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SUPPLEMENTARY REPORT

A. INTRODUCTION

1. Following my testimony on the 06 and 07 February 2020 at the re-opened inquest of the late Dr Neil H Aggett, this report responds to specific questions posed on 14 February 2020 by the legal team of Webber Wentzel Attorneys to myself.

2. I was also given electronic copies of the original police Occurrence Book and Meal Register which recorded the daily activities of the holding cells and Dr Aggett' meals respectively.

B. QUESTIONS AND RESPONSES

1. With the post-mortem findings on record, and given the possibility that Dr Aggett may have been rendered unconscious before his suspension and which you averred in oral evidence cannot be excluded, you indicated five possible causes of unconsciousness in this context, as follows: (1) Intoxication of some sort; (2) Concussion (traumatic); (3) Electric shock-induced; (4) Hypoxia-induced; and (5) Hypoxia/Anoxia from carotid arterial obstruction.

   Elaborate for each of the above five possibilities by describing:

   a) Its mechanism, rapidity of onset, and period of unconsciousness;
   b) Whether and after how long could actual brain damage occur;
   c) Whether Dr Aggett could recover without medical assistance;
   d) Whether such mechanism could be excluded on the post-mortem findings.

The enquiry points raised above cannot be answered with accuracy as there is no scientific experimental data on human cases, and any answer would be based heavily on the application of pathophysiology and experience of similar or related cases as may have been experienced or reported in the scientific literature.

It is necessary to first distinguish between concussion and structural brain injury:

Concussion (or traumatic concussion) is a transient neuro-electrical paralytic disorder of brain function immediately after a blunt mechanical impact to, or transmitted to, the head and with no visible resultant structural damage to the brain); no physical brain damage is seen but it is a *functional* condition. It may manifest with a temporary loss of consciousness, amnesia (loss of memory) as well as post-concussion symptoms of headaches, dizziness, confusion, nausea or vomiting, fatigue, concentration loss and a stunned and dazed state to onlookers. Other symptoms include sensitivity to light (photophobia), blurry vision and interrupted sleep. These could lead to various emotional and cognitive issues. Post-Concussion Syndrome may be worse in people who have had previous concussions or head trauma. It may also be more severe in those who have earlier symptoms of headache or who have experienced mental changes such as amnesia, fogginess or fatigue. Following concussion, the brain enters a state of altered physiology and homeostasis. Immediately following head injury, cerebral metabolic rate increases and may account for the initial alterations in consciousness.[1] [2] In its common mild state it is frequently seen in contact football sports or falls or assaults. There may be no features of a scalp or facial impact visible. Recovery is usually complete. This cannot be excluded in Dr Aggett's circumstances.

Traumatic brain injury (TBI) of a structural nature (actual visible brain damage) on the other hand may be due to any significant physical head impact. There was none of this seen in Dr Aggett.

[1] Bergsneider M, Hovda DA, Shalmon E, et al. Cerebral hyperglycolysis following severe traumatic brain injury in humans: a positron emission tomography study. *J Neurosurg* 1997; 86:241–251;

[2] Yoshino A, Hovda DA, Kawamata T, Katayama Y, Becker DP. Dynamic changes in local cerebral glucose utilization following cerebral conclusion in rats: evidence of a hyper- and subsequent hypometabolic state. *Brain Res* 1991; 561:106–119.

Returning to possible contributory factors to Dr Aggett's death:

Intoxication by alcohol, or drugs which were tested to a limited extent, were said to have been excluded at the post-mortem. In my oral evidence I advised that the drugs testable by the public/state health laboratories were limited then in 1982 in the spectrum (number of possible toxins and drugs), sensitivity and specificity of the techniques and the level of laboratory mechanisation. This raises the possibility of the administration of a drug or drugs that could not be identified at that time. This possibility must, however, be verified by a toxicology expert.

Traumatic concussion may have occurred and cannot be excluded from the autopsy findings of Dr Aggett, despite the absence of any scalp/skull/brain/facial injury. The onset of concussion would be immediate after blunt injury (of direct impact type or by indirect transmitted type) and can persist for days or longer. If the mark on the forehead of Dr Aggett allegedly seen by certain witnesses were to have been a bruise, this may have been evidence of direct head impact. In traumatic concussion actual visible brain damage does not occur. Regaining consciousness if lost can be very quick in the mildest of cases but can take many minutes or more in more severe cases, and the post-concussion symptoms and signs can persist for many hours or even days or weeks.

Electric shock-induced unconsciousness is possible with any electrical injury case with certain high-enough current and voltage levels, especially if convulsions were to occur. Generalised convulsions certainly can occur with electrical shocks in general. However, it is not known to me what the current and voltage levels were as used in such shock treatment by the police in this context, except that with hand-operated cranking devices it may usually be low current and voltage and I am unable to expertly confirm whether this could cause generalised convulsions and unconsciousness, aside from painful, distressing and disabling muscle contractions, and possible skin surface burns.

Hypoxia-induced unconsciouness from airway occlusion cannot be excluded from the autopsy findings of Dr Aggett. If an individual is deprived of air (and thus of oxygen), his progression to unconsciousness would depend upon his state: - if calm,

as for example in voluntary situations such as with free or deep-sea diving, fit and conditioned persons may remain conscious and continue so for several or multiple minutes without active breathing; - if agitated, stressed, distressed and anxious, the person would succumb to unconsciousness within a minute or two.

Regarding the effects of the hypoxia and recovery:

(1) If the airway patency and respiration are immediately restored, such persons would regain full consciousness within several minutes, and there are usually no long-lasting effects;

(2) If the airway obstruction or air deprivation is maintained for longer, brain neurons will suffer damage within 5 to 6 minutes of complete hypoxia (anoxia). Some authoritative sources record that brain neuronal damage may occur in as short as 4 minutes, depending upon what the oxygen demand was and how rapidly consumed (Dr Aggett may be considered to have been at that stage in a state of exhaustion, panic and distress and in very high oxygen demand). This can cause permanent brain damage with no full recovery of function, or death if the hypoxic/anoxic state is marginally prolonged any further than that.

In either of the two instances above, if the death occurred with Dr Aggett soon after the hypoxic/anoxic incident, there would be nothing to be seen at the post-mortem that would point to this having occurred. This is because at least about 12 hours of *survival* after the hypoxic/anoxic incident is needed to even detect the earliest manifestations (whether by naked-eye or by histology under microscope) of hypoxic brain damage in a fatal case.

Hypoxia/anoxia of the brain from actual bilateral carotid arterial obstruction: Such would cause immediate unconsciousness (at least within about 15 seconds) if bilateral occlusion (by pressure upon both sides of the neck). If the blood supply to the brain is obstructed for approximately 1 minute (60 seconds)[3] at the earliest or

[3] Sixty-five (65) seconds was the shortest time for death after circulatory arrest in a review article [Gardiner D et al International perspective on the diagnosis of death *British Journal of Anaesthesia* 108 (S1): i14–i28 (2012)], whilst the longest was 11 minutes.

more (can be several minutes in some instances), death can supervene. Here too, no findings would be seen of this in the brain itself if an autopsy were conducted because there would be no appreciable survival period whatsoever, unless something such as bruises were to be seen within the neck where the carotid pressure was applied. Attention must be drawn to the finding of a bruise at the right carotid sheath and at the left horn of the hyoid bone in Dr Aggett's body, but this could be also attributable to the ligature itself as in hanging.

The question as to how soon such bilateral arterial obstruction could cause brain damage but not death itself would be difficult to answer, except that the following points can be confidently made:

a. The brain is damaged by death of neurons from lack of oxygen (blood).

b. When the vital neurons of the *brain-stem* die, respiration ceases and full death as we know it of the individual occurs.

c. Brain stem vital neurons are more resistant to lack of oxygen than the neurons in the cortex (outer surface) grey matter. It is therefore more likely to have cortical neuronal damage earlier that that of the brain stem. If at some earlier stage (probably at 1 minute or earlier) blood circulation is restored, the cortical grey matter neurons could already be dead but the brain-stem may still be alive. In other words, the person would still be alive but brain-damaged and unconscious (in a coma). At what precise stage the individual cortical neurons would die is not known, but there is possibly a drop-out of individual dying neurons in an unpredictable and random manner.

d. Cortical brain damage (without brain-stem death) would render the victim largely permanently and irreversibly unconscious.

Death by carotid obstruction/lack of blood circulation to the brain-stem) can be excluded since the deceased was alive at the time of the hanging. However, what cannot be excluded is brain damage from carotid obstruction arising from the destruction of cortical grey matter neurons, and the subject remaining alive if blood circulation was restored timeously, resulting in irreversible unconsciousness. As

mentioned, such brain damage would not be detectable at an autopsy if the subject died by other means shortly thereafter.

It must be finally added that the five possible causes of a state of unconsciousness proposed above, if such unconsciousness had occurred, would not have been mutually exclusive to one another, and that a combination of one or several of these occurring together is not excluded, and if so their individual effects would be additive to the whole.

2. According to the "Meal Register"(B8.59) completed for each meal of the prisoners at the John Vorster Square, Dr Aggett took no meals between the [Thursday] 28 January 1982 and [Monday] 02 February 1982. Comment on the ability of the deceased to execute the hanging manoeuvre if the hanging was self-imposed.

According to the Occurrence Book, the periods of his interrogation sessions were as follows:

Date	Booked out	Returned	Period	In between
Thurs 28-01-82	08h25 16h09	14h41	>7 hrs	1 hr 50 min
Sun 31-01-82		03h30	>59 hrs	<29 hrs
Mon 01-02-82	08h26	15h30	>7 hrs	<17 hrs
Tues 02-02-82	08h17	15h41	>7 hrs	>17 hrs
Wed 03-02-82	08h20	11h30	>3 hrs	>15 hrs
Thurs 04-02-82	08h37	15h44	>7 hrs	±8 hrs before death

According to the Meal Register:

i. The meal on 28 February consisted of 6 slices of bread, tea, and 2 eggs.
ii. The next recorded meal on 02 February of 4 slices of bread, cereal, mince, rice and tea. No meal times are recorded.
iii. The interval between these meals amounts to approximately three (3) days or seventy-two (72) hours without recorded meals.

The content and nature of these recorded meals was also largely non-nutritious. Even on the assumption that Dr Aggett was given something to eat during the lengthy period of the long weekend of interrogation on the 10th floor of the John Vorster Square, the almost-certain lack of adequate food would have been most detrimental to whatever remaining energy and muscular ability he would have had along with the continued and accumulated stress of the repetitive interrogations, physical exertions, sleep deprivation, bodily pain and insufficent time for recovery in between sessions.

As testified by myself, the magnitude of muscular action is shown to be less affected by sleep deprivation but there is a much shorter time-to-exhaustion, whereas the combined effects of physical bodily injury and physical exertions, inadequate nutrition, together with a prolonged sleep-deprived state, would in my view be additive and potentiate the impairment of muscular coordination and focus towards synchronised and purposeful activity, This assumes Dr Aggett had not already reached a state of disorientation, disordered thinking and hallucinations or close to a psychosis or toxic delirium by that time (refer to my reference Waters F, et al. Frontiers in Psychiatry. July 2018 Vol 9, Article 303" in my main report).

3. **You have seen pictures of Dr Aggett's cell bars during your testimony, including the narrow-edged horizontal traverses. You have indicated that you could not attribute the fresh triangular bruise on Dr Aggett's back to 'pinching' or convulsing on these bars during the act of hanging. Advise on what sort of injury you would have expected such convulsions to produce. Further, suggest as to what could have really caused this bruise given its size, shape and description as 'fresh'.**

The precise nature of this lesion remains unclear. It is important to consider that the only evidence that this triangular and red lesion of the upper right side of the back was a bruise identified by Dr V D Kemp. This "bruise" was not reported on by Dr Botha and was not taken for histology testing towards its objective confirmation,

In these circumstances possibilities other than a bruise (causing a triangular red mark to naked-eye examination) cannot be excluded. If the apparent "bruise" was not a

bruise proper but an erythema (reddened area of skin such as might have been seen fleetingly on Dr Aggett's forehead), the possibility of causation by application of a surface that may have been heated, or by scalding, or alternatively being chemically or similarly irritated, or an electrical burn cannot be excluded.

On the assumption that it was a bruise, (apart from forming the view that the triangular lesion was most probably sustained before the hanging), it is difficult to suggest what could have been the cause, except that it appears to have been from a patterned instrument or object or surface resulting as a blunt impact injury by direct forceful impaction or by heavy sustained pressure distorting the skin at that point.

The transverse bars of the cage against which Dr Aggett's body posterior (back and limbs) lay were angular only at their upper and lower edges, the portion of the bars against the actual body being somewhat flattened. The flat bar surfaces against the body are also otherwise smooth and not irregular or roughened or coarsened with concretions or irregularities of any sort .

If there were terminal convulsions after descending or dropping to the full stretch of the ligature around his neck, I would firstly expect no real visible injuries at the back of the trunk. This is because the repetitive knocking of any part of his back against the flat bars during convulsions, even if vigorous, would in my view not be enough to manifest with abrasions or bruises. Any transverse "impressions" caused by the weight of the body against the bars would be due to pressure-dispersion of tissue fluid only, but this will rapidly regress and disappear after the body was laid onto the floor of the cell and at the mortuary on a flat trolley before the autopsy.

In any event, as it is my confident view that Dr Aggett died very shortly after the constriction of the neck by the ligature (within a few seconds at the most), there would not have been enough time for any significant bruising to "evolve".

4. **Witness Jabu Ngwenya's original affidavit of June 1982 has been shown to you. The witness described that when he was taken to the Protea police station on the 17 November 1981 his clothes were taken off, hands handcuffed behind his back, a canvas bag drawn over his head, he was ordered to lie down, his hands and legs**

were tied together, four (4) wires stuck onto his shoulder-blades, and shocked through these wires for a "fairly" long time – this done repetitively. Ointment was placed on the places (shoulders) that he had been shocked. Comment on whether the account above could be matched against the four (4) scars on Dr Agett's back.

RESPONSE:

The four (4) scars of Dr Aggett were also located around his shoulder blades. Dr Kemp description of them were as "old healed scars" and his dimensions, although vague, suggested that these were each at 9mm in diameter, and may be interpeted as possibly all of the same size and possibly shape as well. The concurrence of size and shape, and resemblance to that which was experienced by Ngwenya over the same part of his body, and its discrete focality that may be comparable to electrodes on the skin, are compelling coincidences and raise the possibility that Dr Aggett's scars were also due to the same type of electrical injury.

Dr Botha described these lesions as "puckered scars" indicating their surface irregularity, and on microscopy (histology) "mild epidermal hyperkeratosis and acanthosis" (thickening of the outer cornified layer and of the squamous layer of the epidermis respectively). This histology showed that the scars were most superficial, i.e. involving only the very upper layer of skin – the epidermis. These very superficial changes may occur with a skin irritiation or chronic or repetitive skin injury. My impression is that electrical burn marks of low voltage cannot be excluded as having caused these scars on Dr Aggett's back. Low-voltage skin electrical burn marks would first be more superficial than deep, and if repetitive at the same spots may be expected to appear initially inflamed but also to swell and blister, the blister at each would then collapse, and with healing develop into a puckered scar. The hyperkeratosis and acanthosis seen on microscopy cannot be excluded as being the consequences of such superficial damage by electrodes bearing an electrical current..

C. OTHER POINTS RAISED IN CONSULTATIONS

1. **I have been advised that Brigadier Swanepoel found Dr Aggett's body cold and stiff at 03h45 and that rigor mortis had begun. What effect does this evidence have upon the time of death?**

I regret I did not note or have this information during my initial analysis. I must declare that I would be most cautious in unreservedly accepting the findings of the brigadier (him not being a forensic physician). The perception of "cold" and "stiff" are particularly important early after-death findings that require some training and much experience as they are very subjective perceptions on examination.

However, on the assumption that this was in fact the actual appearance of the body, cold and stiff would mean that the body had cooled down to at least perhaps room temperature, and that rigor mortis had set in to the large muscles of the trunk and limbs (which needs at least a minimum of, more or less, 2 to 3 hours just to begin and between 6 and up to 12 hours to be "set" into rigor). It would then suggest that Dr Aggett was possibly dead for at least two to three hours or more at 03h45.

2. **Would assaulting a detainee after they were made to perform exercises cause the injuries from assault to be less visible or difficult to detect?**
   No. Neither exercise, no medication, nor any balm applied, would affect the visible manifestation of injuries at an autopsy.

3. **Would an interrogator - from observing a detainee – be able to gauge with any accuracy during asphyxiating techniques when this had to cease to avoid unconsciousness, collapse and/or death?**
   This is not known to me.

   What is known is that when "accidents" happen in these scenarios, the bodies are likely to be examined by pathologists at autopsies. We have noted in fatal cases where it is alleged that asphyxiating techniques (such as a dry plastic bag, or "tube", or wet bag or waterboarding, etc.) are used by police on detainees, some of the "eyewitness" accounts are very similar: there is a sudden cessation of screaming by the victim, and water is then hurriedly thrown upon the collapsed victim's face in an attempt to revive him.

4. **Could any of the findings that Dr Aggett was hung alive (but whilst unconscious) have been influenced by the shortcomings (6.2 of your main report) you identified of the post-mortem examinations?**

As per my testimony, the post-mortem medical findings as found cannot distinguish between the two scenarios of conscious/self-infliction versus unconscious/hanging by another. The following can however be stated:

a) Had a chief or senior pathologist or experienced district surgeon attended the scene it is difficult to say what opinion he would have held upon whether the deceased would have been able to carry out the hanging manoeuvre himself. This would have been a view based on his own assessment, insight, attitude and perspective.

b) The undue haste in conducting the autopsy may have compromised Dr Botha's full assessment of the injuries (it must be remembered that he did not make a finding of the triangular bruise of the back reported by Dr Kemp).

c) The failure to examine for deep-seated bruises) may have facilitated non-detection of deeper injuries that might have pointed to more severe and extensive physical bodily trauma.

.....Dr S R Naidoo

DATE: 23 March 2020

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