CASE NUMBER: CV16C00513

SUMMARY SCHEDULE OF EXPERT OPINION

Mr William Newman – Opthalmology – Retinal Haemorrhages		
Reports	Substantive report dated 11.07.16	E10-85
Transcript	Evidence to HHJ Cleary – 5.10.16	J162-188
1.	Overview '*There has been no disclosed history of severe accidental trauma and investigations have not identified any known underlying medical condition. In my opinion, the retinal haemorrhages seen are not due to birth, immunisations, vomiting, seizures, raised intracranial pressure or minor trauma'.*	E12
2.	Extent and appearance Spread and appearance: bleeding in all four quadrants and multilayer – more in the left eye than the right eye.	
3.	Timing Retinal haemorrhages likely to been caused within the 17 days prior to their last identification (so 5.4.16 onwards) Consistent with having occurred at or around the time that M became acutely unwell on 19.4.16	

4.	<u>Likely causation and differential diagnoses</u>	
4.1	The haemorrhages are '*most consistent with a shaking or shaking with impact injury'*.	
4.2	The pattern and spread haemorrhaging seen in M's eyes are consistent with shaking / shaking impact injuries.	
4.3	It is not possible to determine from the type / distribution / quantity of retinal haemorrhaging alone whether it is more likely to be a shake or a shake-impact injury.	
4.4	Statistically (rather than case-specific) 80% of cases of non-accidental shaking injuries include retinal haemorrhaging. Typically there is bleeding within multiple layers of the retina.	
4.5	There is nothing to suggest an underlying disorder or deficiency that would or could result in these haemorrhages.	
4.6	The haemorrhages '*are in my opinion not related to minor trauma, seizures, vomiting, hypertension, immunisations or raised intracranial pressure'*	
4.7	Where haemorrhages in the retina are caused by raised ICP one would expect them to be concentrated around the optic disc, which is often swollen. It was not in this case. Where, hypothetically, there *is* raised ICP in a case and the optic discs look normal '*you don't normally see retinal haemorrhages'*	J173-174
4.8	Normal and / or rough handling of a child is '*very unlikely'* to cause the retinal haemorrhages seen in M's eyes. The same applies to attempts to rouse an apparently unconscious child.	
	'*The combination of findings of retinal haemorrhages, intracranial bleeding and breathing problems are much more likely to be associated with inflicted brain injury than accidental head injury'*	

5	Mechanism	
5.1	*'The cause of retinal haemorrhages in shaken injury is considered to be due to the shearing forces caused by shaking upon the vitreous within the eye. The vitreous gel is strongly adherent to the peripheral retina, around the optic disc and retinal vessels. The movement of the gel during shaking is thought to exert traction and 920 shearing forces together with induced local tissue changes at the interface of the vitreous and retina resulting in haemorrhages and in some cases causing splits and or folds within the retina'.*	E39-40
5.2	A shake is likely to require significant force to generate the required motion – certainly one which would be clear to an observer and to the actor him or herself that it was inappropriate.	
5.3	Likely mechanism: '*The current view, and I agreed with that view, is that it's a multiple-faceted injury, partly caused by rotational forces and the rotation of the gel within the eye, partly caused by the hypoxic and change in the blood vessels' flow and probably likely due to trauma within the orbit (that's behind the eye and where the blood vessels actually go into the eye) and it's likely that there is some haemorrhage that occurs within that'.*	J177
6.	Level of force. We '*don't know what the true incidence is or what the threshold is for causing the retinal haemorrhages',*	J176
7.	Other	
7.1	Retinal haemorrhages indistinguishable from those associated with inflicted head injury commonly occur at birth. They also occur (with less frequency) in 'pure' elective Caesarian births. We do not understand the mechanisms that cause them, or why they occur	J178-179
7.2	The retcam images are of poor quality but were taken in a conventional manner and confirm the findings of Fiona Dean, the opthalmologist, who examined M on 20.4.16	J169, J182

7.3	'*There is nothing contained therein to suggest that there was any pre-existing eye condition or pre- existing medical condition or any inherited medical condition that would result in, or lower the threshold for, retinal haemorrhaging'*.	E19
7.4	Retinal haemorrhages '*don't necessarily trump anything else'*.	J181
7.5	There were '*no signs from an ocular point of view of raised intracranial pressure'*	J184
7.6	CPR would not cause the retinal haemorrhaging	J185
7.7	Attempts to rouse an unconscious child – would not result in retinal haemorrhaging as seen here and '*it's a common question and it was assed by the Royal College in its Guidelines. And we don't find it'*	J186
7.8	Nothing in the oral evidence has caused Mr Newman to change the opinions in his report	J186

<u>Mr Peter Richards – Consultant Paediatric Neurosurgeon</u>		
Reports	Substantive report dated 15.07.16	**E86-113**
Transcript	Evidence to HHJ Cleary – 30.09.16	J117-161
1.	**<u>Qualifications to review scans</u>**	

1.1	I have reviewed the CT scan of 19.4.16. Interpretation of infant CT scans is a core skill of paediatric neurosurgery which I carry out on a daily basis. I have received appropriate neurosurgical training in this but have not undergone formal radiological training and have no radiological qualifications. I have reviewed the MRI scan of 22.4.16. As with infant CT scans, review of infant MRI scans is a standard technique in paediatric neurosurgery which I perform on a daily basis and for which I have received appropriate neurosurgical training but I have not received any neuroradiological training or any specialist neuroradiology qualifications.	E92-93, E96
1.2	Accepts in XX (PSQC) that he will see things when operating that indicates the neuroradiological interpretation was wrong and that no too much importance should be attached to radiology alone, including where interpretation of dark fluid (ie equivocal in its appearance as between ATE and chronic) is in issue (XX by JVQC)	J124, J137
2.	**Extent and appearance of bleeding / brain injury**	

2.1	Over the surface of both cerebral hemispheres and the left cerebellar hemisphere there is intermediate density fluid within which there is also white material – seen in both cerebral hemispheres and in the region of the falx and posterior fossa.	E93
2.2	MRI scan of 22.4.16 – mixed intensity fluid seen over the surface of all intracranial compartments, within which there are areas with signal compatible with recent haemorrhage.	E96
2.3	No signal change to suggest hypoxic / ischaemic change.	E96
2.4	The fluid seen in the subdural space could either be a mixture of fresh blood and CSF (acute traumatic effusion) or a previous (chronic) subdural haematoma. The two can be difficult to tell apart, radiologically.	
2.5	However, the absence of visible subdural membranes on the scan, coupled with the absence of any sign of enlarging head circumference or separation of the skull sutures makes it likely that the fluid seen is an acute traumatic effusion.	
2.6	Chronic subdural collections commonly '*just keep growing and growing and growing*'. The child's presentation at hospital after acute collapse following by brief hospital treatment and no invasive surgery points away from the existence of a chronic subdural collection because nobody has done anything about the subdural collection '*and yet it hasn't continued to grow'*.	E153, J154
2.7	A re-bleed from a chronic collection is also rendered less likely by the location of the bleeding. Rebleeds '*tend to occur in one compartment as opposed to multicompartments and are usually clinically silent and not associated with sudden colllapse'*, although if caused by minimal trauma then '*the same forces could*	E100, J155

	influence the membranes in all three compartments'	
2.8	Acute traumatic effusions are associated with recent head injury which, together with M's clinical presentation on 19.4.16 further support the contention of a recent event.	
2.9	'*I would not expect a chronic subdural haematoma re-bleed to cause an acute encephalopathy of sudden onset'*.	E110
2.10	In the absence of a recognisable identifiable disease a known cause of fresh subdural bleeding, particularly multicompartment fresh subdural bleeding, is following a recent episode of head injury.	E99
2.11	Clinical experience is that the development of a chronic subdural haematoma would not be expected to cause retinal haemorrhaging	J151-152
2.12	Given the combination of moderate encephalopathy with the neuroradiological findings (fresh subdural blood in all three intracranial compartments and mixed intensity fluid in these compartments) and retinal haemorrhaging – the most likely cause is a '*recent episode of head injury'*.	E101
2.13	When attended by paramedics and on arrival in hospital M's presentation indicated acute moderate encephalopathy.	E107
2.14	In the absence of identifiable medical disease, a recognised cause of a sudden onset of encephalopathy is following a recent episode of head injury.	E99
2.15	The point of collapse on 19.4.16 is likely to be the point of injury.	E106

2.16	If, contrary to his and to Dr Stoodley's opinion, the court finds that the child had a chronic subdural collection, this not only would not account for the evidence of multicompartmental fresh bleeding, but would itself have to have a primary cause .That is unlikely to have been birth. In the circumstances, the '*commonest cause is a shaking injury that would have occurred some weeks before the scan of 19th April 2016. This would imply at least two episodes of inappropriate handling of M'*	E105
2.17	If caused non-accidentally and on the one occasion shortly before admission, the injury would be consistent with a momentary loss of control on the part of the adult carer.	J120
2.18	Discussion re ALTEs (acute life threatening events) and an adult's possible inclination to shake a child experiencing one discussed– referring to the research (in which this was a side issue)	J120-123
2.19	Fluid can move within the dura	J127
2.20	Different reasons for subdural bleeding discussed	J128-129
2.21	Birth-related subdural, leading to formation of chronics discussed and limitations of Rooks / Looney / Whitby birth-related subdurals considered. Accepts that birth-related subdurals could hypothetically lead to formation of chronics going beyond the 3 month time period of the research but later notes that '*birth subdurals developing into chronic ones, it's not been demonstrated as happening yet',* but '*some people interpret [this] as meaning birth subdurals never become chronic. I personally don't accept that, it's just we haven't shown it as yet'.*	J129-131, J155
2.22	Birth-related or other chronic subdural '*on radiology alone' 'remains a possibility…but if you throw in the clinical side, I think it's very, very unlikely'* but cannot be excluded completely *(*XX by PSQC)	J131

2.23	'*If you take into account the eyes and the bruises*[1] *I can't see how simply a re-bleed into a chronic subdural haematoma could explain either of those. You would need other explanations for that'.*	J157
2.24	Acutely raised ICP (intra-cranial pressure) unlikely. Deviating eyes suggest seizure activity instead. '*it is unlikely, given a child who is conscious, it is very unlikely to be significantly and critically raised intracranial pressure that's doing it'.*	J133
2.25	*If* a pre-existing chronic collection was present then any action taken (whether inappropriate handling or response to a perceived ALTE) could result in a subdural rebleed	J136
2.26	A child with abnormally enlarged subarachnoid spaces has a predisposition to acute subdural bleeding	J140-141
2.27	If a chronic collection *were* to be present then the cause of the acute bleed may be unknown, and the chronic subdural itself represents a predisposition towards further acute bleeding and may be caused by minimal or no trauma	J141
2.28	If subdural blood causes any symptoms simply by virtue of its presence, those symptoms are likely to be seizures;	J149
2.29	Head circumference – the charts are so abnormal as to suggest '*one of these measurements is very inaccurate'.* Were the head circumference to have increased by 2 ½ centimetres in 12 days '*you'd expect this child to be very very ill indeed'* in the January of 2016, with a an underlying organic cause, such as a cyst / tumour	J142-143, J147-149, J155

[1] Note – bruising not pursued in light of Dr Cartilidge's evidence so this falls away

Dr Neil Stoodley – Paedatric Neuroradiology – Head / Brain Injuries		
Reports	Substantive report	**E114-129**
Transcript	Evidence to HHJ Cleary – 29.09.16	J1-39
	The abnormalities seen on M's scans are likely to be due to an episode of abusive head trauma involving a shaking mechanism. There is no definite scan evidence to suggest more than one such episode.	E116
1.	Interpretation of scans	
1.1	CT scan of 19.4.16 There is evidence of high attenuation (bright) material in the posterior interhemispheric fissure on both sides, over the left frontal region and in the posterior fossa. All of this bright material is acute (recent) subdural blood. It is not possible to assess accurately the age of the blood on the basis of the scan appearances alone as acute blood can appear bright on CT from soon after an episode of bleeding for up to 7-10 days. There is also a small focal area of acute probable subarachnoid haemorrhage in the right frontal region.	E119
1.2	Reduced white / grey differentiation in the cerebral hemispheres is consistent with degree of hypoxic ischaemic damage	E120
1.3	CT head scan – dark subdural fluid seen over both cerebral convexities – this could represent either older subdural blood (at least 2-3 weeks) – ie chronic subdural collection or an acute	E119-120

	traumatic effusion ('ATE'). The latter is more likely. This would be consistent with a recent head injury.	
1.4	Disagrees with Mr Richards' interpretation of the scans: that there is white fluid within the darker subdural fluid: '*It clearly is not'*.	J27
1.5	Most of the bright material (ie acute bleeds) is at sites away from the dark subdural fluid. This is contraindicative of pre-existing chronic collections and is further support for the dark material being ATE as the acute bleeding, being at different sites, would not be explicable on the basis of any re-bleeding chronic collections: they are not anatomically related.	J28, E126
1.6	If, on the other hand, Mr Richards' interpretation of the scans (which sees darker fluid over all of the intracranial compartments and all of the convexities of the brain, then the acute bleeding could be explicable as re-bleeding chronics *if* the darker material is evidence of previously asymptomatic chronic collections	J28
1.7	Disagrees with hospital neuroradiologist that the scans show acute on chronic subdural haematomas	J29
1.8	MRI scan – 22.4.16	

	The scan confirms the distribution of acute subdural blood seen on the CT scan. The blood that was bright on CT is of high signal on T1 and low signal on T2. This suggests that the predominant blood breakdown product present is intracellular methaemoglobin. Whilst not absolute, this tends to be seen between around 3-7 days after an episode of bleeding.	
1.9	No evidence of membrane formation or loculation. This is supportive (although not categorically so) that the darker material is ATE rather than chronic subdurals	E120-121
1.10	When challenged on interpretation of scans and Mr Richards finding lower density fluid in all same compartments as acute blood, did not accept Mr Richards' interpretation but, if Mr. Richards' interpretation of the scans was right then his interpretation of the scans and understanding of the mechanics of rebleeding was wrong and the acute blood seen was anatomically related to the other, lower density fluid and would raise the possibility of a re-bleed.	J28
2.	Timing There is nothing in the clinical history which would lead me to suggest that any birth related subdural bleeding could have persisted until the time of the acute admission and any birth related subdural bleeding would not have still appeared bright on the CT scan.	E122
3.	Likely causation and differential diagnoses Accelerative / De-accelerative forces likely to be required to cause injuries:	E122-123

	have involved shaking. Majority medical opinion is of the view that what is likely to be required to produce such injuries is the repetitive backwards and forwards movement of the unsupported infant head pivoting on the neck. It is believed that this leads to a degree of acceleration / deceleration and rotational forces and that the consequent differential rotation of the brain and skull leads to stretching of the subdural veins which cross the subdural space and it is this that leads to the bleeding in the subdural space. Whilst this is certainly likely to be at least one mechanism by which subdural bleeding can occur (indeed it is sometimes possible to see focal haemorrhage associated with larger bridging vessels on scans), it is unlikely that this is the only mechanism by which subdural bleeding can occur as it is unlikely that this mechanism is, for example, the cause of birth-related subdural bleeding. The scan abnormalities are however explicable on the basis of being due to an episode of abusive head trauma. The mechanism of injury is likely to	
4.	<u>Degree of force</u>: Absolute degree of force unknown but 'we do not see these features following normal handling, typical domestic trauma or rough play'. Safe to conclude 'that the minimum degree of force required is likely to be such that an indpendent witness would regard it as being likely to lead to harm to the child, i.e.it would be obviously inappropirate'.	E123

Dr Patrick Cartlidge – Consultant Paediatrician		
Reports	Substantive report dated 06.08.16 Letter 17.08.16 Addendum report dated 19.09.16 Second addendum report dated 13.04.17	**E130-215** **E243** **E286-289** **E291-292**
Transcript	Evidence to HHJ Cleary – 29.09.16	J40-116
	Overall view	
1.	4.7. What is the most likely mechanism of the head injury? In my opinion, the subdural haemorrhages and the retinal haemorrhages were caused primarily by trauma, most probably by shaking, with or without an impact with a semi-yielding object. The absence of scalp swelling and a skull fracture makes it less likely that any impact was with an unyielding object. I think that the injuries were caused either non-accidentally, or in an accident that a carer has chosen to conceal.	E153, E155

	In my opinion the subdural bleeding, the acute traumatic effusions and the retinal haemorrhages were caused by trauma. I think that the injuries were caused either non-accidentally (probably primarily shaking), or in an accident that a carer has chosen to conceal.	
2.	<u>Acute Traumatic Effusion vs Chronic Subdural</u>	
2.1	Child's growth and consistent increase in head circumference is '*most consistent with the absence of a chronic subdural collection although it does not exclude the condition'*.	E145
2.2	However, the fact that the collection was shallow means that it would not necessarily have caused an increase in head circumference	J50
2.3	(Accepting Dr Stoodley's evidence): • There were no subdural fluid collections in the posterior fossa and so re-bleeding from a chronic collection cannot be the cause of acute subdural blood at this location.	E145
2.4	Chronic subdural collections would not explain the subarachnoid blood (accepting Dr Stoodley's interpretation of the scans). These indicate an acute injury.	E145
2.5	Chronic subdural collections could not explain the retinal haemorrhaging. These indicate a recent injury	E146, J61
2.6	The subdural collections were '*almost certainly sustained at the same time as the subdural bleeding (i.e. all features are explicable on the basis of a single event')*.	E146

3.	Discounts medical conditions and birth-related trauma, noting in respect of the latter that: • I do not think that M had chronic subdural fluid collections. • The acute subdural blood in the posterior fossa cannot be explained by a re-bleed since fluid collection was found at this site. • The subarachnoid blood cannot be birth-related. • I defer to Mr Newman with respect to the retinal haemorrhages, but I cannot envisage them being birth-related. In my opinion the intracranial lesions are not birth-related.	E150-152
4.	Does not think (XX by JVQC) that the child had an abnormal head circumference growth	J52-J56
5.	Thinks that the Semmekrot et al (Eur J Pediatr. 2010 Feb;169(2)) paper on Acute Life Threatening Events is not helpful in considering whether a parents' response in shaking a child who has experienced an ALTE could be sufficient to cause subdural haemorrhaging	J69-70
6.	If a child (without a pre-existing chronic subdural haemorrhage) were to be held by a parent in an unsupported way so that the head was wobbling back and forth while that parent ran downstairs and / or hit an object while running, while that cannot be excluded as a mechanism for causation of the subdural haemorrhaging, it would be unlikely (example given of where this was considered a possibility in a different case in the witness's experience). The account given by F in police interview of running, holding the child's head in a supportive way would not cause the subdural haemorrhaging. '*It only starts to become at all plausible with the extremes of hitting..or the head not in the palm of the hand*'.	J73-76, J81-87, J89-93

	[NB - Subsequently clarified by parents in evidence and by JS in XX (referencing F's police interview) that F was not running with the child in this way].	
8.	From the paediatric perspective the case 'boils down' to the robustness of the finding of subarachnoid haemorrhage, whether there is fluid in the posterior fossa, and the causes of the retinal haemorrhages;	J62
9.	On the basis of the clinical presentation of the child and scan appearances it cannot be said whether the injury was caused prior to or after the child appearing unwell or resuscitation of the child carried out by F and then by the attending paramedics.	J76,
10.	Does not think that there was a marked raised intracranial pressure. If raised it was '*probably of a moderate degree, a mild to moderate degree and that might help Mr Newman in interpreting whether that could be the cause of the retinal haemorrhages'*.	J104
11.	Does not think that it was necessary to do extended screening for platelet functioning disorder in light of child's clinical presentation and progress since.	J114

DATED 2.9.17