

WITNESS STATEMENT

(CJ Act 1967, s.9; MC Act 1980, ss.5A(3)(a) and 5B; MC Rules 1981, r.70)

Statement of Professor Robert James Flanagan..... URN: [] [] [] []

Age if under 18 Over 18..... (if over 18 insert 'over 18') Occupation: Clinica/Forensic Toxicologist.....

This statement (consisting of: 7..... pages each signed by me) is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything in it which I know to be false, or do not believe to be true.

Signature: R. J. Flanagan..... Date: 12 March 2011.....

Tick if witness evidence is visually recorded [] (supply witness details on rear)

This statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything that I know to be false or do not believe to be true.

I confirm that I have read guidance contained in a booklet known as Disclosure: Expert's evidence and unused material that details my role and documents my responsibilities in relation to revelation as an expert witness. I have followed the guidance and recognize the continuing nature of my responsibilities of revelation. In accordance with my duties of revelation, as documented in the guidance booklet, I thus:

- a. Confirm that I have complied with my duties to record, retain and reveal material in accordance with the Criminal Procedure and Investigations Act 1996, as amended;
b. Have compiled an index of all material. I will ensure that the index is updated in the event that I am provided with or generate additional material;
c. Have complied with my duty to the Court to provide independent assistance by way of objective unbiased opinion;
d. That in the event my opinion changes on any material issue, I will inform the investigating officer as soon as reasonably practicable and give reasons.

Signed: R. J. Flanagan..... Date: 12 March 2011

Introduction

1. My full name is Robert James Flanagan and I am a toxicologist currently employed as a Consultant Clinical Scientist at the Toxicology Unit, Department of Clinical Biochemistry, King's College Hospital. I hold the degrees of Bachelor of Science and Doctor of Philosophy. I am a Fellow of the Royal College of Pathologists, a Chartered Chemist and Fellow of the Royal Society of Chemistry, a State Registered Clinical Scientist (Registration no CS 768), and a

Signature: R. J. Flanagan..... Signature witnessed by:

Continuation of Statement of

EuroTox Registered Toxicologist. I have some 40 years experience of clinical and forensic toxicology and am the author or co-author of more than 190 articles in scientific journals and 4 books. I am Visiting Professor in Analytical Toxicology at the University of Loughborough and Honorary Professor of Analytical Toxicology in the University of London. I am an examiner in Toxicology for the Royal College of Pathologists and an examiner in Forensic Medical Science for the Worshipful Society of Apothecaries. I was a Registered Forensic Practitioner, Toxicology (Registration no. 01862) until the closure of the register on 31 March 2009. I am a member of the British Pharmacological Society and of the British Association for Psychopharmacology. I am a member of the Council of the British Academy of Forensic Science.

2. You have supplied me with copy statements of (i) Dr Alexander Richard ALLAN dated 21 July 2003, 18 August 2003, and 17 September 2003, (ii) Dr Nicholas Charles Alexander HUNT dated 25 July 2003, (iii) Anne Mirabel Louise FRANC dated 2 September 2003, (iv) Renee GILLILAND dated 4 August 2003, (v) Roy James GREEN dated 27 September 2003, (vi) Eileen HICKEY dated 3 October 2003, (vii) David T MCGEE dated 21 July 2003, (viii) John Phillip SHARPLEY dated 1 August 2003, (ix) Andrew Marshall HODGSON dated 31 July 2003, and (x) Mark Jason SCHOLLAR dated 5 August 2003, all produced as a result of the investigation into the death of Dr David KELLY.
3. You have also provided me with a number of communications critical of the work done by Dr ALLAN and have asked me to provide an independent review of his work in this case and *inter alia* of the toxicological evidence relating to the death of Dr KELLY.
4. On 8 March 2011 I visited LGC Forensics, Culham, the organisation that is now in possession of Dr ALLAN's case file, in order to verify the information contained in his statements by reference to the original source material.
5. In this report I have referred to the analysis of exhibits collected by Dr HUNT at the postmortem examination. The labeling of the exhibits is detailed in the statement of Dr HUNT dated 25 July 2003.

Dextropropoxyphene and Paracetamol

6. Dr ALLAN (his statement of 21 July 2003) reported the presence of paracetamol (97 micrograms per millilitre) and dextropropoxyphene (1.0 micrograms per millilitre) in plain (unpreserved) blood sample NCH/47 (site of collection from the body not stated). Dr ALLAN also reported the presence of substances derived from dextropropoxyphene and of caffeine. These results are consistent with the ingestion of a life-threatening overdose of co-proxamol, a

Signature:



Signature witnessed by:

Continuation of Statement of

formulation containing 325 milligrams paracetamol and 32.5 milligrams dextropropoxyphene hydrochloride per tablet. It is of no significance that dextropropoxyphene and paracetamol were measured in only one of the blood specimens available, presumably a peripheral blood sample.

7. Inspection of Dr ALLAN's original source material confirmed that these results had been reported accurately in his statement (duplicate paracetamol measurements 95 and 98 micrograms per millilitre, and duplicate dextropropoxyphene measurements 1.00 and 0.98 micrograms per millilitre). The presence of norpropoxyphene amide, a compound formed during the course of the analysis from the dextropropoxyphene metabolite *N*-desmethyldextropropoxyphene (norpropoxyphene), and of caffeine were also confirmed. Also noted was the presence a compound derived from paracetamol by comparison with the ultra-violet absorption spectrum of paracetamol, most likely paracetamol glucuronide or paracetamol sulphate. Caffeine is derived from coffee and other beverages and in the quantity detected is of no toxicological significance in this instance.

8. Dr ALLAN (his statement of 18 August 2003) reported the presence of paracetamol (66 micrograms per millilitre) and dextropropoxyphene (0.56 micrograms per millilitre) in the vitreous humour sample NCH/53. Inspection of Dr ALLAN's original source material confirmed that these results had been reported accurately in his statement. These results are consistent with the concentrations of paracetamol and dextropropoxyphene reported in blood sample NCH/47 (paragraph 6 above).

9. Dr ALLAN (his statements of 21 July 2003 and 18 August 2003) also reported the presence of paracetamol (approximately 67 milligrams) and dextropropoxyphene (approximately 19 milligrams) in stomach contents item NCH/49. Inspection of Dr ALLAN's original source material confirmed that these results had been reported accurately in his statements (paracetamol concentration 780 micrograms per millilitre, dextropropoxyphene concentration 22 micrograms per millilitre, total amount of stomach contents supplied for analysis 85.7 grams). These results are consistent with the ingestion of a life-threatening overdose of co-proxamol (paragraph 6 above), and may represent (i) paracetamol and dextropropoxyphene remaining in the stomach after ingestion of co-proxamol, (ii) secretion of dextropropoxyphene and/or paracetamol into saliva and thence passage into the stomach, (iii) diffusion of absorbed paracetamol and/or dextropropoxyphene across the stomach wall, or indeed a combination of all three factors.

10. Dr ALLAN (his statement of 18 August 2003) reported the presence of dextropropoxyphene in sample AMH 2/1 [liquid decanted from AMH 2 (Evian water bottle)] at low concentration. Inspection of Dr ALLAN's original source material confirmed that this result had been reported

Signature: *R.S. Kanoyan* Signature witnessed by:

Continuation of Statement of

- 16. As with all opioids, dextropropoxyphene is more dangerous if ingested by someone who has not been taking it regularly than if taken by someone who has acquired some tolerance to the toxic effects of opioids through regular recent use. Drug tolerance cannot be assessed postmortem.
- 17. Dextropropoxyphene is more dangerous if co-ingested with alcohol, but the absence of alcohol does not make dextropropoxyphene itself less dangerous.
- 18. Dextropropoxyphene is a strong analgesic hence some insensibility to pain resulting from self-harm, for example, will be induced, especially in the presence of paracetamol. Should self-harm result in a lesion that leads to blood loss, dextropropoxyphene-induced hypotension may lead to less blood flow through the lesion than might be expected in a normotensive individual.
- 19. Generally blood dextropropoxyphene concentrations above 1 microgram per millilitre are indicative of serious toxicity, but there are many reports of deaths involving dextropropoxyphene alone and in which the postmortem blood dextropropoxyphene concentrations were less than 1 microgram per millilitre (see RC Baselt, Disposition of Toxic Drugs and Chemicals in Man, Edition 7. Foster City: Biomedical Publications, 2004, page 954).
- 20. Although NCH/47 was only labeled 'plain blood', the fact that NCH/43 was labeled 'heart blood' suggests strongly that NCH/47 was peripheral blood. Collection of peripheral blood would be normal practice. In any event, a postmortem blood dextropropoxyphene concentration of 1.0 microgram per millilitre is consistent with fatal dextropropoxyphene toxicity.
- 21. It is not possible to calculate the quantity of either paracetamol, or dextropropoxyphene ingested from a postmortem blood measurement. If vomiting has occurred, the amount vomited is unknown. Similarly the amount remaining in the gastrointestinal tract at death is unknown, and the amount metabolised prior to death is unknown. There is also the possibility of changes occurring in blood concentrations after death. In the case of fat-soluble drugs such as dextropropoxyphene there is the possibility of an increase in even peripheral blood concentrations by diffusion from solid tissues to blood after death. Any increase is more likely if the interval between death and sampling was prolonged, if the body had been stored at a higher rather than lower temperature prior to sampling, and if death had followed later rather than sooner after ingestion of the drug since more drug may have distributed into tissues prior to death. However, these same possibilities apply in other cases where postmortem blood dextropropoxyphene concentrations have been measured, and there are many reports of deaths involving dextropropoxyphene alone and in which the blood dextropropoxyphene concentrations were less than 1 microgram per millilitre, as discussed above (paragraph 19).

Signature: R. J. Haragan Signature witnessed by:

Continuation of Statement of

29. There is nothing in the information available to me that leads me to question the conclusions reached by Dr ALLAN (his statements of 21 July 2003, 18 August 2003, and 17 September 2003) and by Dr HUNT (his statement of 25 July 2003).

I would be willing to revise aspects of this statement should more information become available to me.

R. J. Flanagan .

RJ Flanagan

Signature:

R. J. Flanagan

Signature witnessed by: