BAHA MOUSA PUBLIC INQUIRY

PATHOLOGICAL ASPECTS OF SUDDEN DEATHS ASSOCIATED WITH STRUGGLE AGAINST RESTRAINT

PATHOLOGY REPORT ON BAHA MOUSA

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Curriculum Vitae: Dr DS James

I am Deryk Simon James and my qualifications are Bachelor of Medicine and Surgery, Fellow of the Royal College of Pathologists and diplomate in Medical Jurisprudence (Forensic Pathology). I have been a Home Office Registered pathologist since 1994. I have been Senior Lecturer in Forensic Pathology to the University of Cardiff and Honorary Consultant in Forensic Pathology to Cardiff and Vale NHS Trust since 1997 and I have 17 years experience in providing post mortem services to the Police Forces of Dyfed Powys, South Wales, Gwent and Gloucester for the investigation of suspicious deaths and to the Coroners within those geographic areas.

I have broad experience within the field of forensic pathology and I have published peer reviewed scientific papers on subjects which include stab wounds, hanging, the performance of post mortem examinations in infants and young children, head injury in infants, medico-legal investigation of deaths and death certification. I have given evidence in a variety of jurisdictions at home and abroad.

Declaration

I have prepared this report at the request of the Baha Mousa Public Inquiry. I confirm my understanding that it is my over-riding duty to assist the Inquiry on the matters within my expertise as detailed in the report in the same way that would apply under Part 35 of the Civil Procedure Rules and the associated Practice Direction and Annex if it were evidence provided before a court of law. I also confirm that I shall continue to comply with that duty in relation to any further written or oral evidence I may provide to the Inquiry.

1 Introduction

1.1 The occurrence of sudden death occurring in circumstances of extreme physical and psychological stress is well recognised. It has been described particularly as occurring during struggle against restraint, when it is often associated with stimulant drug use (such as cocaine and amphetamine), and in some forms of acute psychosis, when it may be accompanied by **neuroleptic** drug administration ('chemical restraint'). Numerous other associations have been identified which have been proposed as "causal factors" but, as yet, the precise mechanism or mechanisms of death in these circumstances are incompletely established and scientific confirmation of the part played by any "**causal factor**" in the individual case is imprecise.

1.2 This review will describe the evolving recognition of this group of deaths, the physiological experiments and observational studies that have taken place and the explanations for these deaths that have been derived from them. It will describe the various circumstances and conditions that are associated with these deaths and attempt to draw conclusions regarding causality.

1.3 The aim of this analysis is to allow the enquiry team to have sufficient knowledge of the medical aspects of this group of deaths to pose such questions about the circumstances of Mr Mousa's death that will allow the most reliable conclusions concerning his medical cause of death to be drawn.

2 Historical

2.1 In 1849 Bell described deaths following prolonged periods of agitation occurring in a background of mental disorder [1]. Such deaths – then, often ascribed to "exhaustion", and now called "agitated delirium", "excited delirium" and "lethal catatonia" – remained well recognised until the introduction of effective treatments such as electro-convulsive therapy (ECT) in 1934[2] and neuroleptic drugs, such as the phenothiazines, the first of which, chlorpromazine, began trials in 1952[3]. Since that time, it is rare for someone to die of the untreated, intrinsic effects of mental illness. However, it has been realised that disturbed, agitated and violent patients may collapse and die and that such deaths are often associated with restraint – more accurately, struggle against restraint – and with the use of certain neuroleptic drugs administered in significant doses to induce sedation (chemical restraint) [4]. Such observations have led to guidance from the Royal College of Psychiatrists[5,6]. Guidance on the use of control and restraint in mental healthcare has been in place since the Mental Health Act 1983 Code of Practice, culminating in training recommendations in the "Tilt Report"[7]. Guidance for police officers[7a] and prison officers[7b] required to restrain or control is also in place.

2.2 There is some suggestion that "excited delirium" was recognised as early as 1914 as a complication of cocaine use[8] but it was not until the rapid increase in cocaine use in the 1980's that the syndrome became common and well-recognised[9]. Those affected are

Neuroleptic: A tranquilizing drug, especially one used in treating mental disorders
Causal factor: A condition or circumstance that appears more frequently in association with an outcome than chance would dictate but lacking a scientifically proven link of cause-and-effect.

described as being in an excited, delirious state, frequently combative and violent often with seemingly heightened strength. Such behaviour is liable to result in public disturbance, police attendance, arrest and/or an effort to subdue the individual; it is at this time, when there is confrontation - perhaps with the use of CS gas or "stun" guns - followed by the use of physical restraint and, possibly, handcuffs, that sudden collapse and death seems to occur.

2.3 As series of such deaths were published the link between stimulant drug-associated behavioural disturbance and sudden death occurring in the initial stages of police custody became evident and, in particular, a link was made with the "hog-tied position" – this being a technique used by single-handed police officers in the USA to keep a detainee restrained during a car journey - consisting of cuffing the hands behind the back, strapping the ankles together and flexing the knees so that the ankle straps and handcuffs can be tied together; this done, the detainee is placed prone on the flat back seat of the vehicle[10]. For Reay, the placing of a detainee in such a position raised the possibility of a condition known as positional asphyxia, that is, death caused by persons being unable to extricate themselves from a position in which they are unable to breathe properly. He subsequently conducted research into the physiological effects of the "hog-tied" position which appeared to show significant respiratory embarrassment. Warnings were issued to law enforcement personnel not to use the "hog-tying" technique but, although the technique was largely abandoned, deaths during restraint did not disappear; rather, a further association was identified with prone restraint, particularly when a "restrainer's" body weight was also applied to the back of the detainee[11].

2.4 Further research began to cast doubt on Reay's initial findings; although a varying effect on respiratory capacity was noted in the "hog-tied" and prone positions, no effect could be found, (even after simulating "struggle against restraint"), which could explain a fatal outcome in young, healthy men. Further observations concerning metabolic abnormalities in those suffering collapse after prone restraint pointed to a more multifactorial explanation. Muscle breakdown (rhabdomyolysis), very high body temperature ("heatstroke" or hyperthermia) and the production of large quantities of **lactic acid** were noted raising the possibility of a severe "acidosis" – this is a serious derangement which has many complications but which the body can compensate for, in part at least, by overbreathing – hyperventilating – something that prone restraint or "hog-tying" might be preventing. Further theories about why restraint is associated with sudden death relate to excessive production of adrenaline triggering cardiac arrest, a mechanism which would fit with cocaine use (which can raise adrenaline levels) and with "fight or flight" situations, such as often accompany these deaths.

2.5 At the present time there is no single view within the medical literature as to the "cause of death" in "restraint cases"; many authors advise caution in attributing causal status to any given association. However, a pattern is starting to emerge and I will explore the current state of thinking below, starting with an examination of whether "restraint asphyxia" is an appropriate name.

lactic acid: a waste product formed when the body exercises vigorously without having a sufficient supply of oxygen

3 What is asphyxia?

3.1 Asphyxia, which means a lack of oxygen and an excess of carbon dioxide in the blood, is an imprecise term also used to describe any sort of interference with respiration, from suffocation to cyanide poisoning. Many pathologists prefer not to use the term, regarding it not only as imprecise but also as a mode rather than a cause of death; instead they adopt terms which describe the nature of the injurious insult itself (such as "pressure to the neck", "hanging", "exposure to smoke", "suffocation due to enclosure in a confined space").

3.2 Common, but non-specific, findings in deaths due to "asphyxia" include petechial haemorrhages and congestion; these are prominent when there has been blockage of the veins taking blood back to the heart (such as occurs during pressure to the neck and chest); this "blockage of venous return" causes the veins to fill with blood (causing redness of the face or "congestion") and the pressure within the vessels to rise. The smallest and less well supported of these vessels burst, leaving pinpoint red spots (petechiae), most obvious in the loose skin around the eyes and in the linings of the eyes and lips. When these are obvious and are present only above the area of pressure (such as above a ligature around the neck) they are a reliable indication of prolonged (probably at least 15-30 seconds[12]) significant pressure. They may also be present and obvious if a person has died face down and remained in that position for some time after death and may be present in small numbers in many other conditions. In the absence of petechiae all that can be said is that there has not been sufficient pressure on the neck or chest during life to cause a prolonged (at least 15-30 seconds) and significant rise in venous pressure.

4 How can pressure to the face, neck and chest cause death?

4.1 There are various mechanisms of death which may act in deaths ascribed to asphyxia; these all prevent oxygen getting to the brain and include blockage of the airway (preventing oxygen getting into the lungs so that the blood contains insufficient oxygen to keep the brain alive); blockage of the arteries in the neck (preventing blood – and, therefore oxygen - getting to the brain); blockage of the veins in the neck (preventing blood from getting into the brain because none can get out back to the heart) and cardiac arrest (stoppage of the heart causing all blood flow to stop).

4.2 Where death results from the inability of the victim to breathe in as much oxygen as is being used, and to breathe out as much carbon dioxide as is being produced (a situation which would result from blocking the airway, as in suffocation with a pillow) there will be a gradual loss of oxygen in the blood – causing, in a white-skinned person, the skin colour to change from pink to blue (cyanosis) – until unconsciousness results. Efforts to breathe would continue, as would heartbeat, until sufficient brain damage had occurred to cause these vital functions to cease. If pressure were to be released during unconsciousness but

whilst there was still control of breathing and heartbeat, breathing would recommence and, as oxygen levels rose, consciousness would return as long as brain function had not been compromised by hypoxia. The time taken for death to result from stopping breathing is variable. Competitors in "static apnoea" competitions can maintain consciousness for in excess of 10 minutes without taking a breath[13]; a much shorter period than this would result in death for a person untrained in the discipline or if oxygen usage was high – such as during exertion (but see 4.5).

4.3 Where death results from complete and unremitting obstruction of the arteries in the neck (such as in some cases of hanging), consciousness is lost 3 to 11 seconds later.[14] Heartbeat continues and pressure needs to be maintained for some minutes in order to cause sufficient brain hypoxia to result in a fatal outcome (but see 4.5). Congestion and petechiae do not occur so long as arterial obstruction is complete.

4.4 Where death occurs from complete and unremitting obstruction of the veins in the neck (such as in some forms of hanging and strangulation), consciousness is lost less rapidly than with arterial obstruction. Pressure will still need to be maintained for some minutes to cause a fatal outcome (but see 4.5). Congestion and petechiae are usually very prominent.

4.5 Where death occurs from cardiac arrest (which appears to occur in hanging, strangulation and other forms of pressure to the face, neck and chest) unconsciousness occurs within seconds of the arrest and death will occur if the arrest continues for some minutes. The rarity, in the literature, of cases of cardiac arrest following neck compression that is not part of an assault (such as in play, martial arts, contact sports and in some medical procedures) makes it physiologically likely that it occurs not only from the effect of direct pressure to the neck but also from other factors resulting from an assault, such as fear, anxiety, struggle, exertion, (all associated with high circulating **catecholamine** levels and with "altered neural control of the heart"). It is also curious that people who suffer strangulation either survive without any brain damage or die; non-fatal brain damage would be expected in some if an assailant, having kept pressure on the neck until unconsciousness, either changed his mind or mistook unconsciousness for death and released the neck-hold; this raises the question as to whether the usual mode of death in many forms of pressure on the neck is cardiac arrest – which would explain well why so few people survive "damaged"- this is in keeping with the widely held view that cardiac arrest can occur very quickly after the onset of pressure *or at any time thereafter*[12,15]

4.6 Some deaths due to stoppage of the heart are caused by blows to the chest (for example, a person hit by a fast-moving cricket ball), which, if they occur at a particular point in the heart's electrical "cycle" can cause the heart to "fibrillate" and then stop[16].

5 Crush asphyxia – traumatic asphyxia

5.1 These synonymous terms are used when the chest is fixed and respiration is impossible; it is described most frequently following building collapses and crowd disturbances. In its

Catecholamine: A group of adrenaline-like compounds produced by the body in response to a variety of physiological and psychological situations.

"mildest" form, the chest is fixed and breathing prevented; with increasing pressure, there is blockage of venous return with profuse congestion and petechiae in the face and neck. Further pressure causes muscle damage with rhabdomyolysis (see below); if the "crush" element is very severe, but relieved before "fatal asphyxia" occurs, then there may be cardiac arrest (from high potassium) or acute kidney failure and other complications of rhabdomyolysis[17].

6 Positional asphyxia

6.1 The term "positional asphyxia" has been used recently by many authors to describe deaths occurring during restraint. I shall use "restraint asphyxia" for all such deaths (except where quoting others). "Positional asphyxia" is a term that was first used to describe deaths explained by the inability to escape from a position in which respiration is not possible or is inadequate to maintain life. It is frequently seen in alcohol intoxication, where a person collapses into a position – such as between a bed and a wall – where breathing is difficult or the upper airway is obstructed.[18] Owing to the intoxication, the victim does not wake or automatically move from the restrictive position, but dies. Other examples include entrapment, such as may occur in a chimney or sloping ventilation shaft – where the weight of the victim pulls them downwards into a narrowing space, thus increasingly compromising breathing.

7 Restraint asphyxia

What is it?

7.1 Restraint asphyxia refers to the death of a person struggling against physical restraint. The term was introduced following the description of deaths occurring in early police custody as police used physical restraint to subdue agitated or violent men who often had been using stimulant drugs. It was soon recognised that similar deaths were also seen in mental institutions when agitated or violent psychosis was treated with physical or chemical restraint. It was argued that the restraint prevented breathing or made breathing difficult in someone who, through their agitation and exertion, had high oxygen demands.

What is the evidence for respiratory compromise, i.e. that it is "asphyxial"?

7.2 Reay et al observed the association between prone restraint – in particular, the "hog-tied" position – and sudden collapse and death; they undertook research to investigate the effects of this position on 10 healthy young volunteers following moderate physical exercise. They found that restraint tended to increase the time taken for blood **oxygen saturation** and pulse rate to return to normal after exercise and concluded that restraint induced measurable physiological changes; they cautioned that the relevance of this to

Oxygen saturation:The percentage of haemoglobin (the red pigment in the blood that carries oxygen) that is actually carrying oxygen molecules. A normal level is above 95%.

sudden deaths during restraint was not clear. Nevertheless, they felt that effects on respiration should be considered in deaths associated with handcuffing in the prone position[19]. This paper was followed in 1992 by a report of 3 cases of sudden death after restraint in the hog-tied position (or very similar to it) attributing death to "positional asphyxia" but offering no new physiological evidence to support this [20]. An article published simultaneously by Luke and Reay[21] was restrained in its advancement of restraint asphyxia: *"there are no easy answers in such deaths"*, *"Deaths in police custody often demonstrate even less pathologic evidence than is the case with most violent asphyxial fatalities and, consequently, with few anatomic findings, circumstantial, historical and scene investigative evidence becomes of the utmost importance"*. Laposata[22] concurred, saying: *"a constellation of factors produces a terminal cardiopulmonary arrest in these cases... Although the position [hog-tied prone restraint] interferes somewhat with respiration, [it] is not in itself a position that would be expected to be fatal in minutes"*.

7.3 Work by Chan et al and Schmidt et al who tried to investigate further the effects of prone restraint after exercise failed to reproduce Reay's findings; neither found differences in oxygen saturation or heart rate between the sitting and hog-tied positions, nor could they replicate the drop in oxygen saturation during exercise, their results being in accordance with the established physiological observation that oxygen saturation remains stable or is improved during moderate exercise[23,24]. Moreover, Chan et al measured lung function during restraint; whilst they demonstrated a reduction in **vital capacity** in the prone and restraint positions, the vital capacities recorded would still be regarded as being in the "normal" range. In a further article Chan argued that "*factors other than body positioning appear to be more important determinants for sudden, unexpected death in individuals in the hogtie restraint position*'.[25] Reay and Howard[26] accepted the evidence put forward by Chan and modified their view: *"the hog-tied position should be viewed as an inherently neutral position... there is no significant physiologic consequence [of the position] in normal people"*. However, they maintained that the position may still play some role and emphasised that the totality of events should be considered and that the cause of death should be regarded as potentially multifactorial.

7.4 Roeggla attempted similar experimental work to Chan and demonstrated a greater degree of reduction in lung capacity and also significant drops in pulse rate and blood pressure in the prone, hogtied restraint position compared with the kneeling hogtied position but no change in oxygen saturation. No differences were identified between the kneeling hogtied position and the unrestrained position.[27] Parkes confirmed the moderate effect on lung capacity found by earlier workers (other than Roeggla) and was also unable to demonstrate any reduction in oxygen saturation[28,29]. Using **pulse oximetry** in "real-life" episodes of restraint in a psychiatric institution Masters and Wandless showed no change in oxygen saturation during restraint and no difference between controls and those being restrained.[30] Further work by Meredith showed no difference in lactate levels or heart rate between unrestrained individuals and those having their wrists restrained front or back.[31] The abandonment of positional asphyxia as a valid explanation for death was encouraged on physiological grounds by Gulino[32] and by Glatter and Karch[33].

Vital Capacity: The maximum volume of air a person can exhale after maximum inhalation – effectively the maximum usable chest volume.
Pulse oximetry: a method of measuring oxygen saturation with a device that clips onto the finger.

7.5 Further work by Chan et al explored the effect on respiratory function of prone restraint compounded by compression of the chest[34]. They found that there was a significant difference between various measures of respiratory function between the sitting and prone restraint positions, (the latter position with and without loading - 25lb and 50lb weights being placed on the subjects' backs) but no significant difference between the weighted and unweighted prone restraint positions. They were unable to detect any evidence of a significant effect on oxygenation: "no evidence of hypoxia or hypoventilation" – they in fact found an increase in oxygenation with exercise.

7.6 Overall, therefore, there appears to be evidence for a degree of respiratory restriction from restraint which should cause no adverse effect in an otherwise fit young person. The potential for an effect when other factors are in play remains incompletely investigated but the potential for prevention of hyperventilation (over-breathing) remains – the implications of this are discussed further below.

7.7 Another way of assessing the role of impairment of breathing as the cause of death is to look at descriptions of how "collapse" occurred. If hypoxia caused by inadequate breathing caused loss of consciousness we should find descriptions of gradual loss of activity and consciousness followed by recovery after the loosening of any holds. What is interesting about descriptions of deaths is that the collapse seems to occur after variable periods of restraint and is sudden: *"all 21 suddenly lapsed into tranquillity shortly after being restrained"* [11,35]; such collapses may even occur after the release of the hold when the individual is getting to his feet.[36] The collapse may be followed by "turning blue" rather than having been preceded by it[37] which suggests that hypoxia is a consequence of the collapse rather than a cause of it. All these observations suggest that the mode of death in "struggle against restraint" is **cardiac arrest** or **cardiopulmonary arrest**.

Other conditions that might be involved in "restraint" deaths

8 Injury

8.1 Any injury that causes significant damage to tissues or blood loss compromises function; the extent to which an individual suffers harm depends on the injury and the

circumstances in which it happens. The loss from a blood donor, of a pint of blood over 10 minutes may result in some light-headedness or a little tiredness; the loss of the same amount as bruising within torn muscle and around fractures in a background of severe exertion, exhaustion and pain would be different – one might be able to demonstrate and measure the effects if one monitored patients with various injuries in various circumstances using medical equipment during life; it is not possible after death to say what the precise effects of particular injuries actually were, just by looking at them. However, with increasing blood loss and tissue damage one would expect a general

Cardiac arrest: sudden stoppage of the pumping action of the heart as it either goes into an ineffective rhythm (ventricular fibrillation)or stops altogether (asystole)
Cardiopulmonary/cardiorespiratory arrest: Cessation of heartbeat and breathing

increase in pain, in physiological effects, such as malaise, high pulse rate and low blood pressure and in psychological effects such as distress, fear and anxiety.

Bruising

8.2 Given the importance of the timing and extent of inflicted injury in this case it may be worth describing bruising in some detail. A bruise is the result of blood leaking from damaged blood vessels into surrounding tissues. If the damage occurs in the skin or just beneath it, it becomes visible very quickly, usually within a minute or two. Examples would include the reddish bruising of a "lovebite" or a boxer's "black eye". The former is simply the bursting of tiny blood vessels in the skin and the leak of very small amounts of blood which will resolve (be broken down and absorbed) in only a day or two – the bruise will fade and perhaps develop a slight yellow tinge before disappearing; the latter involves not just the leaking of more substantial amounts of blood but also damage to tissues – it will be accompanied by swelling, which is a reaction to injury in which fluid leaks from blood vessels and fills the injured area making it enlarged, tense and painful. Such swelling begins within minutes of injury and may last at least a day or two. The more substantial collection of blood will take longer to be broken down, and over time it will spread out under the skin of the face until it covers an area larger than the area actually injured. As the blood breaks down, green, yellow and brown pigments are formed causing the bruise to change colour; as the bruise heals these pigments will be absorbed, making the bruise fade and disappear. The process of resolution is of variable length – it can vary from a day or two, to many weeks. There is nothing in the literature to suggest that this process is accelerated in high ambient temperatures.

8.3 If the damage is deeper within the body the leaked blood may not be visible from the surface; over time that blood will spread out (that movement assisted by gravity and muscular activity), the blood will break down and pigmented fluid will diffuse out from the collection of blood – bruising then may become visible at the skin surface. An example of this would be bruising appearing on the fleshy parts of the thigh several days after a fall.

8.4 The initial colour of a bruise will be reddish purple if it is in the skin itself but it may be blue or even brown if it lies deeper beneath skin and/or a layer of fat (which is yellow). If it is red or purple and accompanied by swelling it is "fresh"; if it appears yellow anywhere, it is at least 18 hours old.[38] No more than this can be said with any accuracy from the external appearance alone – despite charts for "ageing bruises" being published in many books, such charts rarely agree with each other and objective research demonstrates how inaccurate they are; the changes in a bruise with time vary greatly and there are too many variables present to allow a valid opinion in an individual case. If samples from the bruise are available to examine under the microscope it is possible to see the first reactive changes a few hours after the bruise occurs; after 72 hours, iron pigment can be demonstrated,[39] after a few days more advanced healing changes may be seen. Even with the microscope one can usually go only so far as to suggest that a bruise appears "fresh" (first day), [40] that it is "at least a day old" or is "at least a few days old".

8.5 After death the blood (within blood vessels) sinks through the body under gravity pooling in the lowest blood vessels and appearing as purple discolouration known as hypostasis seen particularly on the back of a body lying face-up after death. This discolouration may appear in other places on the body as well, depending on the position of the body after death; wherever it occurs it may be mistaken for bruising. Bruises are difficult to cause after death – injury to a dead body will still damage blood vessels but if there is no blood circulating in those vessels then there is none to leak into the surrounding tissues, so no bruise will form. The exception to this is if injury occurs to an area of hypostasis – as the vessels in such an area are full of blood they will leak blood if damaged, although this is usually to a limited extent and it will not be accompanied by swelling.

8.6 Bruises that have been caused before death will change after death. The drainage of blood from the background skin will alter the appearance very soon after death; decomposition can change the colour, the outline and how "obvious" a bruise is; this process will be accelerated if the body is maintained after death at a high ambient temperature. The process of diffusion of broken down blood through the tissues which happens in life will continue after death – it is well known to pathologists that a body several days after death may show evidence of bruising not seen at the time of death or shortly after.

Soft tissue injury

8.7 Both muscle and fatty tissues can be damaged by the same forces that cause blood vessels to tear; this will lead to torn muscle fibres and burst fat cells. This damage to tissues results in the leaking into the bloodstream of cell fragments and constituents, some of which can be toxic and some of which may be "inflammatory". Fat globules getting into the bloodstream will get distributed mainly to the lungs, where they can block the tiny blood vessels in which oxygen is taken up from the air spaces of the lung and cause a severe inflammatory reaction in the lung tissue itself. An important element of muscle, myoglobin, can also be released (see below); some of the released elements may cause the body to react as it does in infection, with a fever and other physiological changes, which may lead to feelings of malaise.

Rib fractures

8.8 Broken ribs may be accompanied by blood or air entering the chest cavity but where this does not happen the major effect of a few isolated rib fractures is to make breathing painful. Every time the chest is expanded the broken rib ends will move against each other, causing sudden, "catching" pain which is often severe. A consequence of this is that the injured person tends to take only shallow breaths – often using the diaphragm to breathe with rather than the chest - in order to avoid the pain. After a period of time, usually in the elderly, this can lead to lung collapse or infection. Fractures that occur during life are almost always accompanied by bleeding whereas those that occur after death (frequent in the ribs due to resuscitation attempts) are not. It is difficult to date fractures by the naked eye unless healing includes new bone formation (callus); under the microscope post mortem fractures can be distinguished from fractures occurring during life and changes due to the reactive and healing process start to become visible in a few

hours. The changes that occur from fresh fracture to healed fracture have been described.[41]

9 Hyperthermia

9.1 Heat injury can arise out of exposure to high environmental temperature or be caused or contributed to, by exertion. It is most frequently seen in association with drug use (especially antipsychotics, amphetamine-like substances, and alcohol), in the elderly and in those with cardiovascular disease[42]. It results in acidosis (acidification of the blood, a serious metabolic problem), tissue injury (which is of particular consequence in the brain, heart and liver) and serious consequences in all organ systems. Where it is associated with an exertional cause there may be rhabdomyolysis, and acute kidney failure due to myoglobin release can occur. Symptoms usually start with restlessness and confusion culminating in collapse and coma; as the effects on the brain predominate in the early clinical picture, this change in consciousness may occur soon and worsen quickly[42].

10 Rhabdomyolysis

10.1 Rhabdomyolysis is the breakdown of muscle tissue releasing toxic substances including myoglobin and potassium into the circulation. It has many causes which include:[43]

direct physical damage to muscle,	
	traffic accidents or crush injury
	electric shock
exertional	
	strenuous exercise[44] (particularly in sickle trait)
	struggle against restraint
environmental	
	high ambient temperature; high humidity
hypoxic	
	prolonged immobilisation
hyperthermia	
drug effect	
	cocaine, "ecstasy", alcohol
	neuroleptic drugs (used to treat psychoses)
	other therapeutic drugs (eg statins; anaesthetic agents)
toxins	
	heavy metals, snake venoms
electrolyte imbalance	
	from vomiting, water intoxication or diarrhoea
	low potassium, sodium, calcium, phosphate

natural disorders

endocrine/metabolic disorders, infections, epilepsy, sepsis,

10.2 Physical injury is probably the commonest cause of rhabdomyolysis but most cases are asymptomatic; of those diagnosed in emergency units the three main causes are cocaine, exercise and immobilisation.[45]

10.3 Whilst myoglobin is released from muscles and can be found in the urine after strenuous exercise, when exertion is "extreme" severe rhabdomyolysis can occur; this is most liable to happen at high ambient temperatures and in high humidity.[46]

10.4 Rhabdomyolysis affects the kidneys, and if severe, results in kidney failure because myoglobin released by the damaged muscle is filtered out of the blood by the kidney and is passed into the tiny tubules of the kidney which concentrate the urine. When a person is dehydrated, has a low blood volume or has acid urine the myoglobin can block the tubules and is toxic to the lining cells of the tubules which stop concentrating the urine – so fluid is lost - and can no longer get rid of excess potassium. Kidney failure – even if total – will usually not result in a dangerously high potassium level for several days, but potassium is also released from the damaged muscle – if sufficient potassium is released the blood level will rise to a dangerous level and cause cardiac arrest. Symptoms of rhabdomyolysis may be mild and indistinguishable from "post-exercise aches and pains" but about 50% of cases have either muscle pain or weakness; dark urine is usual. There may be malaise, fever, fast heart rate, nausea and vomiting. The early complications are abnormal cardiac rhythms/ cardiac arrest; later ones occurring usually after 12-72 hours, are kidney failure and DIC (disseminated intravascular coagulation – a potentially catastrophic effect on blood fluidity).[43]

11 Acidosis

11.1 The blood, like water, is neither acidic nor alkaline. When it becomes acidic there is said to be an "acidosis". This occurs in many conditions such as kidney problems, diabetes and septicaemia; when it is severe it is life threatening and some forms of acidosis are associated with sudden death,[47] including deaths in young men.[48] Acidosis can result from physical exercise because as muscles work they produce lactic acid as a waste product. Acidosis can also result from starvation; this is usually mild but it can be severe if it is accompanied by physiological stresses.[49] Extreme acidosis has been identified in victims of sudden death during restraint (mostly but not exclusively after the use of stimulant drugs) and it has been suggested that it occurs because there is a massive build up of lactic acid produced by the considerable physical effort of struggling, possibly made worse in such situations by indifference to pain leading to exertion beyond normal physiological boundaries. Severe acidosis can result in autonomic instability with cardiovascular collapse and fatal abnormalities of heart rhythm.[50] The body reacts to this acidosis by "compensating", which it does by overbreathing (hyperventilating) which causes the loss of carbon dioxide; this directly affects the acidity of the blood, tending to

improve the acidosis. It may be that a person who is struggling against restraint and is acidotic can breathe enough to keep their oxygen levels normal but is not able to overbreathe. This means they cannot correct any acidosis, which will worsen as exertion continues; the worse the acidosis, the more prone the individual to autonomic instability and cardiac arrest.

12 Cardiac arrest

12.1 There is well established evidence that people who suffer from various forms of heart disease are in jeopardy of developing abnormal or fatal disturbances of heart rhythm when exposed to physiological stresses such as exercise, cold, heat, eating and dreaming[51]. It has been observed that events liable to lead to widespread fear and anxiety are associated with a rise in the number of "sudden cardiac deaths" (sudden collapse and death due to an abnormal heart rhythm)[52,53]. Experimental work on the effects of "psychological stresses" on the autonomic nervous system (that part of the nervous system that controls automatic functions like heartbeat) demonstrates that relatively mild stresses – such as doing mental arithmetic or recounting an emotionally charged experience – may, in susceptible individuals, precipitate abnormal rhythms [54]. In extreme cases, such as watching the attempted resuscitation of a spouse, intense emotional response can lead to sudden cardiac death without there being any structural heart disease[55]. Usually, however, the young healthy heart will withstand substantial physiological and psychological stresses though it will still be affected by those stresses, as has been demonstrated in animal work. Heart rate can be raised in rats by inducing fear/anxiety[56] and experimental restraint in rats raises both heart rate and blood pressure[57]. Pigs made susceptible to abnormal heart rhythms and then subjected to experimental restraint reacted with an increased heart rate, increased catecholamine levels in the blood and brain and an increased frequency of potentially fatal heart rhythms[58,59]. Work using dogs has demonstrated that an increase in catecholamines circulating in the blood results in an increased susceptibility to abnormal heart rhythms[60]. Links between stimulation of parts of the brain and the development of abnormal heart rhythms are established[61]. Restraint and handling of rats and mice causes an increase in body temperature: "stress hyperthermia" [62,63]. All this suggests a complex interplay between the brain, the autonomic nervous system, temperature regulation, adrenaline release and the heart itself, in response to stressors such as exercise, restraint and fear/anxiety [64]; the link between mental stresses and abnormal activity in the midbrain (part of the brain stem) precipitating abnormal rhythms has been demonstrated in humans[65]; that this is mediated by abnormal sympathetic nerve discharge (part of the autonomic nervous system) is strongly supported by recent work[66].

13 Are there any natural conditions that might predispose towards sudden death during struggle against restraint?

13.1 As described above, people with impaired cardiac function are at increased risk of heat stroke. There are in addition a number of cardiac conditions (of which a person need not be aware), which can cause sudden death or be a substrate upon which other factors may

act in order to precipitate sudden death. These include **myocarditis** and **cardiomyopathy**, both of which may be diagnosed by thorough autopsy, and the **arryhthmogenic disorders** which cannot be diagnosed at autopsy. It is known that sudden death as a result of an abnormal rhythm can be the first symptom of these disorders and may be precipitated by exertion and various other physiological "stresses". Although not susceptible to autopsy diagnosis, these conditions run in families and symptoms of dizzy spells, faints and blackouts are often experienced; previous medical history, family history, and in some cases, genetic analysis, can allow diagnosis or exclusion.

13.2 Non-cardiac natural diseases to consider include asthma, where a history should be available and in which there are usually pathological findings; anaphylaxis, where a history and clinical picture should make the diagnosis; sickle cell disease/trait, epilepsy, and Addisonian crisis, where history of maintenance steroids and/or pathological findings should be present. There are few other conditions which might be relevant where there might be an absence of pathological evidence.

14 How do we determine a cause of death?

14.1 Determining how a disease or condition causes death is not straightforward. There are some diseases – mainly infections – where there are quite simple lines of cause and effect in which there are events (exposure to organisms) which are *necessary* for an effect (the disease) to happen and which, if they happen, are *sufficient* for the disease to occur – that is, there are no other conditions that are required in addition to exposure, for the disease to develop. Rabies is an example: it doesn't develop unless you are exposed – bitten – by a rabid animal; if you are bitten, that is enough(almost) to ensure you get rabies. Most diseases are not as clear cut in their development as this – they happen in some people but not others, they may require other, predisposing, conditions to be present or they may occur at a greater rate in some specific populations rather than others. For example, we know that blockage of the coronary arteries causes heart attacks; people are more likely to develop this condition if they are obese, smoke, exercise little, have a poor diet, have a family history of heart disease and have a high cholesterol level but none of these "factors" is enough in itself to cause blockage of the arteries nor is any of them, individually, necessary. Some people may smoke and be obese but never suffer heart disease; others may have no "risk factors" that we are aware of and yet still develop heart disease. "Risk factors" may be no more than our observations about the incidence of heart disease in large populations – excellent for determining healthy lifestyles and helping groups of people but of very limited use in working out what extent of disease an individual will have.

14.2 How can we analyse the role of a single factor out of many which may have had a role in causing a death? Sometimes we can look at how different factors might interact with each other and compare such interactions with past observations of similar patients; sometimes the interaction of multiple factors remains a mystery and we are unsure which played a role,

Myocarditis: an inflammatory disorder of heart muscle often following a viral infection
Cardiomyopathy: a group of disorders of the heart muscle – usually of longstanding
Arrhythmogenic disorders: a group of usually inherited disorders affecting the electrical control of the heart

which did not and how great or small any effect was.

14.3 An analogy might be helpful: a car fails to negotiate a bend; the driver is young, male and inexperienced; the car is old and in poor repair; the road is difficult, downhill and has sharp bends, the weather is terrible. How can we answer the question: "would he have left the road if it hadn't been raining"? On the face of it we are struggling here because though it may be a question we would like an answer to, it is impossible to re-run the event without the rain and how else could we find out the answer? We know that cars leave the road when it is dry as well as when it is raining so why aren't the other risk factors entirely responsible?

14.4 What is perhaps of more use to us is if there is something that stands out from the other factors – let us change the analogy and say it was a steep downhill road to the bend and examination of the car after the crash demonstrated that the brake pipes had been cut. In this case, it would be hard not to implicate the lack of brakes as having played a part (although it might be argued that even with brakes the crash would still have occurred as a result of the other factors).

15 Conclusions regarding current thinking on the causes of death during struggle against restraint

15.1 It cannot be said that there is a consensus in the literature or between pathologists as to the "cause of death in restraint cases" but the balance of opinion is in favour of a "multifactorial causation" with differing factors operating in individual cases. The running theme is "struggle against restraint" combined with "other conditions/circumstances". The occurrence of sudden deaths in the background of those "conditions/circumstances" but *without* "struggle against restraint" is not apparent in the literature. Nor is the occurrence of sudden death in "struggle against restraint" in the absence of any accompanying "conditions/circumstances" [67] ; this suggests that "struggle against restraint" alone is insufficient to cause death –it requires other factors in order for death to ensue.

15.2 If the view is accepted that "struggle against restraint" is not *sufficient* of itself to result in death but that, in the presence of certain other factors, it comes close to being a *necessary* factor for death to occur, then how close is it to the "cut brake pipes" in the analogy above? I feel that it fails to reach the standard because it is not enough on its own; it could be regarded as triggering the death, in the right background, and being, therefore, *a* cause but not *the only* cause, of death. The role of other factors is less certain but some other factor appears to be necessary in every case for a fatal outcome to occur and it is striking how the factors associated with these deaths can be shown, physiologically, to lead to susceptibility to the action of a trigger of abnormal heart rhythms.

15.3 The extent to which "struggle against restraint" depends on individual predisposition is unknown. Whilst it may be shown to have fatal effect in the right background in one individual we do not know how many people subjected to the same background

circumstances would have suffered a fatal outcome and how many would have survived, i.e. whether the fatal effect is predictable and reproducible, or uncommon and idiosyncratic.

15.4 Whilst there may be uncertainty as to precisely how dangerous is "struggle against restraint" in a particular background, it has become sufficiently recognised for detailed guidelines to have been drawn up by police and health care services for the use of physical restraint in situations that those professions have recognised as being "high risk". Thus, in the context of police custody, stimulant drug use and behavioural disturbance were identified as indicators of risk; in the context of mental health care, acute psychosis and neuroleptic drug use were identified.

15.5 Within the prison service, officers must be prepared to treat certain observations as indications of a potential medical emergency (REF). These observations are:

- Unusual rises in body temperature (*suggesting hyperthermia*)
- Exceptional or unexpected strength) *All suggesting*
- Exceptional violence) *severe behavioural*
- Abnormally high tolerance of pain) *abnormalities seen*
- Bizarre behaviour – as if 'high' on drugs) *in restraint deaths*
- Sudden abnormal passivity (*suggesting unconsciousness*)
  Noisy or laboured breathing (*suggesting obstruction to breathing, unconsciousness or imminent respiratory arrest*)

These are in accord with literature at the time of publication (1999) recognising factors associated with a risk of a fatal outcome if restraint is continued.

15.6 I do not know whether the factors possibly present in this case which have the potential to predispose towards a fatal outcome during restraint have been recognised by the military and investigated – it is not an issue which I have been able to address from the general medical literature.

15.7 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 that I have expressed represent my true and complete professional opinions on the matters to which they refer.

Signed.....Dr DS James

Dated.....14/9/9.....

16 REFERENCES

1. Bell L On a form of a disease resembling some advanced stages of mania and fever, but so contradistinguished from ordinarily observed or described combinations of symptoms as to render it probable that it may be an overlooked or hitherto unrecorded malady. American Journal of Insanity (1849);6:97-127
2. Detweiler MB et al Delerious Mania and Malignant Catatonia: A report of 3 cases and review. Psychiatric Q (2009)80;23-40
3. Delay J, Deniker P. Neuroleptic effects of chlorpromazine in therapeutics of neuropsychiatry.Journal of Clinical Experimental Psychopathology (1955);16(2):104-12.
4. United States General Accounting Office Report to Congressional Requesters. Improper restraint or seclusion use places people at risk. GAO/HEHS-99-176
5. The association between anti-psychotics and sudden death. Report of the Working Group of the Royal College of Psychiatrists Psycho-pharmacology sub-group. Council Report CR57. January 1997;
6. Consensus report on high-dose anti-psychotic treatment. Royal College of Psychiatrists, May 2006
7. Report of the Review of Security at the High Security Hospitals, February 2000

7a Prison Service Order No 1600 "Use of Force" PSI Ref. No. 48/1999 – Exhibit MW Doc 1

7b Safer restraint. Police complaints authority. 2002

8. Karch S Pathology of Drug abuse, 3rd edition, CRC Press, Boca Raton, 2002 p8 citing Williams E (1914) Negro cocaine "fiends" are a new southern menace, New York Times, Feb 8, p1
9. Wetli CV, Fishbain DA; Cocaine induced psychosis and sudden death in recreational cocaine users. Journal of Forensic Science (1985);30:873
10. Reay DT et al. Positional asphyxia during law enforcement transport. American Journal of Forensic Medicine and Pathology (1992);13:90
11. O'Halloran RL, Frank JG. Asphyxial death during prone restraint revisited. American Journal of Forensic Medicine and Pathology (2000); 21(1):39-52
12. Saukko P & Knight BHKnight's Forensic Pathology. 3rd edition, Arnold, London 2004 p371.
13. International Association for the Development of Apnea. Static apnea record:Stephane Mifsud; Heyres, France 8th June 2009, 11mins 35 secs.
14. Rossen R et al Acute arrest of the cerebral circulation in man. Archives of neurology and Psychiatry (1943); 50:510-28.
15. Adelson L The pathology of Homicide. CC Thomas, Springfield USA 1976 p523
16. Maron BJ et al Blunt impact to the chest leading to sudden death from cardiac arrest during sporting activities. New England Journal of Medicine (1995); 333(6):337-342.
17. Bywaters E & Beall D Crush injuries with impairment of renal function. British Medical Journal (1941)1:427-432.
18. Bell MD et al Positional asphyxiation in adults. American Journal of Forensic Medicine and Pathology(1992);13(2):101-107.
19. Reay DT et al Effects of positional restraint on oxygen saturation and heart rate following exercise. American Journal of Forensic Medicine and Pathology (1988); 9(1):16-18
20. Reay DT et al. Positional asphyxia during law enforcement transport. American Journal of Forensic Medicine and Pathology (1992); 13(2):90-97

21. Luke JL. Reay DT. The perils of investigating and certifying deaths in Police custody. American Journal of Forensic Medicine and Pathology (1992);13(2):98-100
22. Laposata EA Positional asphyxia during law enforcement transport. American Journal of Forensic medicine and Pathology (1993);14(1):86-87
23. Schmidt P, Snowden T. The effects of positional restraint on heart rate and oxygen saturation. Journal of Emergency Medicine (1999); 17(5):777-782
24. Chan TC et al. Restraint position and positional asphyxia. Annals of Emergency Medicine (1997);30:578
25. Chan TC et al. Reexamination of Custody Restraint Position and Positional Asphyxia. American Journal of Forensic Medicine and Pathology (1998);19(3):201-205
26. Reay DT, Howard JD.Restraint position and positional asphyxia. American Journal of Forensic Medicine and Pathology. (1999);20(3):300-1.
27. Roeggla M et al Cardiorespiratory responses to hobble restraint. Wiener Klinische Wochenschrift (1997);109(10):359-361
28. Parkes J Sudden death during restraint: a study to measure the effect of restraint positions on the rate of recovery from exercise Medicine Science and the Law (2000); 40(1):39-44
29. Parkes J Sudden death during restraint; do some positions affect lung function?Medicine, Science and the Law (2008);48(2):137-41
30. Masters KJ, Wandless D Pulse Oximetry during restraint episodes. Psychiatric Services (2005);56:1313-1314
31. Meredith C et al Effect of wrist restraint on maximal exercise capacity in healthy volunteers American Journal of Forensic Medicine and Pathology (2005); 26(2):117-120
32. Gulino SP et al Restraint asphyxia. American Journal of Forensic Medicine and Pathology (2000);21(4):420
33. Glatter K, Karch SB Positional Asphyxia; inadequate oxygen or inadequate theory? Forensic Science International (2004);141(2-3):201-2
34. Chan TC et al. Weight force during prone restraint and respiratory function. American Journal of Forensic Medicine and Pathology (2004); 25(3):185-189
35. Pollanen MS et al Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. Canadian Medical Association Journal (1998);158(12):1603-1607
36. Siebert CF & Thogmartin JR Restraint-related fatalities in mental health facilities. American Journal of Forensic Medicine and Pathology (2000);21(3):210-212
37. O'Halloran RL Re-enactment of circumstances in deaths related to restraint. American Journal of Forensic Medicine and Pathology (2004);25(3):190-193
38. Langlois N & Gresham G. The age of bruises; a review and study of the colour changes with time. Forensic Science International (1991); 50:227-238
39. Betz P & Eisenmenger W. Morphometric analysis of hemosiderin deposits in relation to wound age. International Journal of Legal Medicine (1996); 108(5):262-264.
40. Knight BH "Contusions or bruises" in Forensic Pathology 2nd edition. Edward Arnold London 1996 p143
41. Heptenstall RB. Fracture Healing in Fracture treatment and healing. WB Saunders. Philadelphia pp35-64
42. Yaqub B, Al Deeb S.Heat stokes; aetiopathogenesis, neurological characteristics, treatment and outcome. Journal of the Neurological Sciences (1998); 156:144-151

43. Huerta-Alardin AL et al Bench-to-bedside review: Rhabdomyolysis – an overview for clinicians Critical Care (2005); 9(2):158-169
44. Harrelson GL et al Acute exertional rhabdomyolysis and its relationship to sickle cell trait. Journal of athletic training (1995);30(4):309-312
45. Glabow P et al The spectrum of rhabdomyolysis. Medicine (1982);62:141-152.
46. Schafer M et al Hazard of sauna after strenuous exercise. Annals of Internal Medicina (1994);120:441-442.
47. Iten PX and Meier M. Beta-hydroxybutyrate: an indicator for alcoholic ketoacidosis as a cause of death in deceased alcohol abusers. Jounal of Forensic Science (2000);45(3):624-632
48. Vasuvattakuly S et al The spectrum of endemic renal tubular acidosis in the North-East of Thailand. Nephron (1996);74(3):541-547
49. Toth HL and Greenbaum LA Severe acidosis caused by starvation and stress. American Journal of Kidney Disease (2003);42(5):16-19
50. Hick JL et al Metabolic acidosis in restraint-associated cardiac arrest: a case series. Academic Emergency Medicine (1999);6(3):239-243
51. Kloner RA Natural and unnatural triggers of myocardial infarction. Progress in Cardiovascular Diseases (2006);48(4):285-300
52. Meisel SR et al Effect of Iraqi missile use on incidence of acute myocardial infarction and sudden death in Israeli citizens. Lancet (1991); 338:660-661.
53. Leor J et al. Sudden cardiac death triggered by an earthquake. New England Journal of Medicine (1996); 354(7):413-419
54. Lown B et al Roles of psychological stress and autonomic nervous system changes in provocation of ventricular premature complexes.American Journal of Cardiology (1978);41:979-985
55. Ujhelyi MR Scared to death Pharmacotherapy (2008);28(11):1311-1314
56. Jelen P and Zagrodzka J. Heart rate changes in partially restrained rats during behaviorally and pharmacologically evoked emotional states. Acta Neurobiologiae Experimentalis (2001);61(1):53-67
57. Irvine RJ et al The influence of restraint upon blood pressure in the rat Journal of Pharmacologic and Toxicologic methods (1997);38(3):157-162
58. Kirby DA et al Behavioral arousal enhances inducibility and rate of ventricular tachycardiaAmerican Journal of Physiology (1991);261:H1734-H1739
59. Johnson DA Catecholamines in cerebrospinal fluid are increased by behavioral arousal and myocardial ischemia. American Journal of Physiology (1992); 263:H83-H87
60. Liang BRL et al Correlation between circulating catecholamine levels and ventricular vulnerability during psychological stress in conscious dogs. Proceedings of the Society of Experimental Biology and Medicine (1979);161:266-269
61. Verrier RLA et al Effect of posterior hypothalamic stimulation on the ventricular fibrillation threshold. American Journal of Physiology (1975);228:923-927
62. Briese E and Cabanac M Stress hyperthermia; physiological arguments that it is a fever Physiology and Behavior (1991);49:1153-1157
63. Zethof TJJ et al Stress-induced hyperthermia in mice: a methodological study Physiology and Behavior (1994);55:109-115
64. Mohr WK and Mohr BD Mechanisms of injury and death proximal to restraint use Archives of psychiatric nursing (2000);6:285-295

65. Critchley HD et al Mental stress and sudden cardiac death; asymmetric midbrain activity as a linking mechanism Brain (2005)128:75-85
66. Saffitz JE Sympathetic neural activity and the pathogenesis of sudden cardiac death Heart Rhythm (2008); 5:140-141
67. Stratton SJ et al Factors associated with sudden death of individuals requiring restraint for excited delirium. American Journal of Emergency medicine (2001); 19:187-191.