself and the other of which is a sort of cap, C-peptide, which is of no physiological purpose but it is in your blood and, every time the pancreas makes a molecule of insulin, it also makes a molecule of C-peptide. Actually, C-peptide lasts longer in your blood than insulin does and so, in normal people, there is a good deal more C-peptide in the blood than insulin. By contrast, if you inject insulin into somebody, there is only insulin, because the bottles of insulin are, if you like, cleaned of C-peptide. The consequence of this is that, when you look at the laboratory results, people who have been given insulin have low blood glucose concentrations because that is what insulin does, they have high insulin concentrations

because you have given them insulin, but the C-peptide concentration is depressed because your pancreas is switched off by the low blood glucose concentration. So, that meant that samples that were still available could be analysed to see whether insulin had, in fact, been the cause of the hypoglycaemia that broke out on Wards A1 and A3.

Here are two of the cases. The first was a 44-year old woman who had what was believed at the time to be multiple sclerosis. In fact, it turned out not to be that but something much rarer. That is not relevant except that a neuro-disability meant that she was liable to chest infections and, when she got chest infections, she needed antibiotics and that is what happened on this occasion. She was admitted to hospital; she was given an antibiotic for her chest and then, for no particular reason, she became seriously hypoglycaemic. There were two samples in her case: one taken when she came into hospital, which showed that her glucose concentration was normal even though she had untreated pneumonia, that her insulin concentration was, if anything, low – which is what we expect in infection – and that her C-peptide concentration was normal. When her glucose concentration plunged, she had the full house: she had a very low glucose concentration, very high insulin concentration and no C-peptide to measure. This was insulin poisoning.

The second, a 41-year old man who had been admitted to hospital reasonably well and later was observed to be in a coma by the man diagonally opposite him on a night when the suspect was on duty. At 6.15, the patient, still deeply unconscious had a blood glucose concentration only 10 per cent of normal. He, too, had had normal readings on admission and grossly abnormal readings, consistent with insulin poisoning, when his blood glucose concentration fell.

There was one other important point about this patient, and that is that when his blood was sent to Cologne, Professor Mario Thevis, the analyst there, was able to detect the breakdown products of insulin glargine, which is a long-acting insulin, which is completely synthetic and could not have been in the body. It will not surprise you to know that the man responsible received 25 life sentences.

I think there are a couple of lessons. First of all, as I have said, if you see hypoglycaemia that severe, look for a cause; second, if you see hypoglycaemia that severe, just consider why it might have occurred.

We are nearly at quarter-to and I want to reach some conclusions: (1) beware of lovers and of healthcare professionals; (2) suspect poisoning; (3) consider clinical pharmacologists as they can sometimes be useful.

This is the conclusion that *The Times* reached on the trial of William Palmer. There's Swaine Taylor and his colleague, Dr Rees, failing to detect strychnine as it happens and *The Times* said, "The contradictory deductions and tetanic complications of medical professors make wise men fumble, good men sad and bad men bold" and here we are. Thank you.

**Ms Sandra Marcantonio:** We have a question from Rob. Which speciality doctors within hospitals do the most work with poisons?

**Professor Ferner:** That depends on the hospital you are in. If you are lucky, you are in one of a half-dozen hospitals that have a poisons unit. Here in London it is St Thomas', in Birmingham it is City Hospital where I was, in Newcastle it is the RVI, and so on.

**Ms Sandra Marcantonio:** Would that be Accident & Emergency?

**Professor Ferner:** No, they have poisons units, so there are clinical pharmacologists or clinical toxicologists with an expertise in poisons but, in most hospitals, most poisonings will be dealt with on an acute medical unit.

**The President:** Do you get called to look at cases and give your opinion and, if so, where do you start?

**Professor Ferner:** Are we talking about live cases or dead cases?

**The President:** Let's start with live ones. Where do you start if somebody asks, "Could this be a potential toxin?" Where do you start if you are called?

**Professor Ferner:** It goes like this, I think. The first thing, as with the whole of medicine, is to obtain an accurate history. It gives you many clues because there are characteristic features of poisoning with different agents. For example, if you are poisoned as the Skripals were, with an organophosphorus compound, then the features are characteristic: you get pinpoint pupils; you froth at the mouth; you are incontinent of urine and maybe of faeces; you lose the power to move your muscles and so on. So, these so-called toxidromes, toxic syndromes, give you a clue.

The second question you ask yourself is what you find on clinical examination if the patient is still alive. If the patient is dead, then there is a large literature on the appearance of the stomach, for example, in arsenic poisoning, which is almost no use I think but some pathological findings are consistent with poisoning. Then you look for the special investigations and the special investigations these days are very sophisticated. Sometimes one may suspect poisoning, but not find it. Some of you know may know about the case of Alexander Perepilichnyy, who was a Russian oligarch, and no friend of the Russian President. He went jogging and was found dying by the side of the road. The Coroner held an inquest at the Old Bailey, which itself was unusual, and the verdict was natural causes.

**The President:** Murders still go on, sadly, at quite a high rate but do you find that the use of poisons is changing progressively over time?

**Professor Ferner:** As I think I said in my preamble, unfortunately, there are not very many good poisonings. There is the mundane business of death as a consequence of drugs of abuse, which is a terrible problem and terribly miserable but does not usually involve charges of murder. Otherwise, either the poisonings are undetected or there are not very many of them.

**The President:** For what it is worth on that, we looked back and found a population of drug takers five years ago and then followed them up to see what had happened and not many people are aware that the death rate between 18 to 25 was 25 per cent. People do not realise that. Yes, it is a very significant area and horrible.

**Mrs Diana Brahams:** Thank you very much for your talk. Like many other people, I quite enjoying watching murder mysteries on TV or reading about them and I often wonder how accurate some of them are, in particular Agatha Christie who was very interested in poisons and a lot of her deaths are due, of course, to poisoning. There was one that we watched recently about nicotine poisoning. I wonder, is she as good as they say about her poisoning?

**Professor Ferner:** I think so. The first thing to say about her is that she worked as a dispenser in the pharmacy at UCH during the Second World War. When I was a houseman, and we were able to obtain the keys to Pharmacy down in the basement of the old UCH building, the cruciform building, you felt that she was looking over your shoulder, which was a rather frightening experience. The second thing is that she does use a wide range of poisons and there is famously the case of the nurse at the Hammersmith Hospital who was looking after a patient who was semi-conscious and who had lost her hair. During the nightshift, the nurse read *The Pale Horse*, which is an Agatha Christie story about thallium poisoning and, in the morning, she said to the consultant, “Your patient has thallium poisoning” as, indeed, he turned out to have.<sup>11</sup> So, sufficiently good to make a clinical diagnosis, I think.

**Mr James Pattison:** Robin, thanks very much. That was a very informative and entertaining talk. The Russian Intelligence Services seem to be keeping you chaps in business. The polonium took a long time to sort out with Litvinenko, didn't it? Can you tell us a little bit about that?

**Professor Ferner:** I am glad you asked me that. Let me tell you a little bit about Litvinenko. The problem there was that it was not truly poisoning. He had been given



something which depressed his bone marrow and made his hair fall out, so it could have been CCNU and, in fact, the late John Henry, who was a world-renowned English toxicologist, had postulated that it was thallium, possibly radioactive thallium, but there was no sign of radioactivity as elicited by a Geiger counter. This is because polonium emits helium particles— I think I have that right, not many gamma rays, a few [0.001% of the emissions] – and, at the time of the Litvinenko poisoning, scientists at the Atomic Weapons Establishment had been setting up kit to undertake gamma ray spectroscopy, itself *recherché* technique I think it is fair to say, and they said, “We’ll have a go and see what we find”. A sample of Litvinenko’s urine was sent down to them and it arrived just at the end of the day and so the chaps put it on the spectrometer and left the spectrometer counting overnight and, when they returned, there was a small peak that had accumulated over the evening which turned out to indicate gamma radiation of a particular energy [803 keV] that could only have been due to a handful of compounds and it did not take very long after that to establish that it was polonium-210. So, it was more or less by chance, as I understand the story. You probably know that there was a substantial inquiry into the Litvinenko case and the data are in the public domain, so those of you not yet satisfied can go to the web browser of your choice and find the report which runs to a substantial number of papers and look it up.<sup>12</sup> James, I don’t know whether that helps. Were you involved in the care of Litvinenko?

**Mr James Pattison:** No, he was at UCL.

**Professor Ferner:** He was, yes.

**The President:** For what it is worth, it is possible that we overlapped at UCL as well. I saw that you had some nice old engravings of people from past cases. Have you written a book on the subject?

**Professor Ferner:** Not yet. I plagiarised those from this, which is *The Times*, the Trial of William Palmer,<sup>3</sup> which has many very beautiful engravings and which I must have acquired at one time, possibly from your library. Actually, it was from a man called Les

Bolland, a retired policeman who took to selling books on crime but whose main interest was Jack the Ripper who was of no interest to me.

**The President:** Anybody else? We are coming up to the hour. I regret that we cannot give people some nibbles and a glass of wine. Are there any more questions?

**Professor Ferner:** I notice there is a question on the chat about Beverley Allitt and I have hidden my involvement in that case because (1) I was young and (2) I provided evidence at the request of the defence. I never met her and I have no idea why she did what she did. You know and will have read some of the popular accounts which suggest that she had Munchausen's Syndrome by proxy but I do not know whether that was so and I think you would have to turn to Harry or one of the other psychiatrists to discover a motive.

**The President:** I will have to look it up. Does anybody else have any questions because we have just come to the hour? If not, we must thank our speaker for a depth of knowledge that I doubt many of us do have on this topic. It is very, very kind of you and it is very nice of you to talk under such difficult circumstances as these. Can we thank you and welcome you back to future meetings. We have a variety of topics. The person on my screen who is just above you is the person who runs them, Sandra Marcantonio, and I am sure she would always welcome you to come and join us. I am just looking at the people on the screen to see if there is anybody else who would like to make a comment or ask a question.

I have been involved in a variety of criminal cases but, poisons, I have learnt new today. Thank you very much. Can we give you applause. (Applause)

I think I probably ought to add that if anybody has a subject or a topic which they feel would be of interest and would hold our fascination, please, do let us know. We have a number of speakers but we are always interested.

The next meeting is on 14 January and is the medico-legal considerations of football. I have to say I know nothing about that whatsoever.

I wish you all a very good Christmas New Year and thank Sandra for keeping things under control and organised. Thank you.

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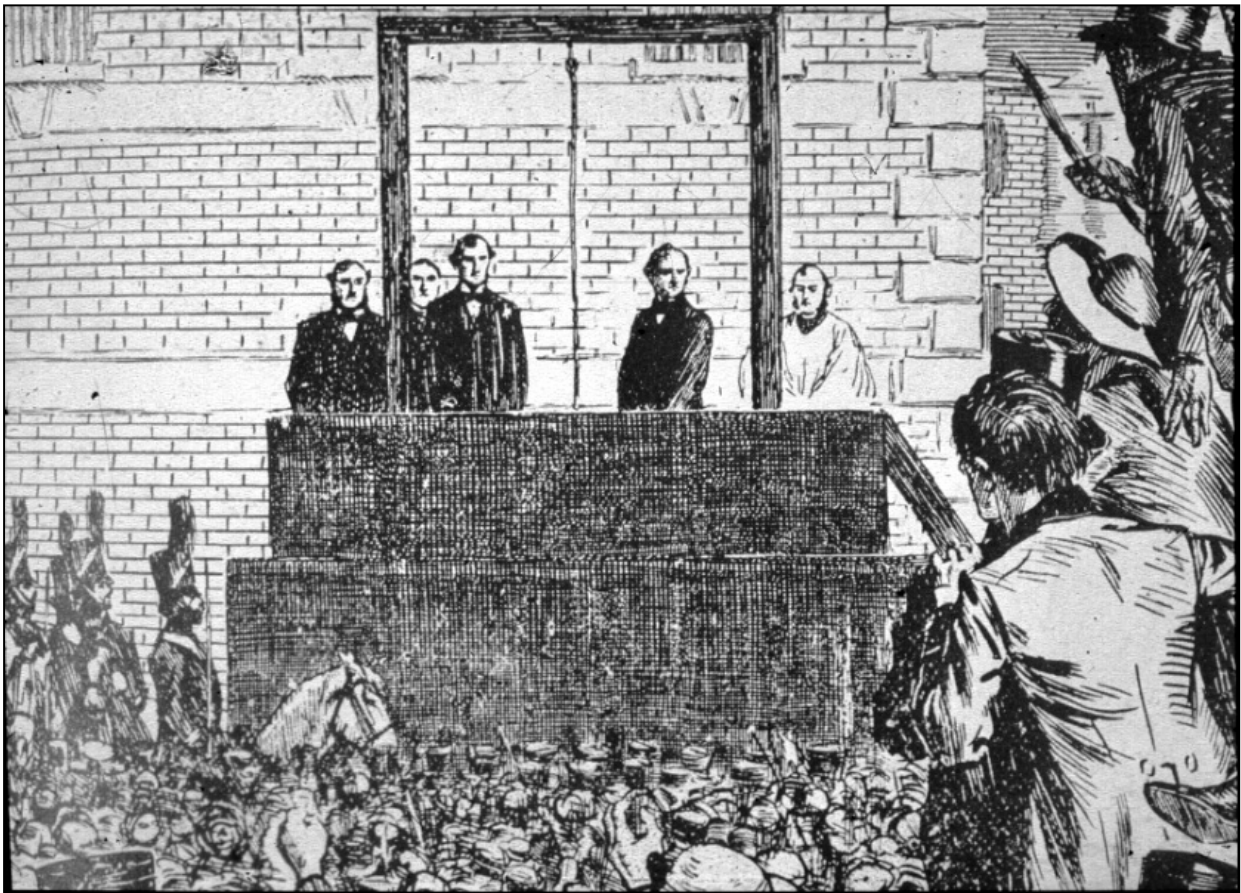
## Legends to figures

Figure 1: Dr William Palmer on the scaffold at Stafford Gaol, 1856. [From reference 3].

Figure 2: Herbert Rowse Armstrong, MA. [By Unknown author - contemporary photograph, Public Domain, <https://commons.wikimedia.org/w/index.php?curid=7610953>]

Figure 3: Dr Alfred Swaine Taylor and Dr Rees conducting analyses, and detecting antimony but not strychnine. The apparatus is probably generating hydrogen from zinc granules and sulfuric acid in a form of Marsh's test to detect arsenic and antimony. [From reference 3].

Figure 1



Dr William Palmer on the scaffold at Stafford Gaol, 1856. [From reference 3].

Figure 2



Herbert Rowse Armstrong, MA. By Unknown author - contemporary photograph, Public Domain, <https://commons.wikimedia.org/w/index.php?curid=7610953>

Figure 3



Dr Alfred Swaine Taylor and Dr Rees conducting analyses, and detecting antimony but not strychnine. The apparatus is probably generating hydrogen from zinc granules and sulfuric acid in a form of Marsh's test to detect arsenic and antimony. [From reference 3].

