To the Court

Medical Report Prepared on:

NAME: Mrs ES

ADDRESS:

Date of Report: 12th May

Client's Date of Birth:

Instructing
Party's Reference:

Prepared by: Mr Jonathan Luck FRCS FRCOphth

Consultant Ophthalmic Surgeon at the Royal United Hospital, Bath
Bachelor of Medicine and Bachelor of Surgery at the
University of Manchester
Fellow of the Royal College of Surgeons of England
(1990)
Fellow of the Royal College of Ophthalmologists (1990)

(See also brief CV in Appendix)

PREPARED AT THE
REQUEST OF:

CONTENTS

1. INSTRUCTIONS

To prepare a report dealing with issues that fall within my field of expertise pertaining to Mrs S's claim against [redacted] Specifically I have been asked to consider the following questions:

a) To what extent do I feel the damage that occurred to Mrs [redacted]'s sight would or could have been avoided on the balance of probabilities?

b) If the concerns of the GP had been acted upon sooner by the Trust involved, would she in all likelihood have suffered as much damage?

2. SUMMARY OF CONCLUSIONS

a) In my opinion, the patient should have been referred to the Eye Department for consideration of a Temporal Artery Biopsy (TAB) on presentation on the 29th December, due to the combination of new onset headache, jaw pain, malaise, and raised inflammatory markers on blood testing which were suggestive of Giant Cell (Temporal) Arteritis (GCA). In my opinion it is more likely than not that a TAB at that time would have been positive, and subsequent prompt administration (and subsequent continuation) of an appropriate dose of corticosteroid medication (steroids) at that time may have on the balance of probabilities prevented visual loss in the left eye (the right eye being unaffected).

b) If the concerns of the GP (about GCA) had been acted upon then in my opinion it is more likely than not that the visual loss in the left eye would have been prevented.

3. DOCUMENTATION SUPPLIED

a) Documentation from the NHS complaint system and responses from the involved General Practice
b) Paginated medical records from General Practitioner and hospital sources
c) A Chronology from the patient

4. CHRONOLOGY / CASE ABSTRACT

4.01 An extensive chronology has been already provided by both the patient and by [redacted] the complaint advocate from the Community Health Council in [redacted] I propose to confine myself to the relevant past medical history and comment in detail on the medical records pertinent to the episode in question.

4.02 The relevant records start with the GP computerised record from 28 November [redacted] (02 007), which states that the patient had been feeling unwell since starting Simvastatin one month ago and "continues to feel so one week after stopping". The complaints were of feeling hot, headache (both frontal and occipital), difficulty in opening her mouth and a dry throat. The GP comments that vision was 'okay'. Temperature was 37.4 (normal), and there was no temporal artery tenderness, no neck stiffness and no photophobia (sensitivity to light). Bloods tests were ordered for the morning, amongst which were a C-Reactive Protein (CRP) and an Erythrocyte Sedimentation Rate (ESR).

4.03 The next entry on 29 December (02 007) reports the results of these bloods tests, the notable findings being an ESR of 35 mms in one hour (mm/hr), and a CRP of 19 milligrams per litre (mg/l). The next comment is that of an emergency hospital admission with persisting headache and malaise and the word '*admit'*.

4.04 The next relevant entry is from Medical Admissions at the [redacted]NHS Trust (05 142). The history given is that of a left sided headache, malaise, and difficulty opening the mouth. The doctor comments that there was no weakness/numbness, and there is the comment *'? Side effect simvastatin taken for 3 weeks, stop for one week'.* There is a medical clerking from a foundation doctor (Dr [redacted] dated 29 December [redacted] the time being seen – 2235 (05 143). The doctor comments that this was a 76 year old female patient presenting with headache, left sided, radiating to the left lower jaw and back of the head. The history of this complaint was a 2 week history of gradual onset of headache, with no nausea, no vomiting and no history of trauma or falls. The headache was noted to be *'burning'* in character and sometimes settled with pain killers. There is further comment of no associated weakness and no focal neurological symptoms. The headache had occurred gradually over the last few weeks, and the patient was noted to have started Simvastatin for raised cholesterol and there is the statement *'? Symptoms secondary to Simvastatin'*. There is a note of the past medical history of hypothyroidism, rheumatic fever, asthma, haemorrhoids, rheumatoid arthritis and osteoporosis.

4.05 On examination there was a note of decreased opening of the left jaw due to pain. Neurological examination appeared to be within normal limits. Other relevant findings were noted to be the effects of rheumatoid arthritis on the hands.

4.06 The summary documented was that of a 76 year old female admitted with left sided facial and occipital pain. The impression was ? Giant Cell Arteritis ? non-specific headache. The plan was to discuss with the Registrar and order blood tests, namely full blood count, urea and electrolytes, CRP, ESR, liver function tests and thyroid function tests. An ECG was also performed and the patient was treated with Prednisolone 40 mgs (a corticosteroid) as a stat (immediate) dose. The results of the blood tests were normal apart from a significantly raised CRP at 19 mgs per litre, and of note (but not in itself necessarily abnormal) was an ESR of 35 mm/hr – see discussion below.

4.07 The next clinical record is review by Dr [redacted] dated 29 December at 2335 (05 150), and the history was noted and the following additional comments made:

1. Started Simvastatin 4 weeks ago.
2. Frontal headache increasing in severity, gradual onset.
3. Stopped Simvastatin one week ago.
4. Headache persists. Now also has pain left occipital area. Lasted 3 days.
5. Increasing jaw pain/discomfort opening mouth.
6. No visual discomfort.
7. No neck stiffness/pain.
8. No history of headache.
9. No visual discomfort (statement repeated).

4.08 There is a note of the result of two blood tests, namely the ESR of 35 mm/hr and a Creatinine Kinase (CK) of 90 international units per litre (normal). No mention is made of the raised CRP, but in the medical admission investigation proforma (05 149) there is an asterisk and upward pointing arrow next to the CRP result indicating that it is abnormal.

4.09 A record of the examination includes the following comments:

1. No temporal tenderness.
2. Mild discomfort in left jaw.

3. Normal eye movements, visual fields, and discs (the appearance of the optic nerve at the back of the eye).
4. No joint pain/weakness

4.10 The concluding statement is "*unlikely GCA'* . The next statement reads, *'On off Prednisolone to see if any benefit'.*

4.11 The next relevant clinical statement is from the consultant post take ward round, by Dr [redacted], dated 30th December [redacted] at 7.30am (05 151). The notes, written by Dr [redacted] include the following statements:

1. Four week history of headache.
2. Frontal initially shortly after starting 'statin' (abbreviation) then occipital discomfort.
3. Due to symptoms stopped 'statin'.
4. Headache continued mainly occipital then has jaw discomfort, no pain on eating.
5. However pain not increasing in severity.
6. No visual discomfort.

4.12 There is a brief record of examination of no temporal tenderness and the concluding statement is *'v. unlikely GCA', 'plan – can go home'.* There is no mention of whether the administration of the single dose of steroid had modified the patient's headache or resulted in any improvement in the feelings of general malaise (although according to the patients chronology she was woken once in the night to see if she felt better).

4.13 The next relevant clinical record is an Optician referral to the hospital eye service dated 16 May [redacted] (05 152). The Optician records the history of same day loss of vision in the left eye and that the patient had noticed *'engorged temporal vessels right* (spelling unclear) *for one week'.*

4.14 There is a record from what I presume to be the hospital eye service dated 16th May 2007 (05 153) which details the following history:

1. Presenting complaint of loss of vision left eye.
2. It was noticed first thing that morning.
3. The patient was not sure when it had happened.
4. Could have been two weeks ago.

5. Non specific headaches *for a few months*
6. Has had *'hot head'* for number of months.
7. No temporal tenderness.

4.15 There is a record of the past medical history which stated as including asthma, osteoporosis, treated raised cholesterol (there is a comment that the patient was started on 'statins and these were then stopped due to *'hot heads').*

4.16 The examination details significantly reduced visual acuity of 6/60 in the left eye and normal visual acuity of 6/6 in the right. Examination of the eye was actually quite unremarkable, and the concluding remark states ?AION (Arteritic Ischaemic Optic Neuropathy damage to the optic nerve due to interruption of blood supply caused by blood vessel inflammation) and the plan to review the patient the following day.

4.17 The next clinical record from the eye casualty is dated 17th May [redacted] (05 154), visual acuities recorded as being 6/6 –1 corrected in the right eye and hand movements only in the left. There is a question mark over a left relative afferent pupillary defect (RAPD – an indicator of a damaged optic nerve). There is a clinical record timed 1545 which documents a positive left RAPD and the fact that the patient had had a headache and prominent, tender, right-sided temporal blood vessels. The blood test carried out at that time showed an ESR of 71mm/hr with a C-reactive protein of 73 mg/l. The signature of this doctor was unclear, but the case was discussed with a Dr/Mr [redacted] who noted that the patient was referred to the medics (medical doctors) and that there was a slightly grey area on the back of the left fundus (the back of the eye) consistent with either a left central retinal artery occlusion, or the end result of an AION secondary to GCA.

4.18 The plan was to *'treat like GCA'*, and that the patient would need Carotid Doppler studies (an ultrasound test to exclude significant disease of the carotid arteries in the neck) and visual fields in the right eye. The patient was started on Prednisolone 70 mgs once daily, the case was discussed with Mr [redacted] the Ophthalmic Consultant, who suggested an urgent TAB to be carried out the following day.

4.19 The next clinical record is dated 17 May [redacted] at 2045 (05 157) when the Specialist Registrar in General Medicine took a history and examined the patient. The history was as given by the Ophthalmic staff, the relevant examination findings were a cord like temporal artery on the right hand side and no palpable artery on the left, and the plan was to admit the

patient, treat as planned with high dose steroids and to go ahead with the planned biopsy under the care of Mr [redacted]

4.20 A note which I presume to be from Mr [redacted] (05 158) dated the 18th May documents a six month history of complaints of a hot head, headaches and neck pain and the fact that the patient was investigated for temporal arteritis late in [redacted] and was noted to have an ESR of 35 mm/hr and a CRP of 19 mg/l. These results were the blood test taken on 29 December [redacted]. There is a comment that the patient was started on steroids and these did not ease the pain so they were not continued. The patient was noted to have felt unwell since then with malaise, lethargy, and a week's history of night sweats and a three day history of loss of vision in the left eye. She was noted to have a ten day history of tenderness in the right temple when specifically, her hair curlers felt like sharp needles.

4.21 There is an operation note dated 18 May [redacted] Mr [redacted] is the operating surgeon which details a TAB (05 170).

4.22 There is a record on 4 June [redacted] (05 181) which I presume is from the ophthalmic clinic where there is documented poor visual acuity in the left eye, a well healed temporal artery biopsy scar, and the fact that the patient was feeling a lot better with decreased pain, increased mobility and no headaches.

4.23 Examination on 19 May [redacted](05 174 - notes slightly out of order) details the following day review after TAB. According to the clinical record the TAB was positive and there follows routine follow up including serial blood tests, which include ESR and CRP. The patient was discharged on 21 May [redacted]

4.24 The patient was subsequently referred to Mr [redacted], a General Physician, who monitored the gradual reduction in steroids (05 185-6 and 05 192-194). The patient's vision according to the notes remained reduced at counting fingers only.

4.25 The last relevant clinical entry of 16 December [redacted] (05 194) from the General Medical outpatient clinic reports an ESR of 3 mms in one hour, with a reduction in Prednisolone to 1 mgs daily, and the fact that the patient was feeling better with more energy.

4.26 It is relevant to comment that the patient's visual acuity in the right eye has remained entirely normal throughout this episode and that a visual field analysis on the right carried out

on the 18th February [redacted] was within normal limits. The patient's vision in the right eye, according to the medical records, is therefore unaffected.

5. TECHNICAL BACKGROUND INFORMATION

5.01 Giant Cell Arteritis, interchangeably termed Temporal Arteritis, is an inflammatory disease affecting blood vessels (a vasculitis) which normally affects the medium to large arteries in the head and neck.

5.02 The importance of Giant Cell Arteritis lies in its potentially devastating affect on vision. It can present with sudden blindness, and this is due to inflammation involving the blood vessels that supply the optic nerve.

5.03 The systemic manifestations of Giant Cell Arteritis can include the following:

- Headache
- Proximal myalgia (aching muscles near the main joints)
- Neck pain
- Scalp tenderness
- Jaw claudication (pain on chewing – see below)
- Fever
- Abnormal temporal arteries (tender, nodular and/or non-pulsating)
- Transient ischaemic attacks / stroke
- General malaise
- Fatigue
- Anorexia (loss of appetite)
- Weight loss
- Depression
- Night sweats

5.04 The most common symptom of Giant Cell Arteritis (occurring in up to 90% of patients) is headache of new onset, which classically involves the temporal area, but sometimes the occipital area (back of the head). Patients may complain of pain on brushing the hair, or of tenderness when touching the scalp. Patients may present with a fever, and pain on chewing (jaw claudication), which is one of the classical symptoms of Giant Cell Arteritis. The word

claudication originally comes from the Latin *'to limp'*, and intermittent claudication refers to the symptom of a patient intermittently limping due to disease of the blood vessels in the lower limb causing temporary reduction in blood flow to the muscles and subsequent pain. In this situation the term *jaw claudication* refers to pain that is induced by chewing. As the Masseter, Temporalis and Pterygoid muscles (the muscles that move the jaw) demand an increased blood supply during active chewing, the patient with GCA who has inflammation of the arteries will have an inadequate blood supply and suffer ischaemic muscle pain (pain caused by the muscle being starved of oxygen).

5.05 Polymalgia Rheumatica (PMR) is a relatively benign inflammatory condition of unknown cause, which leads to morning stiffness and pain in the muscles of the neck, shoulder and pelvic areas, without loss of vision. It is considered by some to be part of the same spectrum of disorder as Giant Cell Arteritis.

5.06 The most common and serious ophthalmic presentation of GCA is permanent visual loss in one or both eyes, which occurs in 20% of patients, varying from partial to complete. Patients may sometimes present with fleeting visual loss which is generally one sided.

5.07 The diagnosis of Giant Cell Arteritis can be difficult. Patients can present with some or all of the above symptoms, and occasionally with no symptoms at all apart from raised markers in the blood.

5.08 The history is very important, and some criteria for the diagnosis of GCA were suggested by the American College of Rheumatologists in 1990. These criteria are:

- Age of onset of 50 years or older
- Onset of a new headache
- Temporal artery tenderness or reduced pulsation
- Raised ESR (greater or equal than 50 mms per hour by the Westergren method)
- Histological evidence of necrotising arteritis (inflammation of an artery with tissue death) in the temporal artery with typical features on microscopic examination

5.09 It is held that if three or more of these criteria are present then that would be highly sensitive and specific in diagnosing Giant Cell Arteritis.

5.10 Some workers have pointed out shortcomings in these criteria, due to the fact that they were suggested by doctors essentially dealing with the Rheumatological complaints that do not usually have serious consequences, as opposed to Ophthalmologists who have to deal with the complication of blindness in patients with GCA. A large literature review carried out in 2005 pointed out the following shortcomings in the criteria (Rahman and Rahman, 2005)

1. Patients with Giant Cell Arteritis may have no systemic symptoms apart from loss of vision.

2. Headache of recent onset and scalp tenderness are not always reliable signs to diagnose GCA because they can be due to other diseases.

3. Important criteria like jaw claudication, neck pain, and a raised CRP are not included, and these are very significant in the diagnosis of GCA. A normal or low ESR does not rule out GCA, as this has been found in 5-30% of patients with GCA.

4. An abnormal temporal artery on physical examination may be present in some patients with a positive TAB and likewise with a negative TAB.

5.11 Treatment of Giant Cell Arteritis is with systemic corticosteroids. The aim of treatment is to suppress the inflammation in the blood vessels and to prevent further complications. Patients with suspected GCA are usually started on oral Prednisolone (a corticosteroid) 1 mg per kilogram per day. Higher doses are suggested for patients with ocular complaints.

5.12 By instituting prompt corticosteroid treatment, the incidence of blindness in GCA can be lowered to 7-25% (Aiello et al., 1993). Involvement of the other eye occurs within 1-10 days in 20-62% of cases where the presentation with an affected eye was not treated. Patients usually retain their presenting vision when appropriate treatment is promptly initiated if they manage to have no further visual problems within the first week of corticosteroid therapy (Hayreh et al., 1998). Unfortunately some patients will deteriorate despite treatment.

5.13 Due its potential effect on vision, GCA is considered to be the “prime ophthalmic medical emergency”. It is mandatory for Clinicians who encounter patients who present with new onset headache associated with raised inflammatory markers to have a high index of suspicion of GCA due to the treatable nature of the condition, the varied ways in which it may present and the potential devastating consequences for vision if the diagnosis is missed or delayed.

Inflammatory markers – CRP and ESR

5.14 The Erythrocyte Sedimentation Rate (ESR) is the rate at which red blood cells precipitate in one hour. A widely used rule for calculating normal maximum ESR values in adults (98% confidence limit) is given by a formula devised in 1983 (Miller et al., 1983)

$$\text{ESR } (mm/hr) \leq \frac{\text{Age } (in\ years) + 10\ (if\ female)}{2}$$

5.15 Another ESR reference range from a large 1996 study (with weaker confidence limits) are given by this table (Wetteland et al., 1996)

5.16 The ESR is raised in any condition where fibrinogen (a protein in the blood essential for blood clotting) levels are increased, such as inflammatory disease, infection and malignancy. It can also be elevated due to other factors such as old age, being female, pregnancy, anaemia and red blood cell abnormalities. Because it is a non-specific finding, a raised ESR is not by itself diagnostic, unless grossly elevated. It should be viewed in conjunction with other more specific tests such as CRP or Plasma Viscosity.

Simvastatin

5.17 Simvastatin is a drug used to lower cholesterol in the blood . It has many reported side-effects, amongst the commonest are upper respiratory infection, headache, abdominal pain, constipation and nausea. A myopathy (muscle problem) causing muscle aching can be detected by a raised Creatinine Kinase (an enzyme).

(ESR 95% limits)	Age (years)		
	20	55	90
Men	10	14	19
Women	15	21	23

6. RELEVANT PAST MEDICAL HISTORY

6.01 This patient's most significant past medical history is that of Rheumatoid Arthritis (RA) as active RA can be a cause of raised inflammatory markers in the blood.

7. OPINION

7.01 It is worth stating at the outset that the diagnosis of GCA can be difficult. I think however that based on the records available to me it is more likely than not that Mrs [redacted] had GCA when she presented on 29th Decembe [redacted] to the [redacted] NHS Trust Medical Admission Unit. In my opinion there should have been enough suspicion regarding the diagnosis, although it was not absolutely typical, to at the very least request an ophthalmic opinion about the advisability of performing a TAB, even though vision was unaffected at that point. I have reached these conclusions for the following reasons:

a. Giant Cell Arteritis is a treatable cause of blindness, and it is because of the potentially devastating consequences of a missed diagnosis that it is generally held that if there is any suspicion at all of GCA that a request for a TAB is made. Traditionally, but not exclusively, these biopsies are performed by Ophthalmologists. This means that Ophthalmologists see a lot of patients with GCA and are generally skilled at assessing the patient with suspected GCA and organising a TAB if indicated. The history given to Mr [redacted] and stated in the patients own version of events is that her symptoms had been largely unchanged and similar for the past six months. This leads me to believe that the headache and raised inflammatory markers in December [redacted] were due to GCA.

b. The patient is of the appropriate age group to develop giant cell arteritis.

c. The patient presented with new onset headache, one of the hallmarks of GCA. On admission there was a question mark as to whether this headache was a side-effect of medication (Simvastatin) but it was noted in the records that the headache had continued despite cessation of the therapy. It could be argued that headaches are common, as indeed they are, and the headache was atypical of GCA. However, it is the *combination* of new-onset headache *and* raised inflammatory markers that should have prompted referral.

d. The patient complained of pain in the jaw. Typically, giant cell arteritis can present with jaw claudication (see technical explanations above). Although it is noted that the pain did not get

worse on chewing, it can be hard to distinguish between different types of jaw pain, and this symptom together with the raised inflammatory markers and new-onset headache should have increased the level of suspicion of GCA.

e. The patient presented with raised inflammatory markers on blood testing.

7.02. From the technical detail given above, is information, we can see that the range for ESR for a 76 year old female could range from approximately 22 to 43. An ESR of 35mm/hr in our patient, taken in isolation, could therefore be considered to be normal if the first method is used for assessment, and abnormal if the second is considered. I would therefore class this result as “equivocal”. An ESR of less than 40mm/hr has been reported in 8-22.5% of patients with GCA and the disease process affecting the eye can be severe even when the ESR and CRP are only mildly raised (Poole et al., 2003).

7.03. Critically, the CRP in December [redacted] was significantly raised at 19mg/l (normal reference ranges for this test are less than 5-6 mg/l). It is recognised that CRP is a more sensitive indicator than the ESR for detection of GCA (in one series it was 100% sensitive - (Hayreh et al., 1997) and furthermore, ESR combined with CRP gives the best specificity (97%) for detection of GCA (Hayreh et al., 1997).

7.04. *For these reasons, a raised CRP, even when the ESR could be taken to be equivocal, considered together with <u>any</u> history of new onset headache and jaw pain, no matter how atypical, in my opinion should have prompted further referral and/or investigation.*

7.05. There are other causes of raised inflammatory markers, and I note that this lady has had a raised CRP in the past. In 1994 she had an acute illness, thought to be a pulmonary embolus, and was admitted to Hospital (05 009). During that admission, the CRP rose to 142 mg/l. It is recognised that CRP, as one of the so-called acute phase proteins, will rise when there is concurrent illness and inflammation in the body, of which GCA is only one cause. It also falls quickly once the acute event is over; this patients CRP fell to 5 mg/l in October 1995 (17 036). The rise in CRP could also be due to a flare-up of this ladies Rheumatoid Arthritis, although there were no symptoms in December suggestive of this. This lady also carried a diagnosis of Sarcoidosis, another inflammatory disease, and a raised CRP could be due to this (Rothkrantz-Kos et al., 2003). It could be argued that no reliance should be placed on the presence of raised markers in this case due to these factors, but inflammatory markers are

non-specific, and it is for that reason that TAB's are performed in patients with new onset headache and raised inflammatory markers, even if there may be another explanation for the abnormal blood tests. Indeed, TAB's are sometimes performed when there are raised markers even when the patients have no other symptoms whatsoever. GCA can also rarely occur with normal inflammatory markers (Poole et al., 2003).

7.06. The patient was treated with a relatively small dose of steroids (40 mgs). This was given as a stat dose (once only) late in the evening on the 29th December, and the patient was discharged the following morning on no treatment and no follow up. Such a "trial of steroids" is not the correct management when GCA is suspected. If there is any doubt about the diagnosis (and clearly the diagnosis was considered in order for the examining doctor to prescribe steroids) then the correct management is to immediately administer a therapeutic dose of steroids at 1mg per kg bodyweight, or if there is visual loss to consider "pulsed" (intravenous) high dose steroids.

7.06. This should be immediately be followed by a referral for a TAB. In any case, the dose of steroids was too small and the patient was not followed up to see if any benefit had been obtained or whether the symptoms were improving. If patients suspected to have GCA are treated with steroids and improve, and do not have a TAB, then the diagnosis of GCA has not been unequivocally been made. If then subsequently the patient reduces the steroids too quickly, or they are stopped in future by another practitioner, then the inflammation can recur with visual loss.

Comment on complaint correspondence

7.07. There is a letter dated 5 June [redacted] from [redacted] e Chief Executive of the [redacted]

7.08. In the fourth paragraph of his letter, Mr [redacted] States the following. *"Importantly, although you had mildly raised inflammatory markers (markers for infection), your ESR was only 35, whereas typically it is over 50 and often much higher in a patient with GCA".*

7.09. My comments here are that there is no mention of the raised CRP, which to my mind is as important, if not more important than the "mildly raised" ESR. The statement is correct in that typically the ESR is over 50 mms per hour and often much higher, but also as we have

discussed above the ESR in GCA can be borderline or low, and the CRP measured at the same time cannot be ignored. Both results must be considered together.

7.10. In the fifth paragraph Mr [redacted] omments that on discharge patients are advised to contact either the medical team or the GP should their symptoms not settle. In my opinion the onus should not be on the patient to report any symptom of GCA, as GCA can present with suddenly with visual loss, which is irreparable. With more minor conditions that do not have serious unforeseen sequelae, then it is perfectly acceptable for the patient to report deterioration and then to be seen. GCA does not fall into this category.

8. RECOMMENDATIONS

None

9. REFERENCES

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10. DECLARATION

I understand that my overriding duty is to the court, both in preparing reports and in giving oral evidence. I have complied and will continue to comply with that duty.

I have set out in my report what I understand from those instructing me to be the questions in respect of which my opinions as an expert are required.

I have done my best, in preparing this report, to be accurate and complete. I have mentioned all matters that I regard as relevant to the opinions I have expressed. All of the matters on which I have expressed an opinion lie within my field of expertise.

I have drawn to the attention of the court all matters, of which I am aware, which might adversely affect my opinion.

Wherever I have no personal knowledge, I have indicated the source of factual information.

I have not included anything in this report that has been suggested to me by anyone, including the lawyers instructing me, without forming my own independent view of the matter.

Where, in my view, there is a range of reasonable opinion, I have indicated the extent of that range in the report.

At the time of signing the report I consider it to be complete and accurate. I will notify those instructing me if, for any reason, I subsequently consider that the report requires any correction or qualification.

I understand that this report will be the evidence that I will give under oath, subject to any correction or qualification I may make before swearing to its veracity.

I have attached to this report a statement setting out the substance of all facts and instructions given to me which are material to the opinions expressed in this report or upon which those opinions are based.

This report has been prepared in accordance with the Code of Guidance on Expert Evidence.

11. STATEMENT OF TRUTH

I confirm that insofar as the facts stated in my report are within my own knowledge I have made clear which they are and I believe them to be true, and the opinions I have expressed represent my true and complete professional opinion.

Signature.  Date:

This Report is provided to those instructing me with the sole purpose of assisting those who are party to the dispute on which the Report comments. It may not be used for any other purpose, nor may it be disclosed to any third party without my express written authority

12. APPENDICES

PDF's of all quoted references below

13. ABBREVIATED CV

Abbreviated CV of Mr Jonathan Luck

I am a Consultant Ophthalmic Surgeon at the Royal United Hospital, Bath, and was appointed in 1995. I was Clinical Director of the Department for five years, and am currently Royal College Tutor overseeing education and training within the department.

I am a Bachelor of Medicine and Bachelor of Surgery at the University of Manchester, a Fellow of the Royal College of Surgeons of England and a Fellow of the Royal College of Ophthalmologists.

My special interests are in Cataract and Refractive Surgery, including Laser Refractive Surgery.

Memberships and Affiliations
Fellow of the Royal College of Ophthalmologists
Fellow of the Royal College of Surgeons of England
Member of the European Society of Cataract and Refractive Surgeons
Member of the United Kingdom Society of Cataract and Refractive Surgeons (Council member)
International Member of the The American Academy of Ophthalmology
Member of the The British Society of Refractive Surgeons (Secretary)
Examiner for the Royal College of Ophthalmologists
Member of the Microsurgical skills faculty and Committee member, The Royal College of Ophthalmologists
Adviser to the Healthcare Commission
Adviser to the Healthcare Ombudsman