UNIVERSITY OF LEICESTER

EAST MIDLANDS

FORENSIC PATHOLOGY UNIT

POST-MORTEM EXAMINATION REPORT

FP3256

DAWN STURGESS

FULL REPORT

PATHOLOGIST:	**PROFESSOR G N RUTTY**
REVIEW PATHOLOGIST:	**DR FH HOLLINGBURY**
CORONER:	**MR D RIDLEY**
POLICE FORCE:	**THAMES VALLEY POLICE**
IDENTIFICATION BY:	**IDENTIFICATION BAG TAG**

LEVEL 3 ROBERT KILPATRICK BUILDING

LEICESTER ROYAL INFIRMARY, PO BOX 65, LEICESTER, LE2 7LX

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STATEMENT OF WITNESS

(Section 9 Criminal Justice Act 1967 and Rule 16.2 Criminal Procedure Rules)

STATEMENT OF: Professor Guy N Rutty

DATE OF BIRTH: Over 18 years

This statement consisting of 72 pages 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 would be liable to prosecution if I have wilfully stated anything which I know to be false or that I do not believe to be true.

Signature : PD Date : 29 November 2018

PRIVATE AND CONFIDENTIAL, NOT TO BE DISCLOSED TO ANY PERSON WITHOUT THE CONSENT OF HM CORONER

POST-MORTEM EXAMINATION STATEMENT

FP3256

DAWN STURGESS

CAUSE OF DEATH

Ia Post cardiac arrest hypoxic brain injury and intracerebral haemorrhage

Ib Novichok toxicity

CONFLICT OF INTEREST

None.

EXAMINATION STANDARDS

Autopsy examinations at the East Midlands Forensic Pathology Unit are undertaken in line with the following standards (application of which in whole or part is case dependent):

1. Codes of Practice and Performance Standards for Forensic Pathologists in England, Wales and Northern Ireland. Royal College of Pathologists, 2012.
2. Post mortem cross sectional imaging guidance from the Royal Colleges of Radiology and Pathology, 2012.

3. Chief Coroner guidance on post mortem scanning, 2013.
4. Standards for Coroner's pathologists in post-mortem examinations of deaths that appear not to be suspicious. Royal College of Pathologists, 2014.
5. Information to be included in the 'history' section of a forensic pathologist's report. Forensic Science Regulator, 2014.
6. The use of time of death estimates based on heat loss from the body. Forensic Science Regulator, 2014.
7. Legal issues in Forensic Pathology and tissue retention: issue 3 guidance. Forensic Science Regulator, 2014.

STANDARD TERMINOLOGY

Where the following terminology is used within this report, it should be interpreted as per the Istanbul Protocol [Chapter V, Section D, Para 187 (a) - (e)], United Nations: New York & Geneva, 2004, which has been modified to include non-trauma pathology:

'Not consistent'	The lesion could not have been caused by the mechanism / pathology described.
'Consistent'	The lesion could have been caused by the mechanism / pathology described, but it is non-specific and there are many other possible causes.
'Highly consistent'	The lesion could have been caused by the mechanism / pathology described, and there are a few other possible causes.
'Typical of'	There is an appearance that is usually found with this type of mechanism / pathology, but there are other possible causes.
'Diagnostic of'	This appearance could not have been caused in any way other than that described.

PROFESSIONAL BACKGROUND

I am a Bachelor of Medicine and Bachelor of Surgery and have a Medical Doctorate. I am a Fellow of the Royal College of Pathologists and hold the Royal College of Pathologists Diploma in Forensic Pathology. I am a Founding Fellow of the Chartered Society of Forensic

Sciences. I am a Founding Fellow of the Faculty of Forensic and Legal Medicine at the Royal College of Physicians and have held (2015-2016) the David Jenkins Chair of the Faculty. I hold the Foundation Chair in Forensic Pathology at the University of Leicester where I am Chief Forensic Pathologist to the East Midlands Forensic Pathology Unit. I am an Honorary Consultant in Histopathology to the University Hospitals of Leicester NHS Trust and I am a Home Office Registered Forensic Pathologist, having been placed on the Home Office Accredited Register in 1996. I was awarded the Chao Tzee Cheng Visiting Professorship of the National University of Singapore in 2015. I am the Responsible Officer to the Home Office Pathology Delivery Board and the Department of Justice, Northern Ireland.

I have served as an elected member of Council of the Royal College of Pathologists and have acted as Chair and member of the Forensic Pathology Specialist Advisory Committee. I have sat on the Academic Committee of the Faculty of Forensic and Legal Medicine at the Royal College of Physicians and have been a member of their Research Committee having been the Foundation Chair of the committee. I have been a member of the Pathology Delivery Board for Forensic Pathology for the Home Office. I am a member of the Netherlands Board of Court Experts Advisory Committee for Standards for Forensic Pathology having been the first international forensic pathologist to be awarded Netherlands Forensic Pathology Court Registration (awarded 2015). I am the past Chair (office held at different levels 2014-2016) of the International Society of Forensic Radiology and Imaging and past Chair of the UK National Post Mortem Radiology Imaging Board. I am the Chair of the Scientific Advisory Board of the Ludwig Boltzmann Institute for Clinical Forensic Imaging. I am an Associate Fellow of the Higher Education Academy.

My principal work relates to the provision of forensic pathology services to HM Coroners and police forces of the East Midlands. I also provide forensic pathology services to other police forces of the United Kingdom as well as opinion work for both prosecution and defence for solicitors and police forces alike. I provide forensic pathology and mass disaster services to police forces and countries internationally. I undertake research and teaching (undergraduate, postgraduate, medical, paramedical and manage a Royal College and Home Office approved training centre for Forensic Pathology) within my academic role and have published over 293 publications including original peer reviewed papers, review articles, editorials, case reports, letters and abstracts (those related to national and international meetings), one paper of which I understand is currently ranked in the top 1% of all papers published in the world in my discipline area, as well as editing 10 autopsy related books with further books in production. I

have authored 34 book chapters (including re-editing of previously published chapters) and assisted with the writing of crime based fictional novels. I was the founder Editor-in-Chief of the International Forensic Journal, *Forensic Science, Medicine and Pathology* which I edited until December 2008 and have acted as guest editor and associate editor to the Journal of Forensic Radiology and Imaging. I am a member of speciality journal editorial boards. I am an examiner for the Royal College of Pathologists for forensic pathology.

I hold membership of appropriate forensic pathology, forensic science, histopathology and radiological associations and societies. I sit/have sat as an advisor to both association and governmental bodies in relation to forensic pathology developments and services, including the Policy Advisory Committee of the British Association in Forensic Medicine, the Home Office in relation to contaminated mass fatalities, the Department of Health in relation to forensic and mass fatality radiology and the National Police Improvement Agency Missing Person's Bureau. I have acted as Chair of the Scientific Advisory Committee of the International Commission on Missing Persons (ICMP). I have acted as Deputy Chair of the Pathology and Anthropology Working Group of the Steering Committee for Disaster Victim Identification of Interpol. I am the lead for the Forensic Imaging for this Working Group.

I received a Metropolitan Police Assistant Commissioners Commendation for my work with the European Commission funded exercise, Operation Torch in 2008. I was awarded the Member of the Order of the British Empire (MBE) in the Queen's Birthday Honours List, June 2010 for services to the police and counter terrorism.

I am a volunteer Response Doctor for East Midlands Ambulance Service (EMAS), being a member of EMICS. I hold the Certificate of the Electronic Pre Hospital Emergency Care Course (E-phec) of the Royal College of Surgeons of Edinburgh whom I am a member of the Faculty of Pre-Hospital Care. I am a member of the British Association for Immediate Care (BASICS). I hold certification in Advanced Adult (ALS) and Basic Paediatric Life Support (PLS), as well as Advanced Trauma Life Support (ATLS). I am a UK Resuscitation Council accredited Immediate Life Support (ILS) instructor.

Finally, I was the pathologist who undertook the pathological examination of the remains of King Richard III, being the principal pathological author of the principal paper that described the injuries that he sustained and proposing the most probable cause of death.

My full curriculum vitae can be provided on request.

EXAMINATION DETAILS

Under the authority of:	Mr D Ridley,	
	HM Senior Coroner for Wiltshire and Swindon	
Location:	[redacted] Designated mortuary	
Name of deceased:	Dawn STURGESS	
Date of birth:	18th June 1974	**Age:** 44 years
Address:	John Baker House, 16-18 Rollestone Street, Salisbury	
Scene of incident:	9 Muggleton Road, Amesbury	
Date of death:	8th July 2018 **at**	20:26 hours
Examination date:	17th July 2018	
Start:	13.20 hours	**Finish** 00.10 hours (18th July 2018)
Identification by:	Identification bag tag	

PRESENT DURING EXAMINATION

Professor G N Rutty, Forensic Pathologist

Dr Philip Lumb, Forensic Pathologist

[redacted] duty Anatomical Pathology Technologist

[redacted] QM73, Organisation for the Prohibition of Chemical Weapons

Others : Exhibits Officers; Photographers; representatives of Thames Valley Police, Dstl, Regional HART Team and British Army

CLINICAL HISTORY

The information contained in the section entitled CLINICAL HISTORY, is my interpretation of the information that was given to me prior to the autopsy examination. This information may, or may not be, factually correct and may alter during the police investigation subsequent to the end of the autopsy examination.

On the authority of Mr D Ridley, HM Senior Coroner for Wiltshire and Swindon and at the request of Thames Valley Police, I attended the [redacted] designated mortuary on 17th July 2018 to undertake an independent autopsy on the body of the deceased, Dawn Sturgess.

Accompanying me during the examination was Dr Philip Lumb, Home Office Registered Forensic Pathologist from the Manchester Group Practice. He was instructed by HM Senior Coroner to be present throughout the autopsy examination and to provide a second independent report concerning the autopsy findings and death of Dawn Sturgess. I can confirm that Dr Lumb and I undertook the examination together, and that I have not had sight of his independent report.

Prior to the autopsy examination I attended a virtual briefing via telephone conferencing on 10th July 2018 between 14:02 hours and 15:30 hours, hosted by Mr Keith Asman of Counter Terrorism Policing South East. I then attended a second meeting on 12th July 2018 at [redacted] a police building [redacted] at 14:00 hours to further discuss the autopsy process and to collect up to date personal protective equipment. At this meeting I was shown a photograph of a dispensing bottle which comprised a small bottle with an elongated dispensing tube. I was also informed that the nerve agent, Novichok, had been identified to the deceased's nose, mouth, face and hand areas.

From these meetings I understood that Dawn Sturgess was a 44-year old female who lived at John Baker House, 16-18 Rollestone Street, Salisbury. I understood that she became unwell at the scene address on the morning of 30th June 2018. I understand that the ambulance service was alerted to a female at the scene who was in respiratory distress. During the ambulance journey to the scene I understand that she went into cardiac arrest. The ambulance service, as well as a HEMS team attended the scene address, providing her with resuscitation. I understand that a ROSC (return of spontaneous circulation) was established. She was taken to Salisbury hospital where despite treatment she died on 8th July 2018.

To assist me in providing this report I have been provided with a number of documents which are listed in the section entitled "Unused Material".

I have been provided with her General Practitioner Printout Summary. Although I have read the entire General Practitioners records I disclose, in keeping with General Medical Council guidance, the past medical history which I believe is relevant to her death and findings that could be identified at the autopsy examination only.

The deceased was listed within the notes as measuring 1.55 m in height and weighing 53 kg. She had a past medical history of an excess intake of alcohol which had started in 2000 and progressively increased to 2016. She was also known to smoke. She had had a previous caesarean section and was fitted with an intra-uterine contraceptive device in 2009. In 2013

she had left sided musculoskeletal pain and a left sided atypical pneumonia. She was known to have anxiety and depression and had had an unwitnessed fall with a head injury in 2017, possible kidney problems in 2018, and an injury to her left eyebrow in 2018. In 2018 she had an episode where she suffered a right humerus (upper arm bone) supra condyle fracture, bruises to her face, severe hypo-electrolyte disturbance and a Clostridium difficile infection. Finally, in 2018 she had suffered from swollen ankles with a skin rash and pitting oedema.

From the ambulance service associated documents I understand the following:

The statement of Keith Coomber suggests that a male in the property informed the ambulance staff that she had complained of a headache and went and had a bath. He had heard a loud funny noise and "found her unresponsive, a possible seizure". He phoned 999 and got her out of the bath. The call was received on 30th June 2018 at 10.14 hrs with dispatch of a resource at 10.16 hrs, who went mobile at 10.17 hrs, attending at 10.30 hrs. At 10.33 hrs her pupils were noted to be both 1 mm diameter and sluggish. She was in asystole. She defecated during resuscitation. There were also faeces noted to be present on the stairs although it is unclear to me when this occurred. Whilst at the scene she was initially provided with basic life support (BLS), followed by advanced life support (ALS). A return of spontaneous circulation (ROSC) was achieved at 10.50 hrs. They left the scene at 11.50 hrs and arrived at hospital at 12.08 hrs.

From a photocopy of the medical notes I understand the following:

A pre-alert was received by the hospital informing them of the deceased's collapse, asystole and then ROSC. Post ROSC she was bradycardic (slow heart beat) with a heart rate 42 bpm, hypotensive with a low blood pressure of 75/45 mmHg, oxygen saturation at 92% and glucose 10.0. The notes also suggest a history of feeling unwell with a headache and that she may have taken some medications prior to a collapse in the toilet and going into cardiac arrest. The pupils were recorded as been 1 mm diameter and sluggish. The possibility of a drug reaction or illicit drug (heroin or cocaine) overdose was considered. It was noted that she was resistant to normal treatment.

A head computed tomography (CT) scan was undertaken at 12.34 hrs as the possibility of a subarachnoid haemorrhage (bleed over the brain) was considered. This was reported as showing normal ventricles, unremarkable parenchyma and no intracranial haemorrhage. A chest x-ray at 13.35 hrs showed inflammatory change to both lower lobes. An abdominal x-ray at the same time showed possible slightly dilated small bowel loops due to gas. At 17.40 hrs the notes suggest no evidence of aspiration. At 18.00 hrs she was still bradycardic and

noted to have a large amount of saliva production, pinpoint pupils, and diarrhoea although this combination was not, as I understand it, considered significant at the time.

On 1st July 2018 her ECG (heart trace) showed prolonged QT duration with T wave inversion. At 11.50 hrs she still had pinpoint pupils. The toxicology showed no paracetamol to be present and a chest x-ray showed new right lower lobe consolidation suggestive of aspiration in the clinical context. The possibility of a drug overdose or organophosphate toxicity was raised, and toxicology samples were sent to Birmingham. At 23.30 hrs she was started on Pralidoxine due to suspicion of organophosphate overdose.

On 2nd July 2018 at 18.45 the acetylcholinesterase (AchE) results are entered into the notes (Appendix A). This showed very marked AchE inhibition. A toxicology result was also entered which showed the presence of clopidogrel, rocuronium, atropine, cocaine and its metabolite, ephedrine and pseudoephedrine, fentanyl, midazolam, ethyl sulphate, mirtazapine and its metabolite, zopiclone and its metabolite as well as nicotine and its metabolite. An EEG (a test to look at brain activity) was performed which showed very low amplitude with little, if any cerebral activity which was considered to represent diffuse cerebral dysfunction which could be due to severe hypoxic brain injury (brain injury due to a lack of oxygen).

Initially the notes refer to possible organophosphate poisoning. The possible diagnosis of Novichok toxicity was considered. On 3rd July 2018 at 00.10 hrs a blood sample was sent to Dstl Porton Down to further consider the diagnosis.

On 4th July 2018 at 15.25 hrs swabs were taken for tests at Porton Down. Her abdominal ultrasound now showed a fatty liver. She may have had a clonic fit on this day followed on the 5th July 2018 by another seizure. She began to show slight cardiac improvement although her repeat EEG showed very low amplitude which was featureless.

On 5th July 2018 at 10.30 hrs the diagnosis of Novichok poisoning is firmly stated in the notes although there is no documented test result or record, as far as I can ascertain, as to how this diagnosis was informed to the hospital.

On 6th July 2018 at 12.00 hrs the medical notes now advise to change gloves when treating her due to skin contamination with a nerve agent. A CT scan of her head at 19.16 hrs identified hypoxic brain injury. There was a large acute intra-parenchymal haemorrhage involving the left basal ganglia and left frontal lobe with associated vasogenic oedema and mass effect (Appendix B). She was found to have thrombocytopenia (low platelets). On 7th July 2018 at 15.11 hrs she had a repeat head CT scan. This showed that the haemorrhage had extended to

involve the brainstem and posterior fossa with associated cerebellar tonsil displacement into the foramen magnum (opening at the base of the skull).

On 8th July 2018 her chest x-ray at 17.14 hrs showed bilateral basal consolidation with pleural effusions. She died on this day. There is then a test result entered on the 12th July 2018 which corrected the original AchE result (Appendix A).

Finally, after the autopsy, on 26th July 2018 a meeting was held at the East Midlands Forensic Pathology Unit at which I was informed that the bottle I had previously been shown an image [redacted] In comparison the known lethal dose of the agent VX, [redacted] is 3-8 mg. It is my understanding that it is thought that the Novichok was dispensed from this device to cause a dermal absorption route to Dawn Sturgess.

SCENE

I have not attended the scene of her collapse.

IDENTIFICATION

The body was identified to me as that of Dawn Sturgess of John Baker House, 16-18 Rollestone Street, Salisbury by means of an identification tag numbered WA166551 which was present within the [redacted] body bag [redacted]. This was identified to me by a Thames Valley Police Officer who attended the autopsy examination. He informed me that he had placed the tag within said wallet. This tag was photographed and retained as an exhibit (GR1). Hospital identification bracelets were present to the right lower arm (x 2) and left ankle (x 1) which I personally checked. Again, these were all photographed.

AUTOPSY EXAMINATION

The autopsy examination was undertaken at [redacted] the designated mortuary [redacted] on 17th July 2018. Due to the suggestion that the deceased had been exposed to Novichok the examination was undertaken as a so called "Chemical, Biological, Radiological and Nuclear" (CBRN) examination using appropriate personal protective equipment (PPE). Prior to the examination

a step by step process map detailing the order of procedures and sampling strategy was developed and agreed. This was modified by myself and Dr Lumb, with agreement by the investigating police force, during the examination of the deceased.

The contents of the external and internal examination have been formulated from a combination of the notes made by Dr Lumb at the time of the examination and, with agreement at the time of the examination with Dr Lumb due to the nature of the examination and the PPE worn, a subsequent review of the autopsy photographs.

EXTERNAL EXAMINATION

Height: Not formally measured

Weight: Not formally measured

Body Mass Index: Not calculated

The body was that of a white adult female whose general appearance was in keeping with the stated age of 44 years. The hair was white/blonde in colour and measured up to 15 cm in length at randomly measured sites. The armpits had been shaved. Pubic hair was present. No glasses or contact lenses were apparent. The eyes appeared blue/green in colour. The right eye showed positional congestion. There was no facial cyanosis and there were no petechiae. The earlobes were free. The right earlobe was pierced once, the left twice. The nose was palpably intact. Natural teeth were present to the mouth. There were historical losses of the upper jaw teeth. The finger and toe nails did not protrude beyond the digit tips. They were dirty. There were flecks of possible blue nail varnish to the right thumb, middle and little finger nails. A similar appearance was seen to the left little finger nail. Cigarette tar staining was present to the right index and middle fingers. There were abdominal striae. The female external genitalia were generally oedematous due to the presence of an indwelling urinary catheter. The anus was dilated due to the presence of a medical flatus tube. Both breasts were normal.

Rigor mortis was absent. Lividity was established in a posterior distribution extending onto the right side of the face. There was slight green discolouration of the abdomen. There was generalised oedema of the soft tissues and muscles. There were two areas of pressure drying / indentation over the mid thoracic and sacral areas of the midline of the back.

Clothing and coverings

1. [redacted]
2.
3. Transportation coverings
4.
5.
6.
7.

Jewellery

None

Tattoos

1. A monochrome tattoo whose design had faded and was difficult to discern was present to the outer aspect of the left upper arm near the shoulder.
2. A 4 cm x 4 cm polychromatic tattoo of a rose with wings design was present to the right side of the lower back area.

Marks and equipment from medical intervention

1. Urinary catheter and attached collection bag
2. Flatus tube
3. Endotracheal tube with cradle. A yellow tube was also present in association with the endotracheal tube.
4. A 0.5 cm diameter bruise was present to the lower lip related to medical treatment.
5. Right sided nasogastric tube.
6. Cotton wool was present to the right and left ear canals.
7. Absorbent dressing taped to left and right hip areas.

8. ECG tabs were present to the right and left clavicular areas of the chest as well as to the left costal margin.
9. A four-tailed left subclavian line.
10. Three needle puncture bruises to the left side of the abdomen.
11. Two needle puncture sites to the left thigh with a yellow needle inserted to the lateral aspect of the upper leg.
12. Needle puncture mark back of right hand.
13. A pink cannula to the anterior aspect of the left lower arm above the level of the wrist.
14. A left wrist radial line.
15. Two needle puncture marks to the midpoint of the left upper arm.
16. Two needle puncture marks to the lateral anterior aspect of the bend of the left arm.
17. A needle puncture mark to the back of the left hand.
18. Needle puncture mark lateral right upper leg.
19. Two needle puncture marks to the top of the right ankle area with a further one to the top of the right foot.
20. Needle puncture site to the medial aspect of the left ankle.

Old marks and injuries

An "old injury" is traditionally considered as an injury greater than 48 hours of age.

1. An oblique scar to the bridge of the nose.
2. A number of small scars to the posterior aspect of the right wrist extending onto the back of the right hand. A prominent scar was present to the length of the right thumb.
3. A number of scars up to 1cm length to the anterior aspect of the right lower arm.
4. There was generalised scarring up to 2 cm long to the back of the right lower arm.
5. A scar typical of a TB immunisation site was present to the lateral aspect of the left upper arm.
6. A 2 cm long scar to the posterior aspect of the left lower arm close to the wrist with further punctate scars between the bend of the arm and the wrist.

7. A 2 cm long scar to the posterior aspect of the left hand.
8. A horizontal suprapubic scar.
9. A group of scars to the anterior aspect of the right lower leg in the upper third.
10. An interrupted healing abrasion 4 cm x 2 cm to the anterior aspect of the right lower leg.
11. Generalised punctate scarring to the top of the right foot.
12. Healing scratch 8 cm long to the medial aspect of the left upper leg just above the knee.
13. Scars up to 5 cm long were present to the anterior aspect of the left lower leg.
14. Two healing interrupted scratch abrasions up to 3 cm long to the lateral aspect of the left lower leg above the level of the ankle.
15. There was generalised faint scarring to the anterior aspect of the ankle.
16. A line of five possible areas of scarring to the left of the mid-thoracic area of the back.

Fresh injuries and changes

A "fresh injury" is traditionally considered to be an injury less than 24 hours to 48 hours old. No fresh injuries other than those associated with medical intervention were identified. There was no blistering or marks to the skin of the neck, wrists or palms of the hands.

Body diagrams completed by Dr P Lumb at the time of the post-mortem examination are reproduced in Appendix C.

INTERNAL EXAMINATION

Cardiovascular System

Pericardial sac:	**Small amount of fluid (not measured).**
Heart:	Weighed 307 g. The heart was of normal shape and size. There was no macroscopic suggestion of alcoholic or drug induced cardiomyopathy.
Right atrium:	Normal.
Left atrium:	Normal.
Right ventricle:	**Appeared dilated.**

Left ventricle: **Apparent trans-circumferential endocardial pallor with slight haemorrhage to the posterior free wall at the junction with the septum. It appeared dilated.**

Endocardium: Normal.

Papillary muscles: Normal.

Chordae tendineae: Normal.

Valves: Normal.

Coronary arteries: The right coronary artery was dominant. All three coronary artery vessels were small calibre vessels. There was no apparent overt calcification and no significant atheroma was identified. No thrombus was identified

Aorta and principal branches: Moderate, non-ulcerated atheroma of the entire length extending into the major branches.

Pulmonary arteries: Normal.

Carotid arteries: Free of occlusive atheroma.

Venous system: Normal.

Respiratory System

Pleural cavities: **There were adhesions from the anterior and lateral aspects of the left upper lobe to the parietal pleura. Approximately 200 mls of clear serous fluid was present to the left pleural cavity. The right appeared normal.**

Hemidiaphragms: Normal.

Hyoid bone: Intact.

Laryngeal cartilages: Intact.

Trachea and main bronchi: **Contained mucoid secretions with slight erythema of the mucosa except where the endotracheal tube balloon had been in contact with the mucosa.**

Lungs: **Weighed: right 1036 g, left 988 g. All lobes of both lungs showed the same appearance. There was generalised congestion and pulmonary oedema. There was no apparent focal lesion.**

Gastrointestinal System

Peritoneal cavity:	**A small quantity of clear ascites (volume not measured). There were some adhesions in the area of the suprapubic abdominal scar.**
Tongue:	Normal.
Mouth:	Normal.
Pharynx:	Normal.
Oesophagus:	Normal.
Stomach:	Empty with a small amount of mucous only. The mucosa was normal.
Small intestines:	Normal. No dusky appearance and no suggestion of infarction. Contained small intestine content.
Mesentery:	Normal.
Appendix:	Present.
Large intestines:	No evidence of infarction. Contained bowel contents.
Rectum:	Normal.
Anus:	**Dilated due to flatus tube.**
Liver:	**Weighed 1953 g. The capsular surface was smooth and transparent. The cut surface showed diffuse fatty change.**
Gall bladder:	Normal.
Common bile duct:	Not examined
Pancreas:	Weighed 240g (not trimmed of excess fat). Normal.

Lymphoreticular System

Spleen:	**Weighed 100 g. The cut surface was diffluent.**
Lymph nodes:	**The hilar lymph nodes were enlarged to both lungs, measuring up to 0.5 cm diameter.**
Thymus:	Normal adult atrophy.

Endocrine System

Thyroid:	Normal.
Adrenals:	Normal.

Pituitary: Fixed for histological examination. Macroscopically normal.

Genitourinary System

Kidneys: Weighed: right 244 g, left 178 g. Both capsules stripped easily to reveal smooth surfaces. There was good corticomedullary distinction. There were no areas of cortical infarction.

Ureters: Normal.

Bladder: **Empty with mucosal catheter change.**

Uterus & cervix: **The uterus contained an intrauterine contraceptive device.**

Fallopian tubes & ovaries: Only the right ovary was identified.

Central Nervous System

Scalp: **A solitary round bruise 1 cm in diameter was present to the anterior superior aspect of the temporal muscle on the right side**

Meninges: No evidence of extradural, subdural or subarachnoid haemorrhage.

Vessels at base of brain: Normal.

Brain: **Weighed 1180 g. The brain showed congestive cerebral surfaces with prominent vasculature. The brain was diffusely hypoxic causing difficulty with removal and examination due to its soft consistency. There was a left sided basal ganglia haemorrhage as seen on the clinical CT scan. There was apparent haemorrhage within the brain stem.**

Eyes: Not examined.

Middle ears: Not examined.

Spinal cord: Not examined.

Musculoskeletal System

Cervical muscles: Anterior: Grossly normal.

Posterior: Not examined due to natural of examination.

Skull:	**Diffuse thickening. No fracture.**
Facial bones:	Normal
Facial structures:	The left masseter muscle and parotid gland were examined and were normal. There was no palpable fracture.
Spine:	Macroscopically normal.
Long Bones:	Palpably intact.
Soft tissue:	**Soft tissue and muscle oedema. There was a bruise within the left groin related to medical intervention.**
Ribcage:	**There was a 4 cm x 3 cm bruise to the left chest wall overlying the left 6th rib. There was callus thickening of the right 2nd to 6th and left 5th costochondral junctions. There were fresh fractures of the right 3rd rib laterally, and 2nd to 6th anteriorly without bleeding as well as the left 2nd and 3rd ribs anteriorly, and 2nd to 6th ribs at the costochondral junctions again without associated bleeding.**
Sternum	**Fracture at the level of the 3rd and 4th ribs.**

SPECIMENS RETAINED

With the authorisation of HM Coroner and following discussion with the Senior Investigating Officer, the following were retained:

Histology: Thirty-six pieces of tissue were placed in plastic cassettes and taken first to Porton Down prior to release to the East Midlands Forensic Pathology Unit where they were processed in wax and three sets of thirty-five glass slides cut. In addition seven sections were cut for histochemical and forty-four for immunohistochemical staining. To date the glass slides and wax blocks remain in the East Midlands Forensic Pathology Unit. The only whole organ retained was the pituitary gland, which was processed to wax in its entirety.

Biological samples: A number of samples were requested by, and provided to Thames Valley Police, Dstl and the Organisation for the Prohibition of Chemical Weapons as listed below in the section entitled "Exhibits".

Clinical Notes: Copies of General Practitioners Summary and medical notes retained with the case file.

Photographs: Taken by Thames Valley Police.

Video The autopsy examination was videoed by Thames Valley Police

X-rays: Copies of the medical CT scans and radiology reports are retained in the case file.

Crimelite: Not used.

The biological samples were retained under the authority the Police and Criminal Evidence Act (PACE).

EXHIBITS

During the examination I seized the following exhibits. Please note that not all exhibit numbers were used. Some were discarded as the process map was adjusted during the examination.

Barrel 1 (F's) (triple bagged) : [redacted]

GR/48 – right sole

GR/50 – left upper leg muscle sample

GR/52 – left lower leg muscle sample

GR/54 – left sole

Barrel 3 (triple bagged): [redacted]

GR/12 – nail clippings

GR/20 – skin right palm

GR/24 – skin left palm

GR/27 – head hair sample

GR/29 – right upper arm muscle sample

Barrel 4 (double bagged):

GR/56 – blood sample leg

519 GR/60 – blood sample leg

520 GR/61 – blood sample leg

521 GR/82 – stomach contents

522 GR/86 - brain

523 GR/91 – kidney

524 GR/98 – liver

525 GR/137 – histology pot [redacted]

526 GR/138 – heart conducting [redacted]

527 RO – pathologists notes [redacted]

528 **Barrel 5 (double bagged):** [redacted]

529 GR/81 – vitreous humor

530 **Russian doll (triple bagged):**

531 GR/1 – body seal [redacted]

532 GR/2 – body tag [redacted]

533 GR/3 – body tag [redacted]

534 GR/4 – body tag [redacted]

535 GR/4A – body tag [redacted]

536 GR/5 – swab front right wrist

537 GR/6 – swab back right wrist

538 GR/7 – swab front left wrist

539 GR/8 – swab back left wrist

540 GR/9 – swab neck right

541 GR/10 – swab neck left

542 GR/11 – control

543 NCL/1 – set of fingerprints [redacted]

544 **Barrel 1 (E's) (triple bagged):** [redacted]

545 GR/14 – skin right front wrist

546 GR/16 – skin left front wrist

547 GR/31 – right upper arm muscle sample

548 GR/34 – right lower arm muscle sample

549 GR/36 – left upper arm muscle sample

550 GR/42 – left lower arm muscle sample

551 GR/43 – left lower arm muscle sample

552 GR/45 – right upper leg muscle sample

553 GR/47 – right lower leg muscle sample

554 GR/49 – right sole

555 GR/51 – left upper leg muscle sample

556 GR/53 – left lower leg muscle sample

557 GR/55 – left sole

558 **Barrel 1 (F's) (triple bagged):**

559 GR/13 – skin right front wrist

560 GR/15 – skin left front wrist

561 GR/30 – right upper arm muscle sample

562 GR/33 – right lower arm muscle sample

563 GR/35 – left upper arm muscle sample

564 GR/44 – right upper leg muscle sample

565 GR/46 – right lower leg muscle sample

TOXICOLOGY

Toxicological samples were examined whilst the deceased was alive in hospital. The details of the results of these examinations are detailed in the section entitled "Clinical History".

HISTOLOGY

Three different examinations were undertaken with the tissue retained at autopsy and released by Dstl Porton Down to the East Midlands Forensic Pathology Unit.

The first was a traditional haematoxylin and eosin examination. This yielded the following results:

1. Skin from right wrist: normal.
2. Skin from left wrist: normal.
3. Right upper arm muscle: normal.
4. Right lower arm muscle: normal.
5. Left upper arm muscle: A degree of vacuolated muscle cells with a degree of pink change. This was considered a non-specific change due to muscle necrosis.
6. Left lower arm muscle: normal.
7. Right upper leg muscle: normal.
8. Right lower leg muscle: Some vacuolated muscle cells present.
9. Right sole of foot: The overlying skin was normal. There was a degree of vacuolation of the deep muscle cells.
10. Left upper leg muscle: A degree of muscle vacuolation.
11. Left lower leg muscle: A degree of muscle vacuolation.
12. Left sole of foot: The overlying skin was normal. There was a degree of vacuolation of the deep muscle cells.
13. Pituitary: Good generalised preservation with focal necrosis.
14. Submandibular gland: Normal.
15. Parotid gland: Normal.
16. Left masseter muscle: Normal salivary gland with muscle showing vacuolation.

17. Right sternomastoid muscle: Focal vacuolation.

18. Left sternomastoid muscle: Possible focal disruption of muscle fibres with a degree of vacuolation.

19. Intercostal muscle: Normal.

20. Right hemidiaphragm: A degree of vacuolation observed.

21. Left hemidiaphragm: Normal.

22. Liver: Poor preservation / necrotic with focal fatty change. There was micronodular cirrhosis with an increase in chronic inflammatory cells in the portal tracts which show portal - portal bridging and bile duct proliferation.

23. Large bowel: Post mortem necrosis.

24. Bladder: Catheter effect.

25. Pancreas: Autolysed.

26. Spleen: Autolysed.

27. Kidney: Within normal limits for age. No apparent myoglobin.

28. Kidney: Within normal limits for age. No apparent myoglobin.

29. Left lung lower lobe: Marked pulmonary oedema. There was an increase in generalised cellularity as can be seen in those who smoke. A degree of acute inflammation within the small airways.

30. Left upper lobe: Similar to 29 with additional proteinaceous debris.

31. Right upper lobe: Similar to 29.

32. Right lower lobe: Solid areas suggest alveolar thickening. There was some focal alveolar haemorrhage present.

33. Coronary artery samples x2: No significant stenosis, vasculitis or occlusive thrombus.

34. No cassette 34 was received. It would be my normal practice to place all coronary artery specimens into one cassette so although there were two cassettes for the coronary arteries I believe there was only one despite the documentation to suggest otherwise.

35. Brain: The section of the cerebellum showed subarachnoid and parenchymal red cell collections. The cerebral cortex section confirmed the presence of cortical haemorrhage and hypoxic brain injury.

36. Brain: As per 35.

The second examination used an immunohistochemical approach. This yielded the following results:

1. Anti-Acetylcholinesterase (AchE) antibody (abcam ab78228 https://www.abcam.com/acetylcholinesterase-antibody-ab78228.html last visited November 2018). This showed positive granular staining within the tissues indicating the presence of AchE. Positive staining was seen to red cells within the blood vessels as well as to the striations of the skeletal muscle samples. Please note that this antibody indicated the presence of, rather than functionality of AchE.

2. Anti-Acetylcholine (Ach) antibody (Novus Biologicals NB100-64656 https://www.novusbio.com/products/acetylcholine-antibody_nb100-64656 last visited November 2018). This antibody was applied to two representative skeletal muscle samples. This showed positive staining to red cells within the blood vessels as well as to the striations of the skeletal muscle samples. Compared to the control sample the staining was subjectively assessed to be more positively expressed in the red cells of the subject samples compared to the control samples.

The third examination used a histochemical approach. This yielded the following results:

1. The Cholinesterase technique, thiocholine method (Gerebtzoff 1959, Ref., a) was applied to the same two skeletal muscle representative samples as per the Ach immunohistochemistry. This method tests functionality of AchE. It is a test that is sensitive to the time of fixation of the material and the pH of the solutions used and thus difficult to undertake (Ref., b). The control samples showed granular dot staining in the blood vessels. Granular dot staining was observed between the skeletal muscle cells. A further positive control (frozen section) was assessed that showed similar granular black deposits but this time in association with the cells. No staining was seen in either the blood vessels or the muscle of the subject samples.

Reference

a. Cholinesterase technique, thiocholine method. In Theory and Practical Histological Techniques. Eds, Bancroft JD, Stevens A. Churchill Livingston, London, 1977, page 298.

b. Moore EJ, Petty CS. A note on cholinesterase activity on post mortem tissues. J Histochemistry Cytochemistry, 1958; https://doi.org/10.1177/6.5.377

Representative photographic images exampling the findings of the three examinations were taken by Dr M Biggs, Clinical Associate Professor, East Midlands Forensic Pathology Unit and are found within Appendix D.

CARDIAC PATHOLOGY

Selected tissue from the heart was retained at the autopsy examination and referred to Professor S.K. Suvarna at the Royal Hallamshire Hospital, Sheffield. A copy of Professor Suvarna's report is found within Appendix E.

In his statement, Professor Suvarna concludes that:

"Conclusions.

- *The majority of tissues are considered within normal limits for this individual.*
- *There are some minimal chronic fibrotic changes, likely not relevant to the cause of death.*
- *There are some ultrastructural mitochondrial changes, which likely to reflect aspects of the cardio-respiratory arrest, rather than the Novichok agent. However, it is recognized that there is no database on the morphological effects of this toxin on normal human myocardial parenchyma for cross-comparison".*

Representative photographic images exampling the findings of the electron microscopy examination were taken by Professor Suvarna and are found within Appendix F.

SPECIALIST CHEMICAL AGENT ANALYSIS

Samples were taken from the body of the deceased and subjected to analysis at a number of independent laboratories. I have been made aware of the following results:

Organisation for the Prohibition of Chemical Weapons

The OPCW produced a summary report which is available within the public domain at https://www.opcw.org/sites/default/files/documents/S_series/2018/en/s-1671-2018_e_.pdf and is found within Appendix G of this report. From this document the following is extracted:

"8. The results of the analysis of biomedical samples conducted by OPCW Designated Laboratories demonstrate that Mr Charles Rowley and Ms Dawn Sturgess were exposed to and intoxicated by this toxic chemical.
9. During the second deployment, the team collected a sample of the contents of a small bottle that the police seized as a suspect item from the house of Charles Rowley in Amesbury.
10. The results of the analysis of this environmental sample conducted by OPCW Designated Laboratories show that the sample consists of the toxic chemical at a concentration of 97-98%. The sample is therefore considered a neat agent of high purity. The OPCW Designated Laboratories also identified a number of impurities constituting the remaining 2-3% of the sample.
11. The results of the analysis conducted by OPCW Designated Laboratories of environmental and biomedical samples collected by the OPCW team confirm the findings of the United Kingdom relating to the identity of the toxic chemical that intoxicated Mr Charles Rowley and Ms Dawn Sturgess. The toxic chemical compound, which displays the toxic properties of a nerve agent, is the same toxic chemical that was found in the biomedical and environmental samples relating to the poisoning of Sergei and Yulia Skripal and Mr Nicholas Bailey on 4 March 2018 in Salisbury (S/1612/2018, dated 12 April 2018)."

This report however does not name the toxic agent. Although I have requested further details concerning this report and nature of the agent identified, to date I have not been provided with the details I have requested.

Defence Science and Technology Laboratory

Samples were examined by Dstl Porton Down when the deceased was alive and in hospital as well as from samples retrieved during the autopsy examination. A copy of the Dstl report is found in Appendix H. The summary of this report provides the following information;

"***Summary***

This report summaries the analysis undertaken at Dstl on biomedical samples recovered from Dawn STURGESS pre- and post-mortem. Analysis of blood collected on admission to hospital confirms that Dawn STURGESS was poisoned with a Novichok nerve agent. Analysis of blood and tissue samples collected from Dawn STURGESS at the post-mortem demonstrate that butyrylcholinesterase and acetylcholinesterase were close to completely or completely inhibited at the time of death, and that the characteristic marker of the Novichok nerve agent were stoill detectable in her blood. Analysis of liver, kidney and brain tissue samples demonstrates the presence of characteristic Novichok metabolites in all three sample types. Furthermore, very low levels of free intact Novichok were detected in the brain homogenate. These results indicate systemic presence of the Novichok agent."

GENERAL COMMENT ON NOVICHOK

The following is a summary of my understanding of the possible signs, symptoms and pathology that might be expected to occur and be identified in a person exposed to "Novichok". Due to the paucity of available information within the public domain concerning Novichok, I am reliant upon the few references that I have identified concerning Novichok and applying more general principals in relation to chemical weapons from the organophosphate group of compounds and the more commonly available literature concerning patient toxicity due to exposure from contact with insecticides [1-3]. The sources of the information I have relied upon are referenced below.

Novichok is an organophosphate nerve agent chemical weapon from the dihaloforamide group which was developed, as I understand it, during a secret chemical weapon program which started in the Soviet Union in 1982 [4]. More than 100 compounds fall into the Novichok category with Novichok 5 and 7 being the best known. It is highly toxic, thought to be five to ten times more toxic than VX agent, another organophosphate chemical weapon, and reported to be practically impossible to treat. Its exact chemical structure is unknown within the public domain. Similar to VX and sarin, other nerve agent chemical weapons, it would be expected to penetrate uniforms.

Organophosphates irreversibly inhibit the action of Acetylcholineesterase (AchE), the enzyme that breaks down Acetylcholine (Ach), a neurotransmitter synthesised and released at nerve endings in both the central and peripheral nervous system. This inhibition leads to the accumulation of Ach within nerve endings with resulting over stimulation of muscarinic and

nicotinic Ach receptors. It will bind to AchE in red blood cells. The toxicity of Novichok is thought not to rely upon the primary inhibition of AchE but is thought to cause permanent neuropathy, hence conventional nerve agent antidotes may not work.

Organophosphate intoxication may occur through inhalation, ingestion or dermal exposure [1,5]. The diagnosis of exposure is made by analysing plasma and red cell AchE activity. The best diagnostic test is red cell AchE inhibition (RBC-ChE). When RBC-ChE activity is less than 75% of the individuals baseline reading, asymptomatic exposure has occurred. Severe symptoms occur when 70% inhibition is recorded [6]. Another enzyme that can be monitored is butyrylcholinesterase.

Considering that Novichok is alleged to be 5-10 times more toxic than VX, then, similar to VX, signs and symptoms would be expected to occur rapidly after exposure to Novichok. Exposure to VX vapour leads to symptom development within a few minutes to 18 hours [1,6]. Dermal contact with VX would be expected to be lethal if not washed off immediately, with a tiny drop of the agent causing local sweating and muscle twitching. Leikin *et al*,. report that dermal exposure of 6mg of VX would be expected to kill 50% of 70kg adults with symptoms starting within 1-30 mins after exposure [5] although the more commonly quoted LD_{50} for VX is 10mg [7,8]. They also report that even after skin decontamination has taken place, absorption can continue with delayed symptoms due to continued absorption from the inner layers of the skin.

Toxicity manifests in three phases. The first, the nicotinic phase, results in hypertension (raised blood pressure) and sinus tachycardia (fast heart beat). The second phase causes sinus bradycardia (slow heart beat), rhythm disturbances and parasympathetic overstimulation, the latter of which causes nausea, vomiting, defecation, profuse sweating, lacrimation (crying), rhinorrhoea (fluid from the nose), salivation, gastric cramps, urination, miosis (pinpoint pupils), muscle fasciculation (twitching), restlessness, tremors, convulsions, seizures, bronchospasm, bronchorrhea, respiratory distress / failure and loss of consciousness [9,10,11]. Headache is a reported symptom along with eye pain [2,12]. The third phase results in sudden cardiac death.

There is no published information of the cardiac pathology of Novichok. However, the group of nerve agents which Novichok is considered to belong to are known to effect the SA node causing inhibition of the node with resultant bradycardia . Organophosphates in general cause prolongation of the QT interval as well as ST and T wave abnormalities. A number of cardiac arrhythmias may occur as well as ultrastructural abnormalities including mitochondrial changes.

Although organophosphate toxicity can cause acute respiratory failure due to both central and peripheral mechanisms [13], which apart from sudden cardiac death may be a primary cause of death, it is also known to cause a delayed intermediate syndrome which can manifest as pulmonary oedema, haemorrhage, acute respiratory distress syndrome and pneumonitis necessitating ventilatory support [12,14-16].

In terms of the other organs of the body, there is an increased risk of pancreatitis. An organophosphate related autopsy study from India reported the non-specific changes of changes to the brain including herniation with coning, congestion and oedema. Tubular degeneration, cloudy swelling and congestion of the kidneys as well as fatty change of the liver were also reported [16]. In those who survive there are a number of reported long term medical problems associated with organophosphate exposure [17,18].

To date only one incident involving exposure to Novichok in the United Kingdom has been reported in the literature [19]. This incident involved three adults who were exposed to Novichok in Salisbury, England on 4th March 2018. All survived.

References

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COMMENTS

1. The body was that of a white adult female whose general appearance was in keeping with the stated age of 44 years.

2. The body was identified to me as that of Dawn Sturgess of John Baker House, 16-18 Rollestone Street, Salisbury by means of an identification tag numbered WA166551 which was present within the body bag. This was identified by the Thames Valley Police Officer who attended the autopsy examination and informed me that he had placed the tag within said wallet. This tag was photographed and retained as an exhibit (GR1). Hospital identification bracelets were present to the right lower arm (x 2) and left ankles (x 1) which I personally checked. Again, these were all photographed.

3. In my opinion the key elements of the medical history are that the deceased collapsed at home after complaining of a headache for which she had gone to take a bath. She may or may not have taken medication for this. She then went first into respiratory arrest, then asystolic cardiac arrest. Those attending her at the scene noted pinpoint pupils and she defecated during resuscitation. Following ROSC she was bradycardic (slow heart beat) with an ECG in hospital showing prolonged QT duration. She was also noted to have a large amount of saliva production and diarrhoea. Organophosphate toxicity was considered, and the results of her AchE examination showed profound inhibition. The diagnosis of Novichok toxicity was made around 5th July 2018. Her admission head CT scan had importantly shown no acute or chronic pathology to explain the clinical presentation of a headache, specifically no evidence of an intracranial or intracerebral bleed. However, the repeat head CT scan on 6th July 2018 showed hypoxic brain injury with an acute left sided intracerebral bleed. This had extended on the third scan on 7th July 2018 to involve the brainstem with associated cerebellar tonsil displacement. She died on 8th July 2018.

4. An autopsy examination was undertaken on the deceased's body under so-called "CBRN" conditions. Two Home Office Registered forensic pathologists from different geographic areas of the country and different group practices undertook the examination. The examination was photographed, videoed and subject to international independent observer observation.

5. The external examination documented a number of marks of medical intervention which have neither caused nor contributed to death.

6. The external examination documented a number of historical scars and healing injuries which do not require further consideration.

7. The external examination identified no fresh marks of injury. Thus, no injuries were identified to the deceased to suggest or support that her collapse had been as a result of a blunt or sharp trauma assault.

8. The internal examination identified pathology to the brain, heart, pleural cavities, lungs, peritoneal cavity, liver, and lymph nodes as detailed within the text of the full report. A number of fractures at various stages of healing were identified to the rib cage and sternum as can arise following cardiopulmonary resuscitation.

9. No natural disease was identified at the autopsy examination or the subsequent histological or cardiac examinations to account for the presenting signs and symptoms or to be considered as her cause of death.

10. Due to period of time that the deceased has survived post ROSC in hospital and the time between death and the autopsy examination the brain's consistency had deteriorated, making it difficult to examine at autopsy. Despite this there are good clinical records in the form of the CT scans that demonstrate that the deceased's collapse was not as a result of an intracranial or intracerebral bleed. Rather the bleed that was demonstrated on CT scanning, and autopsy, developed in hospital on a background of post cardiac arrest hypoxic brain injury. In common with intracranial haemorrhage associated with organophosphate toxicity, for which I have only found one reference referring to a case of subarachnoid haemorrhage [10.1], post cardiac arrest intracranial haemorrhage has, to my knowledge only been reported once in the literature. The paper of Cha *et al*., [10.2] describes similar findings to this case in as much that the first CT scan undertaken 4 hours after ROSC showed no intracranial haemorrhage and yet a repeat scan undertaken 7 days later showed bilateral basal ganglia and thalami haemorrhage with subarachnoid haemorrhage. Thus, I am of the opinion that the deceased has developed a post cardiac arrest intracerebral bleed on a background of hypoxic brain injury which has extended to involve the vital cardiorespiratory centres of her brain and led to her death.

<u>References</u>

10.1 Gokel Y. Subrachnoid haemorrhage and rhabdomyolysis induced acute renal failure complicating organophosphate intoxication. Renal Failure, 2002, 24:6; 867-871.

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11. The deceased had fluid accumulations within her pericardial sac, and pleural and peritoneal cavities as well as generalised oedema (fluid) to her soft tissue and muscle compartments. Although there are a number of papers that suggest that organophosphate toxicity can cause alveolar capillary membrane breakdown leading to oedema of the lung, as well as parenchymal haemorrhage and increased risk of pneumonia, as these findings can also be seen in those not dying of organophosphate poisoning who are in multi-organ failure from other causes, I am of the opinion that these observations, although reported in organophosphate toxicity, are not necessarily specific in their own right to organophosphate toxicity.

12. In life the deceased had a toxicological examination undertaken. This identified a number of therapeutic and non-therapeutic drugs to be present. Although I have not been provided with the levels of the drugs identified, I am not aware that there is any indication to suggest that the deceased's collapse was a direct result of the action of either a therapeutic or illicit drug.

13. I am informed within the clinical notes and from Thames Valley police as well as Dstl that the deceased has been exposed to Novichok, specifically Novichok [redacted] This, as I understand it, is thought to have been through a dermal exposure route following the application of the agent via a dispensing device. [redacted] Based on the known LD_{50} of VX, which is all I have to go on, then if this was VX not Novichok, [redacted] then a single dispensing action could potentially deposit [redacted] the amount of material required to kill 50% of adults via a dermal route.

14. The Dstl report concerning the ante and post-mortem sample analysis informs me that Dawn Sturgess was poisoned with a Novichok nerve agent and that free intact Novichok was still present within her brain at autopsy.

15. I am aware through the open source document produced by the OPCW that they have independently confirmed the presence of a near pure "toxic chemical". Although, to date, I have not been provided with a document which names this toxic chemical or provides me with any further information in relation to the samples I provided to the OPCW at autopsy, although this document does not name the "toxic chemical" it does state in

925 section 11 of the open source report that the chemical is the same as that identified by the
926 United Kingdom. Thus, on this basis I have assumed that they too have identified the
927 presence of Novichok although should this not be the case and I am informed otherwise
928 at a later date I reserve the right to review any aspect of this report and amend any
929 comment I have made within it.

930 16. The immunohistochemistry examination for AchE undertaken at the EMFPU
931 demonstrated the presence of AchE in the samples examined. However, the antibody
932 used identifies the presence of AchE but does not access its functionality. Thus it can be
933 present but non-functional due to the action of Novichok. Unfortunately, no
934 immunohistochemical antibody was available to us, to my knowledge, to test AchE
935 functionality. Thus we resorted to using a historical histochemical stain to test
936 functionality. This proved difficult to undertake, as suggested it would in the historical
937 literature, due to the effect on AchE functionality caused by prolonged tissue fixation and
938 its sensitivity of the pH of the solutions used. However, compared to the control tissue,
939 the results suggested that no functional AchE was present, assuming that the control
940 material result is being interpreted correctly as we have no prior experience of the use of
941 this test. Finally, the immunohistochemical test undertaken to assess the presence of Ach
942 showed subjectively more positive staining for the test material than the control. It is
943 hypothesised that this is the expected result as Novichok should cause a build-up of Ach
944 due to the blocking of AchE.

945 17. Thus, I am of the opinion that the clinical presentation in terms of the signs and
946 symptoms, as well as the in-life laboratory tests and the tests and reports received
947 following the autopsy examination all support that Dawn Sturgess did not collapse or die
948 from a natural medical event, an assault or the result of a therapeutic or illicit drug
949 overdose but rather due to the complications resulting from a cardiac arrest caused by
950 Novichok toxicity. Having been exposed to the nerve agent Novichok, which appears
951 from the information I have been provided to have occurred through a dermal exposure
952 route, and with the knowledge of the expected action of organophosphate nerve agents I
953 would have expected Dawn Sturgess to have deteriorated relatively quickly. It is
954 documented that she first went into respiratory arrest and then asystolic cardiac arrest.
955 Although CPR was successful and resulted in a ROSC, she continued to exhibit
956 organophosphate toxicity post ROSC. Although her cardiac function did begin to show
957 some improvement, she had sustained severe hypoxic brain injury which developed into

an intracerebral haemorrhage. The intracerebral haemorrhage then extended into the vital cardiorespiratory areas of her brain. This was the final pathological process that, in my opinion, led to her death.

CAUSE OF DEATH

Ia Post cardiac arrest hypoxic brain injury and intracerebral haemorrhage

Ib *Novichok toxicity*

INDEX OF UNUSED MATERIAL

Contained within the case file at the time of completing this report was the following material.

1. Photographs of hand written notes and annotated diagrams made during the post-mortem examination.
2. Copy of autopsy contemporaneous notes of Dr P Lumb.
3. Photographs and video of autopsy
4. Draft versions of the report, as well as a file copy of the final report.
5. EMFPU Tissue Retained at Post Mortem form.
6. EMFPU Histology Request form.
7. Handwritten notes re Histology.
8. abcam Product Datasheet – Anti-acetylcholinesterase antibody ab78228.
9. abcam Product Datasheet – Anti-acetylcholine antibody ab34935.
10. List of PM Samples.
11. The result of the Critical Conclusions Check required by Home Office guidelines.
12. Copy of the provisional Body Release Statement.
13. Copy of original and modified Dawn Sturgess Autopsy Process Map.
14. Copy of Counter Terrorism Policing Unit [redacted] – Dressing.
15. Black and white images of heart with handwritten notes.
16. Copy of images of mortuary.

17. Floor plan of mortuary with room index.
18. Record of address and contact numbers for Mr D Ridley.
19. Various notes with telephone contact numbers.
20. Various telephone message slips.
21. Small slip of paper headed "The George" with hand drawn drawings of biceps, leg and lower arm.
22. Email correspondence between Professor Rutty and Wendy Pitts, EMFPU Group Practice Manager.
23. Email correspondence between Professor Rutty and VN513.
24. Email correspondence between Professor Rutty and Phil Lumb.
25. Email correspondence between Professor Rutty and QM73
26. Email correspondence between Professor Rutty and Martin Cook, Salisbury NHS Foundation Trust.
27. Email correspondence between Professor Rutty and David Ridley and Wiltshire & Swindon Coroner's Office.
28. Email correspondence between Professor Rutty and Professor Morgan, Consultant Radiologist, UHL NHS Trust
29. Email correspondence between Professor Rutty and Claire Robinson, Forensic Radiologist, UHL NHS Trust.
30. Email correspondence between Professor Rutty and Bob Winter, NHS England.
31. Email correspondence between Professor Rutty and VN534, Thames Valley Police.
32. Email correspondence from VN535 to Professor Rutty re exhibit list.
33. Email correspondence between Professor Rutty and Mrs Angie Gillies.
34. Email correspondence between Professor Rutty and Jasmin Amoroso.
35. Email correspondence between Professor Rutty and Gareth Howard and VN920.
36. Email correspondence between Professor Rutty and Dean Jones.
37. Email correspondence between Professor Rutty and VN916.

38. Email correspondence between Professor Rutty and Dr Amanda Jeffery.

39. Email correspondence between Professor Rutty and Ishbel Gall.

40. Copies of expenses incurred by Professor Rutty to undertake autopsy examination.

41. Email of thanks from VN532, Thames Valley Police.

42. Handwritten notes from Meeting on 26th July 2018 with VN513, MK26 and [redacted]

43. Copy of letter to Professor Rutty from MK26 Dstl dated 2nd August 2018.

44. Copy of Op Read Sample Tracking.

45. Copy of OPCW Note by the Technical Secretariat.

46. Copy of General Practitioners Summary.

47. Handwritten notes of Professor Rutty from review of the General Practitioners Summary.

48. Copy of Salisbury hospital medical notes.

49. Hand written notes of Professor Rutty from review of the hospital medical notes.

50. Copies of clinical CT and radiology imaging.

51. CD of Metropolitan Police Images re Op Read Ref. ERV/558/18 and ERV/562/18.

52. CD of Index A Medick Notes

53. Copy of OPCW open access report.

54. Copy of DSTL Summary Analytical Report.

55. Copy of Letter to Professor K Suvarna.

56. Email correspondence between Professor K Suvarna and Professor Rutty.

57. Letter from Professor K Suvarna to Professor Rutty

58. Copy of Cardiac Pathology Report of Dr K Suvarna.

59. Email correspondence between Professor Rutty and Sally Munton, Finance Assistant, East Midlands Forensic Pathology Unit.

60. Email communications between Wendy Pitts and Keith Asman.

61. Copies of references used for report.

Signature: PD 29 November 2018

62. CD containing EM images of the heart provided by Professor K Survana, Sheffield Teaching Hospitals.

In addition to the case file, the following material was stored within the East Midlands Forensic Pathology Unit:

1. The microscopic slides and tissue blocks relating to this case.

DECLARATION

I, Guy Nathan Rutty, declare that:

1. I understand that my duty is to help the court to achieve the overriding objective by giving independent assistance by way of objective, unbiased opinion on matters within my expertise, both in preparing reports and giving oral evidence. I understand that this duty overrides any obligation to the party by whom I am engaged or the person who has paid or is liable to pay me. I confirm that I have complied with and will continue to comply with that duty.
2. I confirm that I have not entered into any arrangement where the amount or payment of my fees is in any way dependent on the outcome of the case.
3. I know of no conflict of interest of any kind, other than any which I have disclosed in my report.
4. I do not consider that any interest which I have disclosed affects my suitability as an expert witness on any issues on which I have given evidence.
5. I will advise the party by whom I am instructed if, between the date of my report and the trial, there is any change in circumstances which affect my answers to points 3 and 4 above.
6. I have shown the sources of all information I have used.
7. I have exercised reasonable care and skill in order to be accurate and complete in preparing this report.
8. I have endeavoured to include in my report those matters, of which I have knowledge or of which I have been made aware, that might adversely affect the validity of my opinion. I have clearly stated any qualifications to my opinion.

Signature: PD 29 November 2018

9. I have not, without forming an independent view, included or excluded anything which has been suggested to me by others including my instructing lawyers.

10. I will notify those instructing me immediately and confirm in writing if for any reason my existing report requires any correction or qualification.

11. I understand that:

(a) my report will form the evidence to be given under oath or affirmation;

(b) the court may at any stage direct a discussion to take place between experts;

(c) the court may direct that, following a discussion between the experts, a statement should be prepared showing those issues which are agreed and those issues which are not agreed, together with the reasons;

(d) I may be required to attend court to be cross-examined on my report by a cross-examiner assisted by an expert.

(e) I am likely to be the subject of public adverse criticism by the judge if the Court concludes that I have not taken reasonable care in trying to meet the standards set out above.

12. I have read Part 19 of the Criminal Procedure Rules and I have complied with its requirements.

13. I confirm that I have acted in accordance with the Code of Practice for Experts.

14. I confirm that I have read guidance contained in a booklet known as *Disclosure: Experts' Evidence, Case Management and Unused Material* which details my role and documents my responsibilities, in relation to revelation as an expert witness. I have followed the guidance and recognise the continuing nature of my responsibilities of disclosure. In accordance with my duties of disclosure, as documented in the guidance booklet, I confirm that:

(a) 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) I have compiled an Index of all material. I will ensure that the Index is updated in the event I am provided with or generate additional material;

(c) in the event my opinion changes on any material issue, I will inform the investigating officer as soon as reasonably practicable and give reasons.

EXPERT WITNESSES SELF CERTIFICATE

Revelation of information

(Criminal Procedure and Investigations Act 1996)

Name of expert witness: Professor GN Rutty

Date of birth: Over 21

Business address: East Midlands Forensic Pathology Unit, University of Leicester, Level 3, Robert Kilpatrick Building, Leicester Royal Infirmary, Leicester. LE2 7LX

I have been instructed to provide expert evidence in relation to the prosecution of the above-named, or an investigation into the following criminal offence:

I confirm that I have read the booklet known as Guidance Booklet for Experts - Disclosure: Experts' Evidence, Case Management and Unused Material that has been given to me with this form, and that I am aware of my responsibilities as an expert witness to reveal to the Prosecution Team any information that might undermine my evidence.

Personal Information

1.	Have you ever been convicted of, cautioned for, or received a penalty notice for any criminal offence (other than minor traffic offences)?	No
2.	Are there any proceedings pending against you in any criminal or civil court? Guidance Booklet for Experts	No
3.	Are you aware of any adverse finding by a judge, magistrate or coroner about your professional competence or credibility as a witness?	No
4.	Have you ever been the subject of any adverse findings by a professional or regulatory body?	No
5.	Are there any proceedings, referrals or investigations pending against you that have been brought by a professional or regulatory body?	No

Signature: PD 29 November 2018

6.	Are you aware of any other information that you think may adversely affect your professional competence and credibility as an expert witness?	No

Should you have any queries in relation to your answers to any of the above, please contact the investigator.

Please note that the questions above apply to any proceedings, findings or other relevant information in this or any other jurisdiction.

If you have answered **yes** to any of the questions numbered 1-6, please give details below.

Declaration

All the information I have given in this certificate is true to the best of my knowledge and belief.

I will notify those instructing me of any change in this information.

I am aware that any false or misleading information I have given in this document, or any deliberate omission of relevant information may lead to disciplinary or criminal proceedings.

I confirm that I understand my duty is to the Court and that I have complied with that duty.

The information given within this report represents my understanding of the views, opinions and circumstances of this case based on the information that I have received to date, either in writing (all forms) or by oral communication. I recognise that in part this may reproduce or rely upon witness statements, oral communications or hearsay evidence of second parties and that the information given to me by others may or may not be factually correct at the time of my consideration.

I reserve the right to reconsider any aspect of this report should a significant typographical or grammatical error, or factual inconsistency, be identified that could be misinterpreted by a reader.

I also reserve the right to reconsider any aspect of this report should further factual information arise that contradicts the information provided at the time of the production of this report, upon which I have based my interpretations.

PD

Professor Guy N RUTTY

MBE MD MBBS FRCPath DipRCPath (Forensic) FCSFS (Foundation) FFFLM (Foundation), AFHEA

Chief Forensic Pathologist

GMC Registration Number 3201440

Signature: PD

29 November 2018

INTERNAL CRITICAL CONCLUSIONS CHECK

This report has been subjected to a Critical Conclusions Check in accordance with the Code of Practice for Forensic Pathologists held by the Forensic Science Regulator. On the information available to me (paperwork) the examination described and the conclusions reached in this report are reasonable – Dr FH Hollingbury, 29th November 2018.

Signature: PD 29 November 2018

APPENDIX A

Copies of the Clinical AchE Test results

"STURGESS, MISS DAWN KELLY" 18/06/1974 PD

PD

Report COM

Report ID 26909104

NHS Number PD

Patient IDs

Sex Female

DOB 18/06/1974

Discipline Lab Medicine

Location Radnor

Requester Donnison PED

Import Date 05/07/2018 20:50:12

Report Date 05/07/2018 20:49

Responsible Clinician Donnison PED

Address Cedar Room, John Baker House, Rollestone Street, Salisbury, SP1 1DX

Blood

Sample Number G.18.0285128.K

Collected 04/07/2018 00:00

Received in lab 04/07/2018 11:44

Service Provider's Comments:
ACETYL CHOLINESTERASE
Assay performed at Sandwell & West Birmingham Hospitals NHS Trust

COM

COM*Free Text Comment F

Test Comments:
RC C'esterase * 268 U/L (7900-17100)

WARNING: Textual results that are too long for the width of the printed page will be word wrapped which may disrupt any original formatting. It is suggested to print structured textual results on a landscape layout. The result may have been amended or updated since printing took place so please check Review.

"STURGESS, MISS DAWN KELLY" 18/06/1974 PD

PD

Report COM

Report ID 26981537

Amendment

Import Date 12/07/2018 13:50:12

Report Date 12/07/2018 13:45

Responsible Clinician Dr K W Shamel

NHS Number PD

Patient IDs

Sex Female

DOB 18/06/1974

Discipline Lab Medicine

Location Radnor

Requester Dr K W Shamel

Address Cedar Room, John Baker House, Rollestone Street, Salisbury, SP1 1DX

Blood

Sample Number G,18.0286210.V

Collected 06/07/2018 09:00 Received in lab 06/07/2018 10:33

Service Provider's Comments:
ACTYL CHOLINESTERASE

COM

COM^Free Text Comment F

Test Comments:
RC Cholinesterase 417 U/L (7900 - 17100)

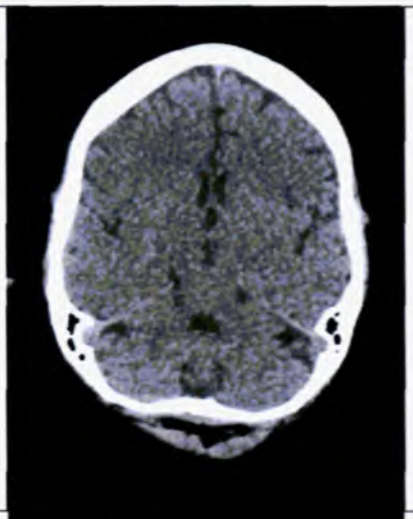
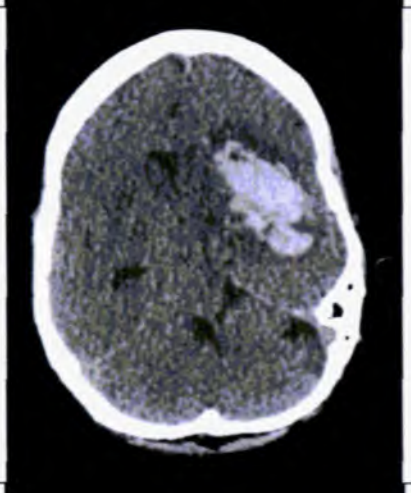
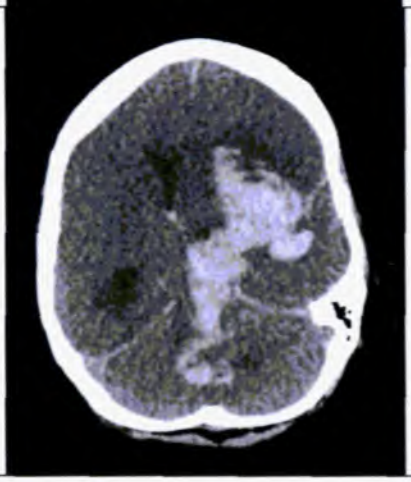
Amended report - previous incorrect RC cholinesterase result

on this lab number. Apologies

WARNING: Textual results that are too long for the width of the printed page will be word wrapped which may disrupt any original formatting. It is suggested to print structured textual results on a landscape layout. The result may have been amended or updated since printing took place so please check Review.

APPENDIX B

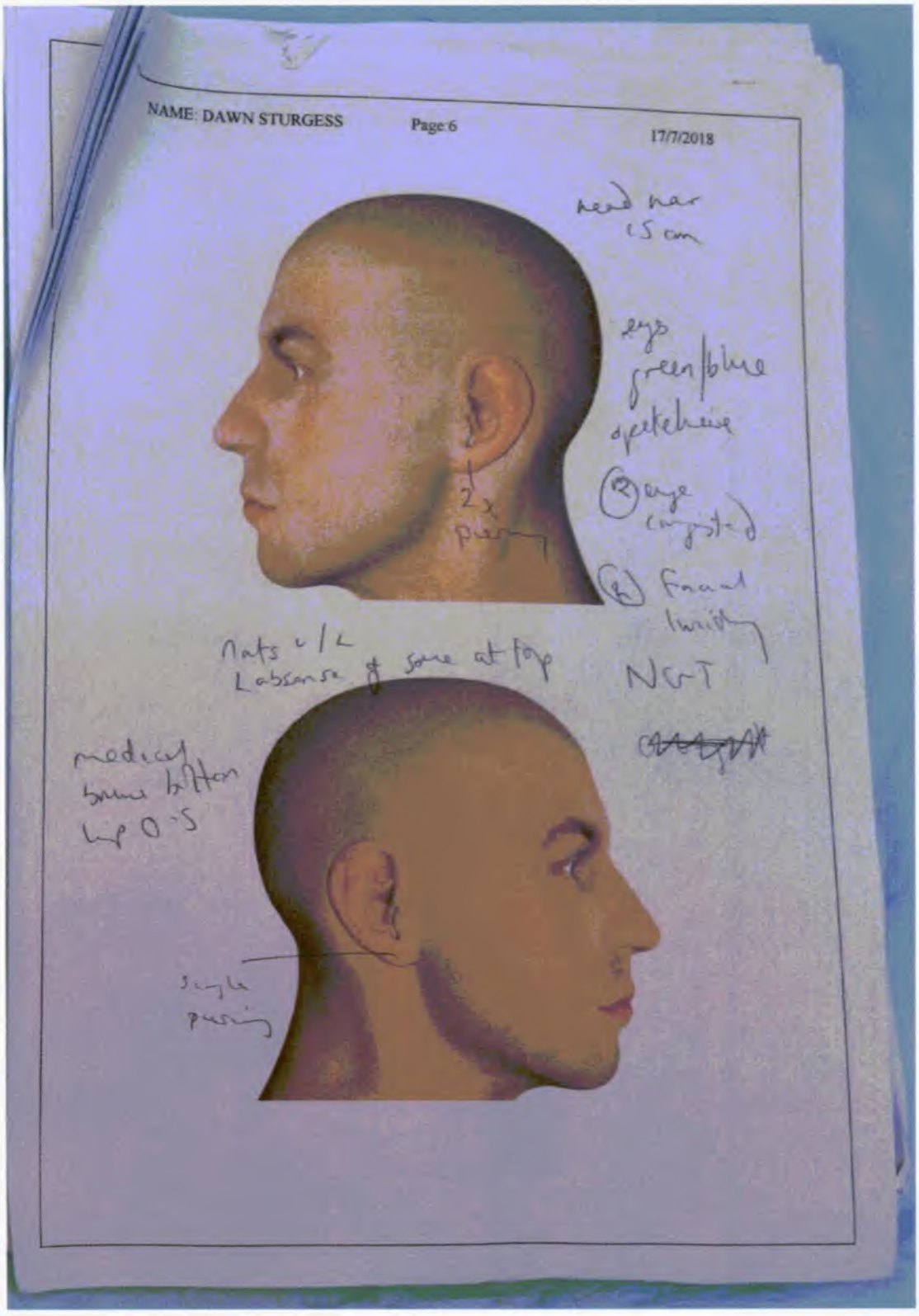
Images from the CT scans in life

	Admission CT scan 30th June 2018 showing no evidence of intracerebral haemorrhage.
	CT head scan 6th July 2018 showing acute left sided intracerebral haemorrhage.
	CT head scan 7th July 2018 showing extension of the haemorrhage downwards towards the brainstem.

APPENDIX C

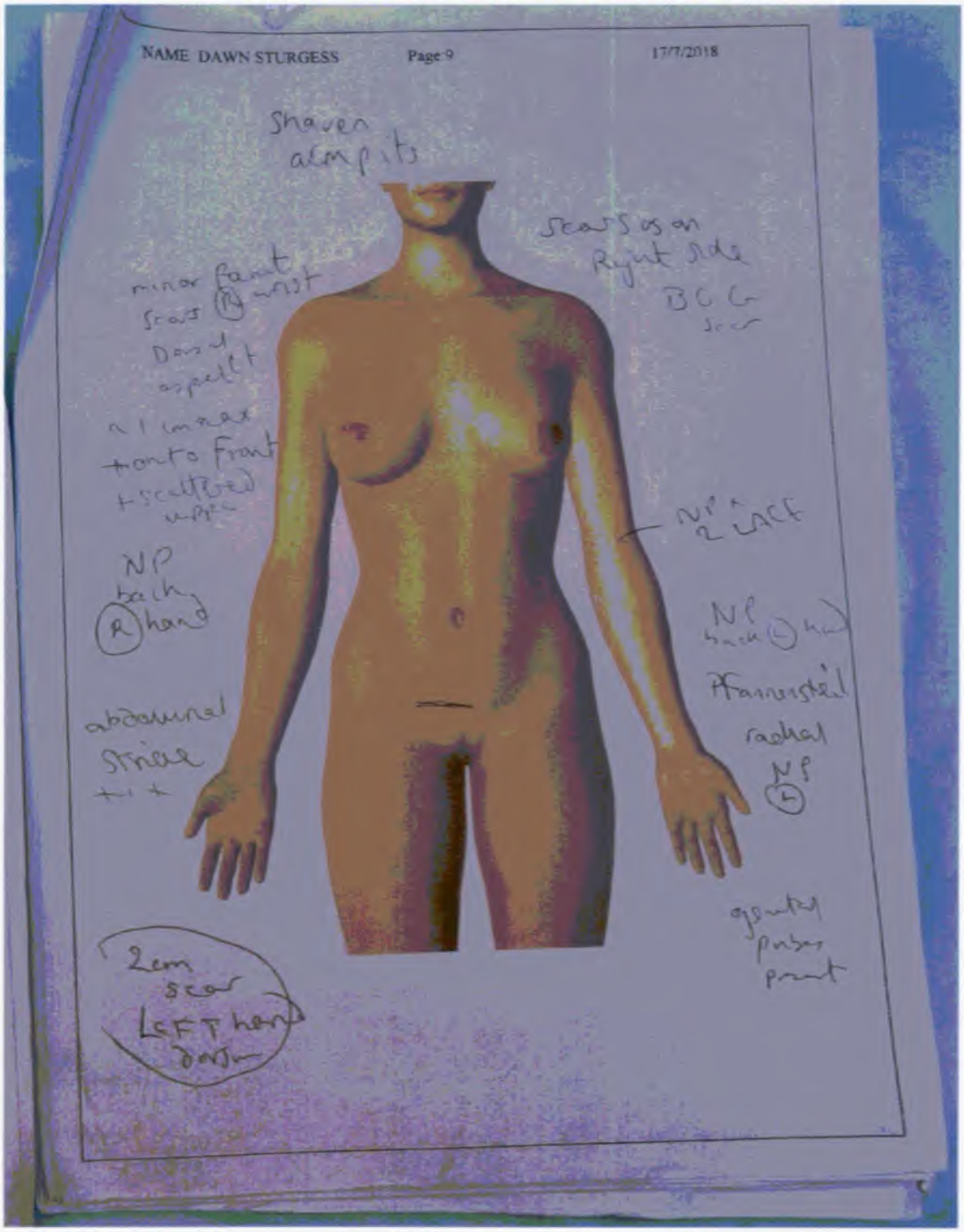
Copies of Body Diagrams

completed at the time of the Post-Mortem Examination



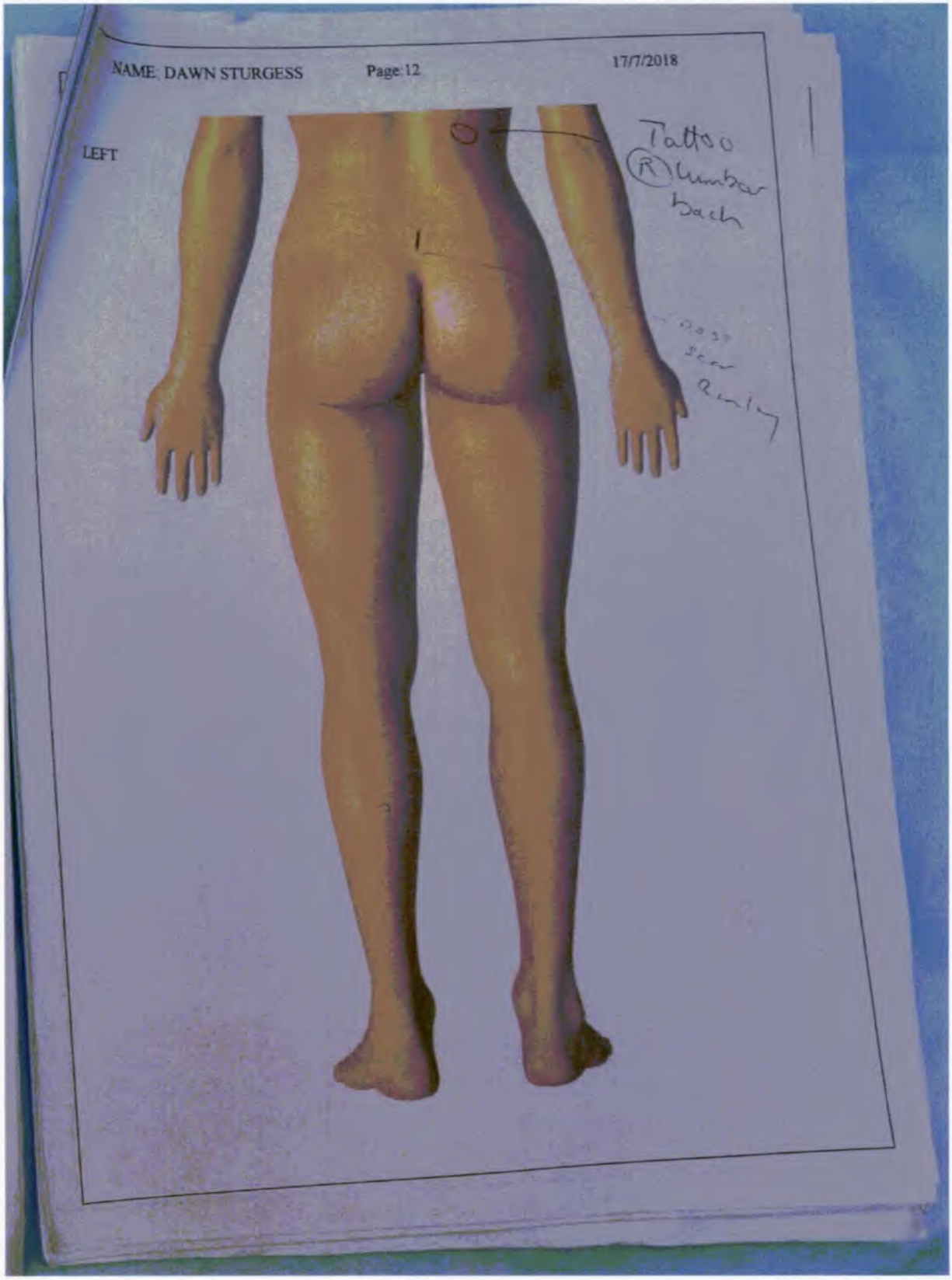
Signature: PD

29 November 2018

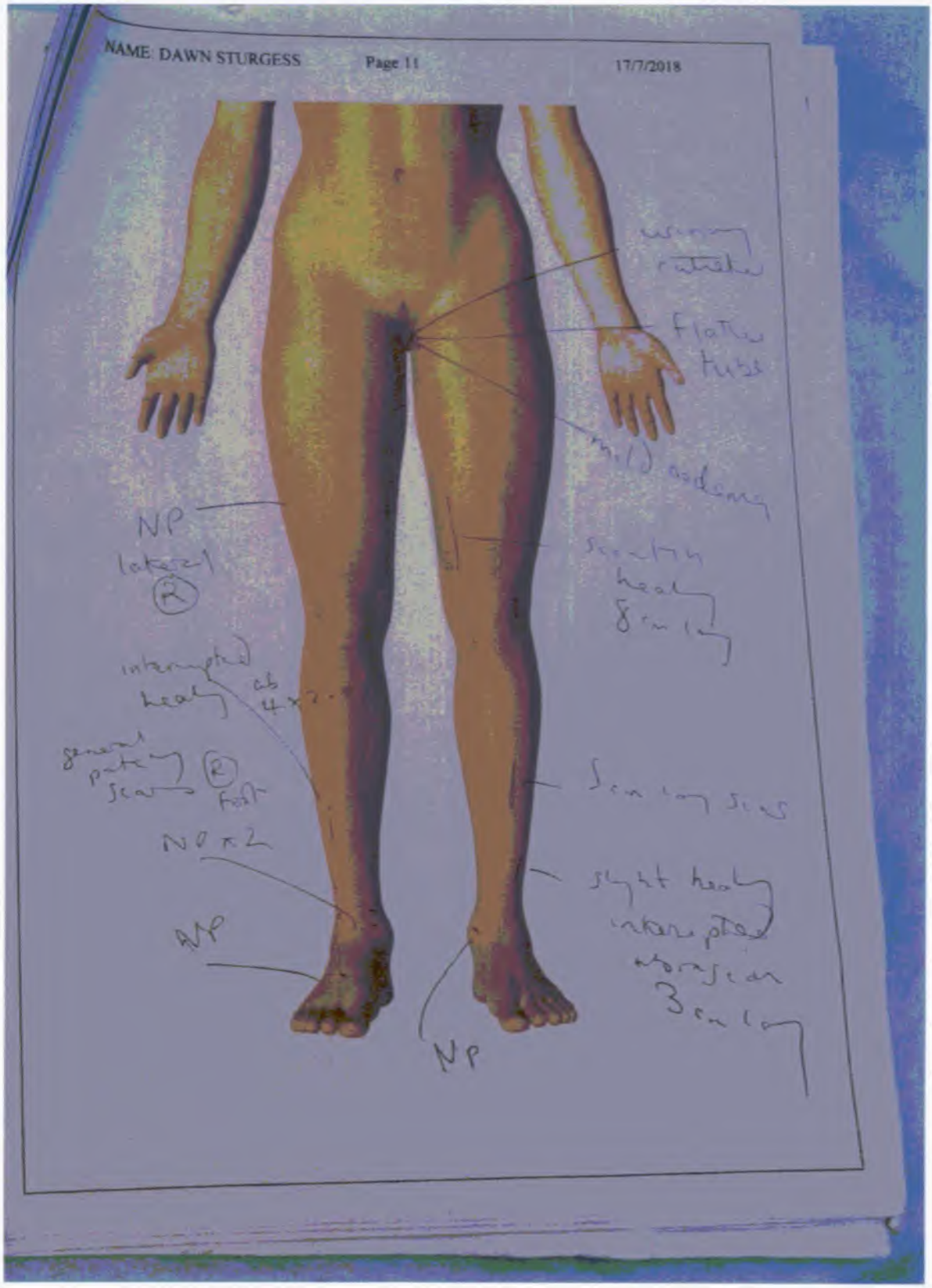


PD

Signature: 29 November 2018 51



Signature: PD 29 November 2018

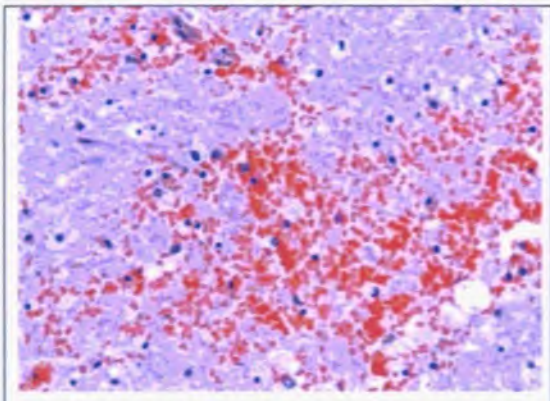


Signature: PD

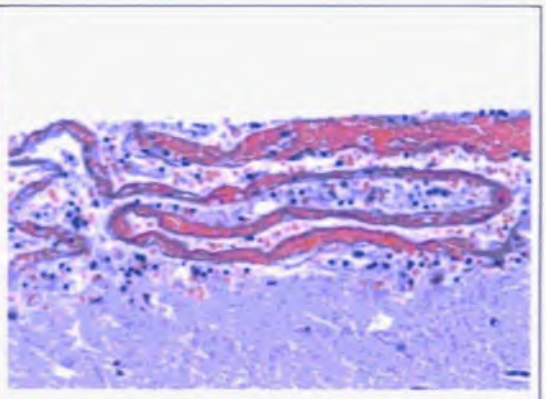
29 November 2018

APPENDIX D

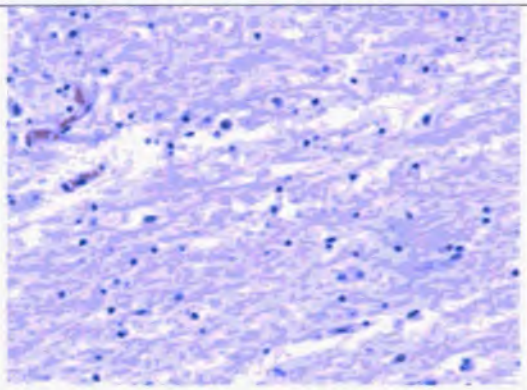
Selected representative images of the EMFPU tissue examination



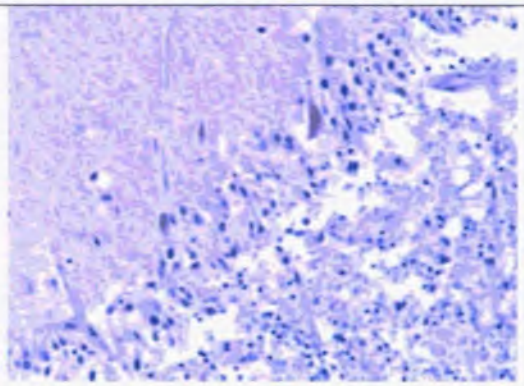
Cerebral parenchymal haemorrhage



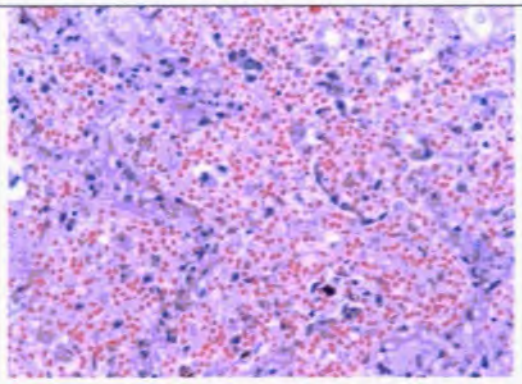
Subarrachnoid haemorrhage



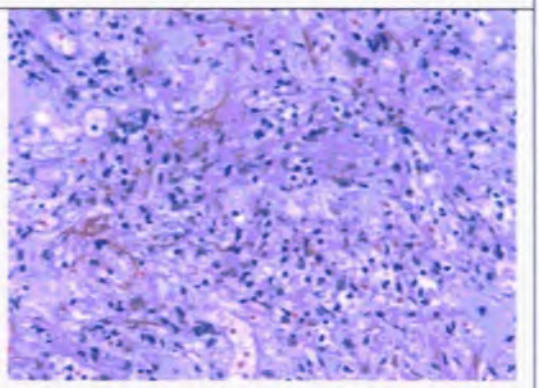
Hypoxic brain injury – cerebral hemisphere



Hypoxic brain injury - cerebellum



Focal pulmonary haemorrhage



Increase pulmonary cellularity and inflammation

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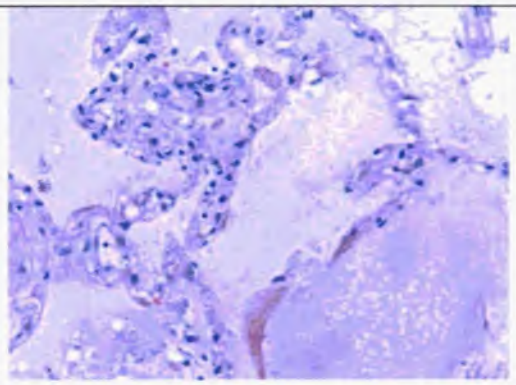
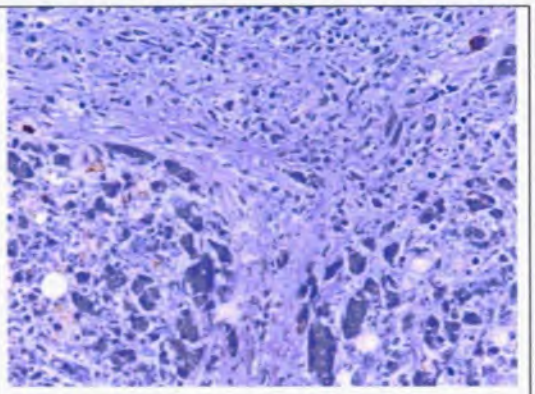
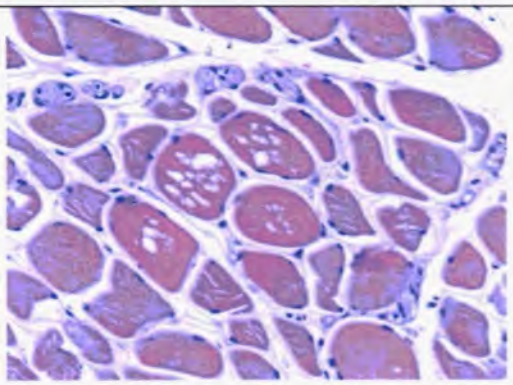


Pulmonary oedema

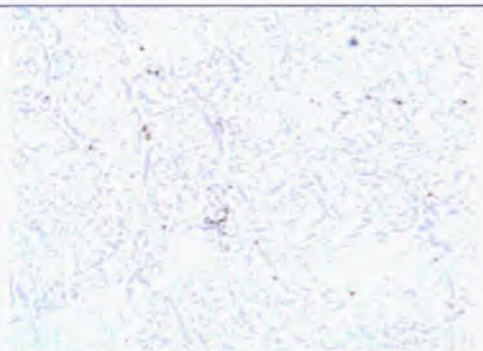


Liver cirrhosis and fatty change

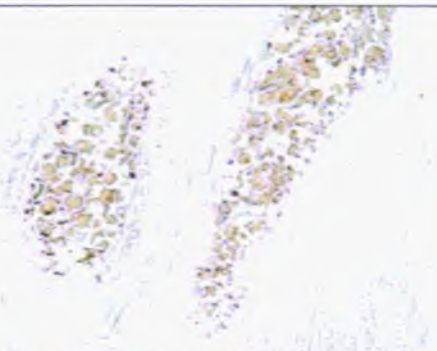


Vacuolated skeletal muscle cells

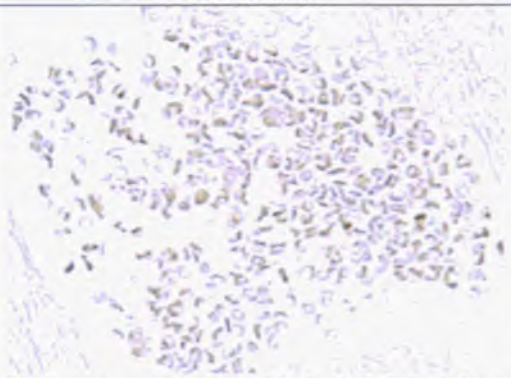
Signature: PD 29 November 2018 56



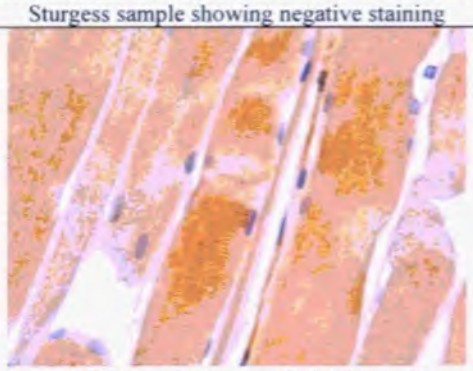
Cholinesterase technique, thiocholine method
Positive control granular deposits between skeletal muscle cells



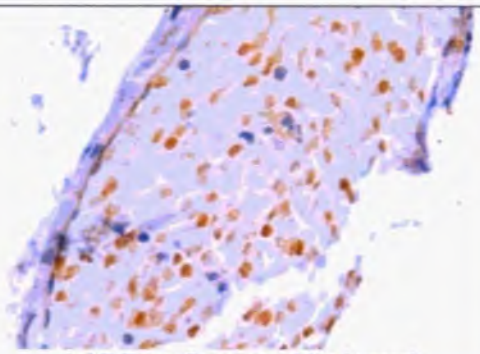
Cholinesterase technique, thiocholine method
Positive control granular deposits in blood vessels



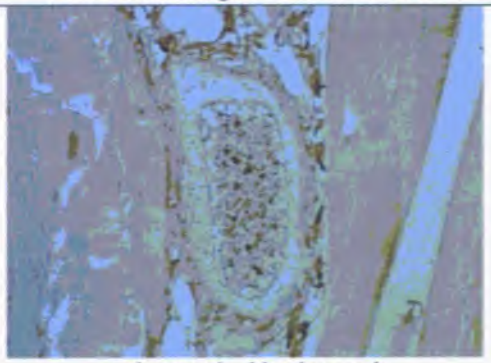
Cholinesterase technique, thiocholine method Dawn Sturgess sample showing negative staining



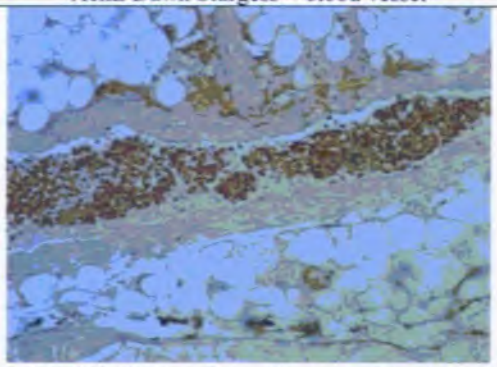
AchE Dawn Sturgess - skeletal muscle



AchE Dawn Sturgess - blood vessel



Ach control - blood vessesl



Ach (1:250 dilution) Dawn Sturgess – blood vessel

Signature: PD

29 November 2018

APPENDIX E

Cardiac Pathology Report

[redacted] Page 1 Suvarna

Medical report

Dawn STURGESS

DOB – 18/06/1974 DOD – 08/07/2018

I have been instructed by Professor GN Rutty to review the histopathology on Ms Sturgess.

Report prepared by Professor Simon Kim Suvarna
Consultant Histopathologist, Sheffield Teaching Hospitals and Honorary Professor for University of Sheffield.

Degrees conferred - MB BS BSc FRCP FRCPath

Professor Suvarna is a consultant in Histopathology at a teaching hospital in Sheffield and has specialised in cardiovascular pathology. He is a member of both the British Association of Cardiovascular Pathologists and the Association of European Cardiovascular Pathologists. The Sheffield Teaching Hospitals has surgery for heart disease and general cardiovascular disease. Professor Suvarna also is involved with Trust-based cardiovascular research.
A full copy of his CV is available on application, with a brief summary on his website: www.kimsuvarna.com

[redacted] Page 2 Suvarna

Overview.

This case was referred in order to consider, firstly, whether the cardiac tissues had any bearing upon the cause of death for this individual and, secondly, to consider whether a putative nerve agent played a part in her death.

Case history.

It is understood that this individual died as a consequence of exposure to a nerve agent 'Novichock' on 06/07/2018. It was indicated that she developed respiratory distress shortly afterwards, prompting ambulance attendance and that she subsequently developed a cardio-respiratory arrest.

Despite resuscitation attempts she remained in a comatose state but died two days following admission (on 08/07/2018) with evidence of hypoxic brain injury. It is understood that Professor Rutty and Dr Lumb undertook an autopsy, approximately one week following the death of the individual with limited samples being taken.

The samples taken hoped to include the sino-atrial node, atrio-ventricular node and a mid-ventricular slice of myocardium.

Autopsy.

Details of this examination are not available to me presently.

Macroscopic examination.

The specimen comprised heart fragments with a referrer's laboratory reference: FP3256
Three pieces of tissue were present in two pots, named on the outside.
The first (48 x 44 x 26 mm) comprised the base of the foramen ovale, part of the tricuspid valve and part of the aortic valve. It also included membranous septum.
The second (48 x 36 x 24 mm) appeared to comprise part of the left atrium with pulmonary vein insertion points.
The third piece of tissue was a transverse section (105 x 71 mm, average 10 mm thick) at mid-ventricular level of cardiac tissue showed right and left ventricle wall thicknesses

of 5 and 15 mm thickness. There were transverse right and left ventricle diameters of 47 of 31 mm. No focal lesion was identified.

Histological examination.

The left atrial tissues were unremarkable, but clearly did not include the sino-atrial node tissues.

The parenchyma around the atrio-ventricular node, His bundle and upper septal tissues was judged within normal limits.

The transverse tissue block of ventricular parenchyma showed a largely normal phenotype, with preserved myocytes set within a mildly oedematous interstitium. There was, nevertheless, minimal and focal scarring fibrosis of a chronic type, without acute infarction changes. There was also some vacuolar change in cardiac myocytes particularly in the left ventricle, not inconsistent with the stated history of cardio-respiratory arrest – in the form of "stunned myocardium".

Overall, the appearances do not support acute myocardial infarction, or indeed the effects of prior zonal ischaemic damage. No features of myocarditis or structural cardiomyopathy were evident. No features of idiopathic myocardial degeneration, amyloid deposition, micro-vasculopathy and granulomatous inflammation were identified.

However, the light microscopy of the myocardial tissue showed a small amount of focal fibrosis of an established type. No acute fibroplasia, infarction or provocative factor was identified in the tissue sections at these points.

Electron microscopy was performed on the tissue (by Mr B Wagner, Senior Electron Microscopist, STH), under my supervision. The images were reviewed by me. There was background autolysis present. The cardiac myocytes appeared normal in size, measuring between 10 and 20 micron in diameter. The myofilaments were normal and without disarray. There was no excess of lipid or glycogen. The secondary lysosomes were

morphologically unremarkable. However, there are moderate numbers of scattered enlarged dysmorphic mitochondria measuring up to 5 x 2 micron in diameter.

Comment.

This cardiac review was prompted by the sudden death of Dawn Sturgess in unusual circumstances.

It is understood that a cardio-respiratory arrest occurred and therefore it seems reasonable to consider the possibility of underlying cardiac disease as a substrate for this event.

Whilst only part of the heart has been available for study, the appearances do not point to an underlying structural cardiac lesion of congenital type. They do not specifically support a major pathological process, such as a heart attack or equivalent. Indeed, the majority of the tissue examination is within acceptable limits for an adult female of this age living in the United Kingdom.

The examination has shown several points of interest, with minimal and patchy scaring within the left ventricle wall tissues. This is clearly chronic – and therefore does not map to the acute events described. The impact of this fibrosis can be debated, but without there being a history of previous palpitations, syncopal events, episodic hypotension or other cardiac symptoms, then it is likely that this mild pathology had no relevance to the death of Ms Sturgess.

Nevertheless, one can speculate as to the likely cause/s for this pathology, with previous myocarditis and some drugs/alcohol as potentially relevant. However, it is stressed that the amount of fibrosis is small and, given the background normal tissues, the fibrosis is not regarded as pertinent in terms of the death of this individual.

The electron microscopy study showed relatively good preservation of cardiac myocytes, the myofibrils and organelle content – save for the mitochondria. The mitochondrial tissues showed a variety of changes, which are not normally seen in surgical biopsy

and/or autopsy parenchyma samples. Nevertheless, it has to be understood that mitochondria are readily susceptible to hypoxic phenomena (eg. following cardio-respiratory arrest). Furthermore, there will be structural changes incurred because of post-mortem autolytic phenomena.

It is understood that the Novichok agent is associated with cholinesterase inhibition of a non-reversible format. This has pertinence to nerve cell conduction with cholinergic axons and acetyl-choline receptors. These are only minimally present in cardiac tissue, mainy aligned to the parasympathetic nervous system interface with the conduction system myocytes. It is difficult to derive a link of the nerve agent to general mitochondrial status, simply on the known actions of the toxin under consideration.

Conclusions.

- The majority of tissues are considered within normal limits for this individual.
- There are some minimal chronic fibrotic changes, likely not relevant to the cause of death.
- There are some ultrastructural mitochondrial changes, which likely to reflect aspects of the cardio-respiratory arrest, rather than the Novichok agent. However, it is recognized that there is no database on the morphological effects of this toxin on normal human myocardial parenchyma for cross-comparison.

I confirm that I have made clear which facts and matters referred to in this report are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete and professional opinions on the matters to which they refer.

PD

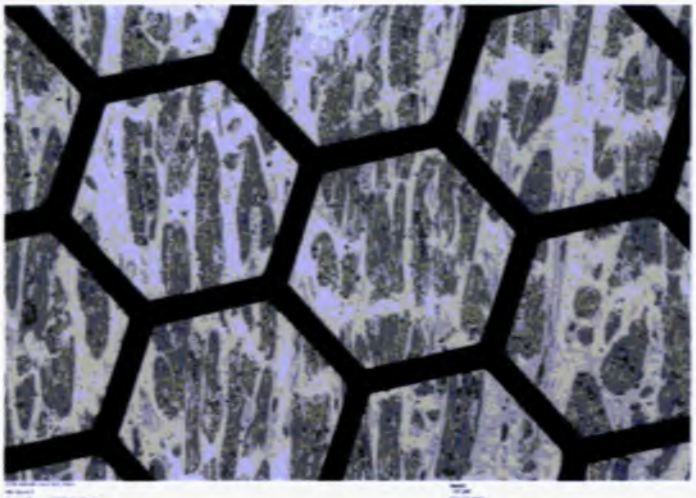
date 16.9.18

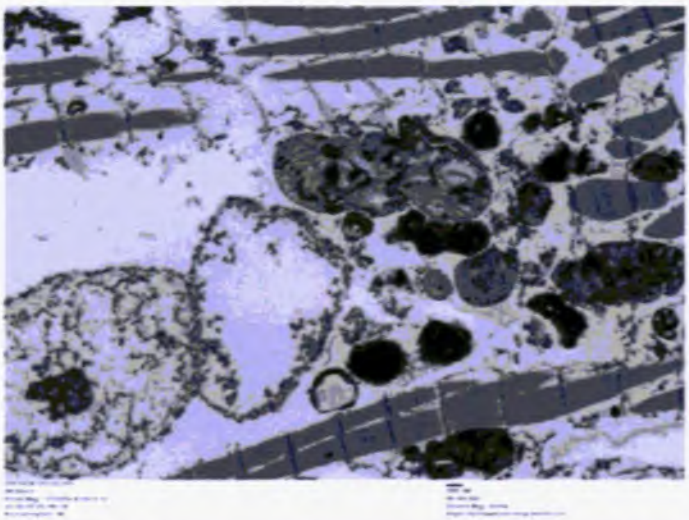
S. K. Suvarna

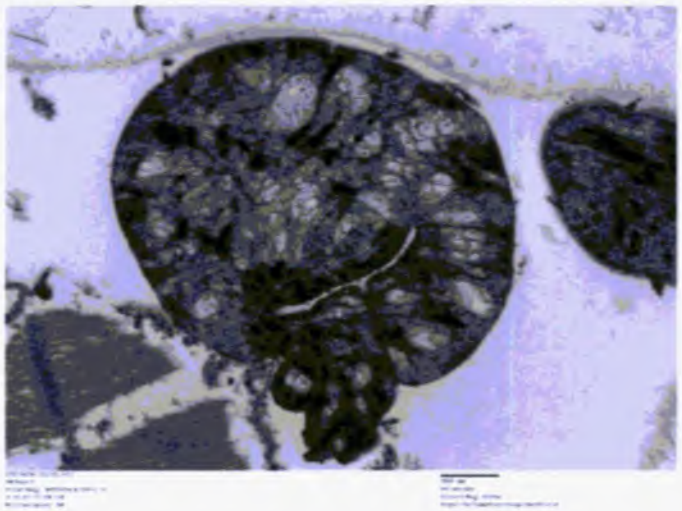
Note. The tissue blocks, slides and electron microscopy material will be passed back to Professor Rutty

APPENDIX F

Cardiac Electron Microscopy Images







APPENDIX G

The Organisation for the Prohibition of Chemical Weapons open source report

CS-2018-1313(E) distributed 04/09/2018 *CS-2018-1313.E*

OPCW Technical Secretariat

S/1671/2018

4 September 2018

Original: ENGLISH

NOTE BY THE TECHNICAL SECRETARIAT

SUMMARY OF THE REPORT ON ACTIVITIES CARRIED OUT IN SUPPORT OF A REQUEST FOR TECHNICAL ASSISTANCE BY THE UNITED KINGDOM OF GREAT BRITAIN AND NORTHERN IRELAND (TECHNICAL ASSISTANCE VISIT TAV/03/18 AND TAV/03B/18 "AMESBURY INCIDENT")

1. The United Kingdom of Great Britain and Northern Ireland requested technical assistance from the OPCW Technical Secretariat (hereinafter "the Secretariat") under subparagraph 38(e) of Article VIII of the Chemical Weapons Convention in relation to an incident in Amesbury on 30 June 2018 involving a toxic chemical and the poisoning and hospitalisation of two individuals and the subsequent death of one. The Director-General decided to dispatch a team to the United Kingdom for a technical assistance visit (TAV).

2. The TAV team deployed to the United Kingdom from 15 July to 18 July 2018 to collect biomedical samples and again on 13 August 2018 to obtain an additional environmental sample.

3. The team received information on the medical condition of the surviving affected individual, Mr Charles Rowley. This included information on his acetylcholinesterase status since hospitalisation, as well as information on the treatment regime.

4. The team was able to collect blood samples from Mr Charles Rowley for transport to the OPCW Laboratory and subsequent analysis by OPCW Designated Laboratories. Mr Rowley was able to give informed consent himself.

5. The team attended and observed the post-mortem (autopsy) of Ms Dawn Sturgess. The team was able to collect a number of biomedical samples (mainly tissue samples) for transport to the OPCW Laboratory and subsequent analysis by OPCW Designated Laboratories. Consent for this procedure was obtained from the next-of-kin of Ms Sturgess, and the activity was carried out in compliance with the United Kingdom Human Tissue Act.

6. The team requested and received splits of biomedical samples collected by the British

authorities for delivery to the OPCW Laboratory and subsequent analysis by OPCW Designated Laboratories. This was done for the purposes of comparison and in order to verify the analysis conducted by the United Kingdom.

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7. The team was briefed on the identity of the toxic chemical identified by the United Kingdom and was able to review analytical results and data from the chemical analysis of biomedical samples collected from the affected individuals by the British authorities.

8. The results of the analysis of biomedical samples conducted by OPCW Designated Laboratories demonstrate that Mr Charles Rowley and Ms Dawn Sturgess were exposed to and intoxicated by this toxic chemical.

9. During the second deployment, the team collected a sample of the contents of a small bottle that the police seized as a suspect item from the house of Charles Rowley in Amesbury.

10. The results of the analysis of this environmental sample conducted by OPCW Designated Laboratories show that the sample consists of the toxic chemical at a concentration of 97-98%. The sample is therefore considered a neat agent of high purity. The OPCW Designated Laboratories also identified a number of impurities constituting the remaining 2-3% of the sample.

11. The results of the analysis conducted by OPCW Designated Laboratories of environmental and biomedical samples collected by the OPCW team confirm the findings of the United Kingdom relating to the identity of the toxic chemical that intoxicated Mr Charles Rowley and Ms Dawn Sturgess. The toxic chemical compound, which displays the toxic properties of a nerve agent, is the same toxic chemical that was found in the biomedical and environmental samples relating to the poisoning of Sergei and Yulia Skripal and Mr Nicholas Bailey on 4 March 2018 in Salisbury (S/1612/2018, dated 12 April 2018).

12. Due to the unknown storage conditions of the small bottle found in the house of Mr Rowley and the fact that the environmental samples analysed in relation to the poisoning of Sergei and Yulia Skripal and Mr Nicholas Bailey were exposed to the environment and moisture, the impurity profiles of the samples available to the OPCW do not make it possible to draw conclusions as to whether the samples are from the same synthesis batch.

13. The name and structure of the identified toxic chemical are contained in the full classified report of the Secretariat, available to all States Parties.

APPENDIX H

Dstl Summary Report

[redacted]

[dstl]

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Summary Analytical Report

Summary

This report summarises the analysis undertaken at Dstl on biomedical samples recovered from Dawn STURGESS pre- and post-mortem. Analysis of blood collected on admission to hospital confirms that Dawn STURGESS was poisoned with a Novichok nerve agent. Analysis of blood and tissue samples collected from Dawn STURGESS at the post-mortem demonstrate that butyrylcholinesterase and acetylcholinesterase were close to completely or completely inhibited at the time of death, and that the characteristic marker of the Novichok nerve agent were still detectable in her blood. Analysis of liver, kidney and brain tissue samples demonstrates the presence of characteristic Novichok metabolites in all three sample types. Furthermore, very low levels of free intact Novichok were detected in the brain homogenate. These results indicate systemic presence of the Novichok agent.

Analysis of Dawn Sturgess blood samples collected following admission to Salisbury District Hospital

Blood samples were received into the [redacted] at Dstl Porton Down on 2nd July 2018. These samples were identified as collected from Dawn STURGESS (exhibit JDB/2, PTN/18/1100) following her admission into Salisbury District Hospital on the 30th June 2018. A sub-sample from PTN/18/1100 was screened for inhibition of cholinesterase enzymes, specifically butyrylcholinesterase and acetylcholinesterase enzymes. Inhibition of the activity of these enzymes is indicative of exposure to a nerve agent or related organophosphorous compound. In PTN/18/1100 negligible levels of both butyrylcholinesterase and acetylcholinesterase activity were detected, indicating that Dawn STURGESS was exposed to a nerve agent or related compound.

A sub-sample of blood from Dawn STURGESS was analysed for the presence of free, unbound nerve agent. In sub-samples from PTN/18/1100 a Novichok nerve agent was detected.

In order to confirm the preliminary result described above, the sample from Dawn STURGESS was analysed for the presence of nerve agent bound to butyryl cholinesterase enzyme. The sample was positive for the presence of a Novichok-butyryl cholinesterase nonapeptide, a characteristic marker for exposure to a particular nerve agent of the Novichok class. In this case the presence of the same Novichok which had been identified as free agent in the blood was confirmed on the 4th July 2018. These analytical results confirmed that Dawn STURGESS was poisoned with a specific Novichok agent.

Analysis of a blood sample collected during the post mortem examination of Dawn STURGESS

A post-mortem blood sample collected into EDTA (GR56, PTN/18/1379) was submitted to Dstl for analysis on the 18th July 2018. A sub-sample from PTN/18/1379 was screened for inhibition of cholinesterase enzymes, specifically butyrylcholinesterase and acetylcholinesterase enzymes. In PTN/18/1379 activity at or close to the limit of detection of both butyrylcholinesterase and acetylcholinesterase activity was detected, indicating that

[redacted]

[redacted]

Dawn STURGESS' blood cholinesterases were still completely or almost completely inhibited at the time of her death.

A sub-sample of PTN/18/1379 was analysed for the presence of free, unbound nerve agent, no free agent was detected in this sample.

A sub-sample of PTN/18/1379 was analysed for the presence of nerve agent bound to butyryl cholinesterase enzyme. The sample was positive for the presence of a Novichok-butyryl cholinesterase nonapeptide.

In addition, the Novichok-acid metabolite was also detected in a sub-sample of PTN/18/1379, this is characteristic for the Novichok in question.

Analysis of tissue samples collected during the post mortem examination of Dawn STURGESS

Samples of liver (PTN/18/1308), kidney (PTN/18/1309) and brain (PTN/18/1310), removed during the post-mortem of Dawn STURGESS were homogenised and analysed for the presence of free Novichok and the metabolites desethyl Novichok, Novichok acid and desethyl Novichok acid. The results of this analysis are detailed in Table 1 below.

LSN	[redacted] Ref	Sample type	Novichok	Desethyl Novichok metabolite	Novichok acid metabolite	Desethyl Novichok acid metabolite
1810451	PTN/18/1308/C1/A	Liver homogenate (20% w/v)	ND	ND	Detected	ND
1810452	PTN/18/1309/C1/A	Kidney homogenate (20% w/v)	ND	ND	Detected	Detected
1810453	PTN/18/1310/C1/A	Brain homogenate (20% w/v)	Detected	ND	Detected	ND

ND – Not detected [redacted] desethyl Novichok, Novichok acid and desethyl Novichok acid).
Note – Dstl has not undertaken analysis of human tissue samples previously. Therefore, while some method optimisation has taken place, these methods should be treated as developmental.

Table 1 – Post mortem tissue sample results

[redacted]
Dstl Porton Down

[redacted]

Signature: PD 29 November 2018 72