Denis Matthew Harte v Dr Tan Hun Hoe and Another
[2000] SGHC 248

Case Number : Suit 1691/1999

Decision Date : 24 November 2000

Tribunal/Court : High Court

Coram : Chan Seng Onn JC

Counsel Name(s) : Raj Singam, Edmund Kronenburg, Adrian Tan and Wendell Wong (Drew & Napier) for the plaintiff; Myint Soe and Daniel Xu (Myint Soe Mohamed Yang & Selvaraj) for the 1st defendant; Lek Siang Pheng, Vivienne Lim and Jamie Yip (Helen Yeo & Partners) for the 2nd defendants

Parties : Denis Matthew Harte — Dr Tan Hun Hoe; Gleneagles Hospital Ltd

JUDGMENT:

Cur Adv Vult

The facts

1. The plaintiff, Mr Denis Matthew Harte ('Mr Harte'), is from New York. He came to Singapore in August 1996 to work as a trading manager with E D & F Man Asia Pte Ltd. It was a promotion for him. His wife, Mrs Michelle Lynn Harte ('Mrs Harte'), later joined him in Singapore.

2. Mr Harte is 36 years of age and his wife is 38. They were married in November 1989. When they were in New York, they started trying for a child in the spring of 1994. Having met with no success after 2 years, they sought medical advice. In April 1996, Mrs Harte saw a gynaecologist, Dr James Jones. On Dr Jones' advice, Mr Harte consulted Dr Lawrence Dubin, a New York urologist. Tests conducted established that:

> (a) Mr Harte produced a normal quantity of sperm and ejaculate. Seminal test on 22 April 1996 revealed that the total sperm count was 98 million as opposed to 60 to 80 million for a normal sperm count.
>
> (b) His testicles were of normal size, the right measuring 25 cc and the left measuring 15 cc.
>
> (c) He had normal testosterone levels.
>
> (d) The quality of his sperm was in a subfertile range. Some 40% of his sperm were motile with poor forward progression, when the normal level should be about 60% with good forward progression. Only 11 % reached full maturity when the normal level should be over 60%.
>
> (e) Clinical examination showed that he had a large left varicocele with reflux, palpable with Mr Harte standing in an upright position while performing a valsalva manoeuvre (i.e. forceful attempt at exhalation with nostrils and mouth closed).

3. Dr Dubin then advised Mr Harte to undergo a surgical procedure known as a left varicocelectomy. According to Dr R.G. Notley, a consultant urological surgeon in the United Kingdom testifying on behalf of the plaintiff, the objective of a varicocele operation is to reduce the pool of warm blood in the varicose veins surrounding the testes, which increases the temperature in the spermatogonia and decreases the efficiency with which they turn into spermatids. By ligating the appropriate number of varicose veins in the scrotum, the pool of warm blood is removed due to the mechanism of thrombosis. The temperature in the

testicular area is correspondingly lowered and fertility is enhanced.

4. On 15 May 1996, Dr Dubin wrote to Dr Jones and copied his letter to the plaintiff. After 3 semen samples from Mr Harte were analysed, Dr Dubin concluded that they were highly sub-fertile specimens due to poor sperm morphology. He suggested a left varicocelectomy to improve the semen quality. All expectations, ramifications and complications were fully explained to the couple. Some concepts of intrauterine insemination together with stimulation of ovulation were discussed. But they were told that the high degree of immaturity in the sperm would perhaps negate this approach. In vitro fertilization was also mentioned briefly to them. The plaintiff did not dispute these discussions as stated by Dr Dubin in his letter.

5. Dr Dubin eventually performed the left varicocelectomy surgery in June 1996. It involved ligation and excision of two markedly dilated internal spermatic veins at the groin area, where a left inguinal incision was made and carried down to the area of the external ring. Subsequently, Dr Dubin checked under 6 power loupes magnification to ensure that the artery and vas were intact. After the surgery, Mr Harte had minor pain and swelling, and some discomfort for about two days. At worst, his scrotum increased about 50% in size. By the third day, the pain and swelling subsided. The pathologist found that the tubular dilated spermatic vein removed by Dr Dubin measured 0.1 cm in diameter.

6. From August 1996 (when the couple arrived in Singapore) to early 1997, they tried again to start a family but there was no success despite that left varicocele operation by Dr Dubin.

7 April 1997 – 1st Consultation

7. On 7 April 1997, Mr Harte decided to consult the 1st defendant, Dr Tan Hun Hoe ('Dr Tan'), who is a specialist urologist and a renal transplant surgeon in Singapore. Dr Tan's private clinic is known as H H Tan Urology Centre Pte Ltd., which was then located at unit #04-03 on the 4th storey of the Gleneagles Medical Centre. Gleneagles Medical Centre is located next to Gleneagles Hospital ('hospital'), which is owned and managed by the 2nd defendants. Dr Tan is an accredited surgeon with the hospital and has an arrangement, whereby he may perform his surgical operations on his private patients using the hospital's facilities. At all material times, Mr Harte was a private patient of Dr Tan.

8. At the first consultation in Dr Tan's clinic, Mr Harte informed Dr Tan of his left varicocelectomy by Dr Dubin. He told Dr Tan that no seminal analysis was done since to determine how effective that surgery was. Dr Tan therefore requested for a seminal analysis. On clinical examination, Dr Tan found that:

(a) there was no abnormality in his genitalia;

(b) his testes were of normal size and consistency;

(c) there was **no varicocele** or hydrocele.

9. The seminal test results dated 8 April 1997 from Sheng Yu Laboratory revealed that Mr Harte had a sperm count of 29.5 million per ml (the normal level being about 20 million per ml) and a good volume of ejaculate of 4 mls after abstinence of 5 days (the normal level being between 1 to 6 mls). However, the sperm motility was 35 % (the normal level being equal or above 40%). Normal forms were only 3 % (the normal level being equal or greater than 30%). The laboratory classified his sperm quality as "Asthenozoospermia (reduction in the vitality of spermatozoa) with severe teratozoospermia (the presence of deformed spermatozoa)." The laboratory's comment was "Adequate volume of semen showed low % sperm motility and low % normal forms with high % tail defects."

10. Instead of improving, the normal forms had in fact dropped from 15% to some 3% after Dr Dubin's operation. Based on this seminal analysis, Dr Dubin agreed that there was no improvement after the operation. In his opinion, about 15% of cases would not show improvement after a varicocelectomy.

14 April 1997 - Dr Tan's letter to Dr Wun

11. On 14 April 1997, Dr Tan wrote to Dr Winnie Wun, the gynaecologist whom Mrs Harte was consulting for her own fertility problems. Dr Wun had recommended Dr Tan to them. As this letter indicates the then state of mind of Dr Tan and reveals his contemporaneous medical conclusions, it featured in my assessment of whether Dr Tan was negligent in recommending a bilateral varicocelectomy operation for Mr Harte. I therefore set it out in full.

> Dear Dr. Wun,
>
> **Re: Denis M. Harte M/32**
>
> Thank you for referring Denis, husband of your patient Michelle.
>
> He gave a history of left variocelectomy in New York in June '96 and a thermal scan subsequently showed improvement.
>
> Clinically no abnormality was detectable in his genitalia. Both testicles were of normal size and consistency. There was no evidence of varicocele.
>
> A 3 D [Three Dimensional] TransRectal Ultrasound Scan revealed a normal prostate and ejaculatory ducts. Both seminal vesicles were distended but he had abstained for 5 days.
>
> A S.A. [Seminal Analysis] revealed Asthensozoospermia with severe Teratozoospermia. Volume and density was adequate.
>
> A Colour Doppler Ultrasound Scan **could be useful** in view of the above findings.
>
> Will keep you informed.
>
> Warmest regards,
>
> (Signed Dr Tan Hun Hoe)

12. It appears that Dr Tan was hoping to find the cause or source of the infertility using the Colour Doppler Ultrasound Scan ('CDUS') after he failed to detect any varicocele clinically. Having checked that the prostate and ejaculatory ducts were normal, what then was the cause of his poor sperm quality? I do not find that Dr Tan's further investigations using the CDUS can be regarded as exceptional, wrong or negligent. I do not expect a doctor to stop his inquiries simply because the clinical investigation has failed to locate the source of the problem. I expect him to continue with other tests, aids and investigating techniques in a systematic manner, whether with or without special instruments, to find the cause and resolve the patient's medical problem if he can.

13. I thus reject the opinion of the plaintiff's experts that it was wrong for Dr Tan to use a CDUS to detect varicoceles simply because no varicocele was detected clinically or that the CDUS was too sensitive an equipment for detection of varicoceles. The following medical literature supports the use of ultrasound equipment to detect clinical and subclinical varicoceles:

> (a) Evaluation and Management of the Infertile Male: What's New and What's Important (Interactive) by Goldstein, Pryor and Schlegel.

(b) Clinical versus Subclinical Varicocele: Improvement in Fertility after Varicocelectomy by Dhabuwala, Hamid and Moghissi.

(c) Correlation of Testicular Color Doppler Ultrasonography, Physical Examination and Venography in the Detection of Left Varicoceles in Men with Infertility by Petros, Middleton and Picus.

14. The caveat I have is that instruments must always be correctly used and the results obtained must be properly interpreted in the light of the peculiar limitations and characteristics of the instruments. Full regard must also be given to clinical observations and other investigations performed. After that, a carefully considered medical judgment is called for whether to recommend proceeding with the operation or not, taking into account the risks involved in that particular type of operation and weighing them against other alternative courses of therapy (i.e. without the operation). Depending on the condition of the patient and the amount of information sought by the patient, the doctor must use his clinical judgment to decide what and how much he should tell the patient of the various risks, the pros and cons of each viable option and then, he has to objectively present and explain them to his patient, taking account of the patient's level of understanding and interest in the matter, so that the patient will have enough information to make the final decision.

15. Mr Harte complained that had he seen the letter to Dr Wun, he would not have agreed to the operation because Dr Tan had found no evidence of varicocele. He would have sought a second opinion. To my mind, that was with the benefit of hindsight.

15 April 1997 – 2nd Consultation

16. On 15 April 1997, Mr and Mrs Harte saw Dr Tan again. They handed to him copies of his surgery reports and other medical documents pertaining to his first varicocelectomy as well as some medical literature provided by Dr Dubin.

17. I will digress a little to deal with the medical literature first. I presumed that Mr and Mrs Harte had read these articles and were not completely ignorant when they discussed their fertility problems with Dr Tan.

18. I note that the article published in November 1977, '*Varicocelectomy: 986 Cases in a twelve-year study*' by Dubin and Amelar had stated, "*We have also performed **reoperations** in 5 other patients who had previously had varicocelectomy by other physicians with recurrence: 3 of the 5 had improvement in semen quality and pregnancy. These cases are not included in this reported series.*" Therefore, variocelectomy **reoperations** are not something unknown or exceptional.

19. Another article published in May 1987, '*Right varicocelectomy in selected infertile patients who have failed to improve after previous left varicocelectomy'* also by Dubin and Amelar, reported that " *Most recently in 1986, McClure and Hricak studied 50 infertile men using scrotal sonography and found that 24 (48%) had bilateral varicoceles as **detected with ultrasound** (as opposed to an 8 % incidence of bilateral varicoceles in fertile controls.)*" This shows that using ultrasound to detect varicoceles is not inappropriate as an aid for diagnosing such a condition. In my opinion, it is a useful diagnostic tool if used correctly. The authors, Dubin and Amelar, had not commented that it was wrong to do so. However, for their own practice they said:

> The clinical diagnosis of varicocele is not always a simple matter. In our practice we rely on careful palpation. Contact scrotal thermography using the Clark Topical Thermograph (Clark Research and Development, Inc., New Orleans, LA) has been a useful supplemental diagnostic tool, but for this study we have relied entirely on our careful palpation for making the decision about whether or not a patient had a varicocele and whether it was small, moderate, or large in size.
>
> Patients must be examined in the upright position because all but very large

> varicoceles will be overlooked because of venous decompression if the patient is supine. Reflux of blood down the internal spermatic veins into the scrotum should be determined by careful palpation of both left and right spermatic cords while the standing patient performs the Valsalva maneuver. This maneuver is extremely important because small varicoceles will not be diagnosed without it, and correct bilateral therapy will not be suggested.
>
> If by this method of careful palpation we cannot detect a varicocele, we do not diagnose a varicocele in that patient. We have not subscribed to the existence of the so-called subclinical varicocele that cannot be detected by careful palpation with the Valsalva maneuver.

20. The authors were clearly concerned that small varicoceles were not missed, although they did not think that these small varicoceles would escape a careful palpation with the Valsalva maneuver. Thus they preferred the clinical approach even for small varicoceles whereas as I see it, others might well prefer using the CDUS as an aid in detection.

21. In the third article given to Dr Tan by Mr Harte, '*Varicocelectomy: Twenty-five Years of Experience',* published in 1988, it recognised that the modification of the Ivanissevich procedure is simple and the **average surgeon can achieve excellent results**. In this procedure, the inguinal incision is made over the internal inguinal ring. An incision is made in the external oblique muscle through the external inguinal ring. The spermatic cord there is then dissected free. Finally, the internal spermatic veins are ligated and partially excised at the internal inguinal ring. **In recent years, the procedure has been further modified by ligating the veins even lower at the level just below the external ring**. However, the article cautioned that the artery, the external spermatic veins, and the vasal veins should not be ligated in this procedure. Table II of the article shows very few incidents of post-operative complication, which attest to the benign nature of the surgery. None of the varicocelectomy operations involving 986 men resulted in a disastrous post-operative complication of testicular atrophy.

22. The article went on to say that surgery is indicated by poor sperm motility and immaturity, **and** the presence of varicocele. Varicocele is established by a large body of evidence as a major cause of male infertility. Where the varicocele cannot be demonstrated by palpation or **crude doppler techniques,** the temptation to ligate the internal spermatic vein in patients with poor sperm motility and morphology must be resisted. This suggests that where an ultrasound doppler scan as an alternative confirmatory test to palpation does confirm the presence of varicocele in a man with poor semen quality, it is in order to proceed with a varicocelectomy operation. Therefore, the contention that it was wrong for Dr Tan to have operated is without foundation if the CDUS that Dr Tan requested for had indeed detected a varicocele condition.

23. On 15 April 1997, Dr Koay Lok Hin, a radiologist practising at Gleneagles Hospital, performed the scrotal ultrasound scan with Mr Harte lying supine. Dr Koay's brief report stated the following:

> COLOUR DOPPLER U/S OF TESTES
>
> > Both testes show normal size and parenchymal echogenicity. No evidence of abnormal focal intratesticular lesions is seen.
> >
> > Both heads of epididymis appear normal.
> >
> > There is evidence of **intermittent** varicoceles demonstrable o n the left side on valsalva manoeuvre. **Very early minimal intermittent** varicoceles is noted on the right side on valsalva manoeuvre.
>
> COMMENTS

Intermittent varicoceles noted on the left side.

24. Mr Harte admitted being told by Dr Tan that the CDUS revealed a varicocele recurrence on the left side, and a varicocele **forming** on the right. Dr Tan further read, explained and showed him the radiological report. I cannot find any deliberate concealment or misrepresentation of the CDUS results by Dr Tan. Mr Harte's complaint was apparently about Dr Tan's failure to disclose that he did not find any varicocele **clinically**.

25. Based on the CDUS, Dr Koay explained that he found evidence of varicocele on the left side and very mild varicocele on the right side. Dr Tan's interpretation and explanation to Mr Harte of the CDUS report was thus fundamentally correct.

26. Dr Tan wrote on 16 April 1997 to Dr Wun informing her that Mr Harte's CDUS revealed '*recurrence/persistence of the left varicocele.*' He noted that only two branches of the internal spermatic veins were ligated previously and suggested a repeat varicocelectomy at a **lower level** for Mr Harte to consider.

27. This letter significantly corroborates Dr Tan's oral evidence that he operated at a different site from that operated by Dr Dubin. Dr Tan testified that he operated at a lower part of the spermatic cord. Thus the problems alluded to by the plaintiff's experts of the dangers or increased risks of re-operating on the same part of the spermatic cord do not apply.

28. According to the plaintiff, Dr Tan advised them to have a second varicocelectomy to enhance his fertility. He remarked that Dr Dubin had been too conservative. Dr Tan said that he still had a left varicocele and a varicocele was developing on the right as well. Even after Dr Dubin's surgery, the quality of Mr Harte's sperm had not improved. So he advocated a bilateral varicocelectomy because the infertile condition was due to bilateral varicoceles. I have no doubt that Mr and Mrs Harte relied on the advice of Dr Tan and consequently, consented to the bilateral varicocelectomy.

29. Dr Tan assured them that there would be no complications following the day surgery other than some minor swelling and general discomfort lasting just a few days. Mr Harte was not surprised because he had experienced these normal side effects after his previous varicocelectomy in New York. Dr Tan however did not mention any risks inherent in the surgery.

30. The plaintiff alleged that Dr Tan was negligent in omitting to advise him that he could suffer a scrotal haemotoma, atrophy of his testes, be incapable of producing sufficient testosterone or cease to produce sperm altogether. Had Dr Tan done that, he would not have gone for the operation.

31. In my judgment, the risk of testicular atrophy is entirely remote as a normal consequence of such a simple surgery. An omission to mention some fanciful and very remote risk does not constitute a negligent omission. Just as death is a possibility in every surgery however minor, I do not think that a surgeon will be regarded as negligent when he omits to mention death as a possible risk in a varicocelectomy, if in fact death unfortunately occurs. The House of Lords in *Sidaway v Board of Governors of the Bethlem Royal Hospital and Maudsley Hospital and Others* [1985] 2 WLR 480 specifically addressed the question of non-disclosure of risks and the duty to inform. By a majority of 4 to 1, House of Lords held that the *Bolam* test applied not only to the diagnosis and treatment of a patient, but also extended to the giving of medical advice. Hence, the question whether an omission to warn a patient of the inherent risks of an operation constitutes a breach of the surgeon's care towards his patient is to be determined by an application of the *Bolam* test. However, Lord Bridge added the following important caveat:

> ..even in a case where, …, no expert witness in the relevant medical field condemns the non-disclosure as being in conflict with accepted and responsible medical practice, I am of opinion that the judge might in certain circumstances come to the conclusion that disclosure of a particular risk was so obviously necessary to an informed choice on the part of the patient that no reasonably prudent medical man would fail to make it. The kind of case I have in mind would be an operation involving a **substantial risk** of grave adverse consequences, as **for example, the ten per cent risk** of a stroke from the operation which was the subject of the Canadian case of *Reibl, v. Hughes*, 114 D.L.R. (3d) 1. In such

> a case, in the absence of some cogent clinical reason why the patient should not be informed, a doctor, recognising and respecting his patient's right of decision, could hardly fail to appreciate the necessity for an appropriate warning.

32. In this case, the plaintiff called no medical expert to condemn the non-disclosure by Dr Tan of the possible risk of atrophy. This indicates perhaps that hardly any urologist would think that such a warning is called for. Probably, the majority hold the contrary view. I doubt for instance that Dr Dubin ever mentioned that risk to Mr Harte prior to his left varicocelectomy surgery. Otherwise, why would Mr Harte be ignorant if Dr Dubin had previously apprised him of it? As the risk of atrophy of the testes in a varicocelectomy is so remote and negligible, I do not regard it as being so obviously necessary that no reasonably prudent urologist would fail to disclose it. As such, Dr Tan is not negligent even if he had not told Mr Harte of the risk of testicular atrophy.

33. Is the bilateral varicocelectomy with a repeat surgery on the left side at a different level, a radical and experimental treatment by Dr Tan? Having perused the medical literature tendered, I think not.

34. A further complaint by Mr Harte is that no advice was given of any other treatment as an alternative to the bilateral varicocelectomy although he and his wife had specifically asked Dr Tan whether other options were available. Dr Tan told them that bilateral varicocelectomy was the only solution.

35. I do not believe that Dr Tan would have been so adamant about the operation being the only solution. Mr Harte exaggerated his evidence somewhat in his affidavit of evidence-in-chief. It is more likely in my view for Dr Tan to have recommended bilateral varicocelectomy operation as their **best** option, which implied that there were indeed other options although Dr Tan might not have enumerated them. In court, Mr Harte moderated his evidence somewhat and testified that Dr Tan told him that the **best** option would be bilateral varicocelectomy but no other alternatives were offered.

36. With the history of their fertility problems and the medical literature they had, surely they must have known of the existence of test-tube babies, in vitro fertilization and a myriad of other similar methods besides varicocele operations. In fact, Dr Dubin himself mentioned in vitro fertilization to the couple. I find it hard to believe their ignorance of other options even if it were true that Dr Tan had not apprised them of their existence and availability. I conclude that no negligence has arisen out of the purported failure of Dr Tan to apprise them of other options.

37. Prior to leaving Dr Tan's office on 15 April 1997, Mr and Mrs Harte asked Dr Tan whether they should store some sperm in a sperm bank before the operation in case something went wrong after the operation. Dr Tan dismissed it as he was confident that nothing would go wrong in the surgery.

38. Assuming that Dr Tan had given that advice, I do not consider that he had acted negligently because testicular atrophy is clearly not a foreseeable consequence of a simple benign operation like varicocelectomy where an average surgeon can achieve excellent results let alone an expert urologist like Dr Tan who has had many years of experience and who has performed 200 of such operations without a single case of atrophy. If it were a reasonable precaution that should normally be taken, and advice was given to the contrary, then there might be grounds to allege negligence on Dr Tan's part for having given that advice which was relied on and followed by the plaintiff.

39. As Dr Tan felt that storage of Mr Harte's semen was of no benefit since his semen was of such poor quality anyway, it was not an unreasonable basis upon which to advise against the necessity of sperm banking. If the worst possible foreseeable outcome is a nil improvement in sperm quality, and since fresh semen for any artificial insemination is generally preferred over thawed out deep frozen semen, it is a fair and rational advice as far as I can see. I also note that sperm banking prior to varicocelectomy is not a routine procedure with all the major hospitals in Singapore.

40. In any event, if Mr and Mrs Harte had wanted to, they could have gone to the sperm bank themselves to bank some of Mr Harte's sperm and pay for it separately, without any need to obtain the green light from Dr Tan. There is nothing to stop them from doing so.

41. There was also the argument raised that subclinical varicocele ought to be left alone and hence, Dr Tan should not have operated to remove the subclinical varicocele. The following papers point to the contrary that the detection of subclinical varicocele may be warranted in infertile men with abnormal sperm and that its treatment may have some beneficial effect on spermatogenesis:

(a) Subclincal Varicocele: The Effectiveness of Varicocelectomy by McClure, Khoo, Jarvi and Hricak

(b) Effect of Varicocelectomy on Sperm Parameters and Pregnancy Rate in Patients with Subclinical Varicocele: A Randomized Prospective Controlled Study by Yamamoto, Hibi, Hirata, Miyake and Ishigaki.

(c) Effectiveness of subclinical varicocelectomy: a prospective randomized study by Okuno, Shichiri, Onishi and Yoshida.

(d) Evaluation and Management of the Infertile Male: What's New and What's Important (Interactive) by Goldstein, Pryor and Schlegel.

(e) Clinical versus Subclinical Varicocele: Improvement in Fertility after Varicocelectomy by Dhabuwala, Hamid and Moghissi.

28 April 1997 - Operation

42. On 28 April 1997, Mr Harte checked into Gleneagles Hospital for his operation as instructed by Dr Tan. The hospital counter staff gave him some forms to sign: admission form, consent form and conditions of services/hospital policies form. Dr Tan performed the surgery under general anaesthesia. It commenced at 0955 hrs and ended at 1115 hrs. After the surgery, Mr Harte was wheeled to a recovery room in the day ward to rest.

Dr Tan's extremely brief operation notes stated the following:

Operation Performed:	Bilateral Varicocelectomy (Redo left)
Incision:	Bilateral groin
Findings:	(Left) varicocelectomy done before
Procedure:	Few external veins (Left) side and small internal veins.
	Veins x 7 ligated.
	(Right) varicocelectomy.
	Veins x 11 taken.
	Huge external veins as well
Closure:	Vicryl closure.

43. Against the advice of the hospital's nurse, Ms Tan Sang Eng, the plaintiff insisted on using the toilet despite the nurse having brought a urinal and offered a bedpan. Mrs Harte told the nurse that 'if he wants to go to the toilet he must'. She said that she would accompany him. Mrs Harte and the nurse helped to seat Mr Harte on the toilet bowl. Mrs Harte remained in the toilet with him.

44. Whilst seated on the toilet bowl, he fainted, fell off the toilet bowl and his head hit the floor first. This fall took place at 12.20 p.m., about an hour after the surgery. Mrs Harte who was present tried to prevent him from falling but was unsuccessful because he was too heavy. These facts are not disputed.

45. The major issue here is whether Mr Harte had in the process contused his scrotum and both testes, as he fell off the toilet

bowl whilst in an unconscious state. Were his testes and scrotum trapped by the toilet seat resulting in additional traction injury to the delicate structures of the veins and arteries within the spermatic cord?

46. Thus, I have to consider (a) whether he had slid and then toppled off the toilet bowl (i.e. a combination of sliding and toppling) and (b) whether his upper torso could possibly rotate about his knees to lift his entire scrotum clear of the toilet seat in the course of the fall. This is a matter of considerable importance when determining whether there was any contusion or traction injury (or both) resulting from Mr Harte's fall from the toilet seat in an unconscious state. It is not disputed that an unconscious man is not capable of activating his leg muscles to lift himself clear from the toilet seat to avoid injury to his scrotum and testes. I will examine these issues in much more detail later.

47. After the fall, Mrs Harte called the nurses to help lift him up and put him on the bed. The hospital's nurse immediately telephoned to notify Dr Tan's clinic of the fall. Dr Tan sent Dr Balaji Sadasivam, a neurosurgeon, to examine Mr Harte's head. Dr Balaji did not examine the surgical incisions or the testicular area.

48. Mr Harte said that he felt pain at the bump on his head but not in his testes or scrotum on that day. By about 5.00 p.m., the effects of the anaesthesia had worn off.

49. Dr Balaji saw Mr Harte again during the evening and informed him that he could be discharged later in the evening unless there were complications. Since no complications occurred, he was certified fit for discharge. After settling his bills, Mr Harte left the hospital at about 7.00 p.m. together with his wife.

50. At the time of discharge, he felt some minor swelling and discomfort at the scrotal area. He was able to walk slowly. Mr Harte assumed that they were the normal effects of the surgery based on what Dr Tan had earlier told him. At bedtime, there was some swelling at the scrotum area but it did not seem unusual to Mr Harte. Then he went to sleep.

51. According to Mr Harte, no one mentioned that Dr Tan would be seeing him. However, Dr Tan alleged that he told the theatre nurses to inform Mr Harte to wait for him. Ms Magdalene Taye, the assistant clinic nurse working for Dr Tan at that time, testified that she visited Mr Harte in the day ward and specifically informed him to wait for Dr Tan to review him. There appears to be some corroborative evidence in the form of an entry at 1350 hours in the Nursing Care Plan for Mr Harte in Gleneagles Hospital which states "*S/B Dr. Tan's clinic nurse – Dr. Tan will review pt in wd.*"

52. With the evidence I have, I think that Mr Harte was not quite truthful in saying that he was not told that Dr Tan would be seeing him. However, I do not think that it was reasonable for Dr Tan to expect the patient to wait indefinitely for him. Mr Harte had waited from 12.20 p.m. (the time of the fall) to 7.00 p.m. Yet there was no sight of Dr Tan, who was too busy with his other operations. Apparently he had lined up 7 operations for himself, one after another, and Mr Harte's was his 2nd operation for that day.

53. In any event, I do not think this is crucial because Dr Tan himself said that even if he had seen Mr Harte, he would not have done anything because there was no complaint of pain in the scrotum and the surgical sites did not bleed. In my judgment, discharging Mr Harte was not substandard post-operative patient care since there were no obvious or alarming signs at that time that his testicles were traumatised.

54. Not being given the details of the fall, Dr Tan assumed that Mr Harte fell from the standing position and hit his head. Even if he had been told that he had fallen off from a toilet bowl whilst in an unconscious state, he would not have envisaged a possible scrotal and testicular haematoma leading to atrophy. He frankly admitted that the thought never even crossed his mind. I believe him. The causation mechanism is not one which is immediately apparent without a meticulous analysis on how a fall like that can possibly contuse the testicles or cause traction injury. In my opinion, it is not likely for a reasonably competent urologist in the shoes of Dr Tan to foresee that possibility at all at that time. Hence, even if Dr Tan discharged the patient after seeing him, I would not consider him negligent. Dr Tan's inability to attend to Mr Harte before he left the hospital had not contributed to the damage caused.

29 April 1997 – Swelling

55. On the following morning when Mr Harte woke up, he found that both his testes had swollen considerably to almost 2 to 3 times their normal size. I think that Mr Harte was describing the swelling of his scrotum as a whole and not the swelling of his testes *per se* i.e. intratesticular swelling without extratesticular swelling. The swelling was much more than what he experienced after his first varicocelectomy by Dr Dubin. He felt excruciating throbbing pain around his testicular area, which was not there the night before.

56. Mr Harte testified that he telephoned Dr Tan's office at about 9.00 a.m. in a desperate attempt to find out what was going on and whether he should go back to hospital immediately. One of Dr Tan's nurses told him that Dr Tan was busy. When he managed to speak with Dr Tan, he told him of the unusual pain and swelling. Dr Tan assured him that no action was needed and the swelling would subside. Dr Tan instructed him to take the pills prescribed and everything would return to normal thereafter.

57. However Mr Harte in his complaint to the Singapore Medical Council stated that he consulted Dr Tan **two days** after the surgery, which suggested that he did not speak at all to Dr Tan on the day following the surgery. This was clearly wrong as Dr Tan was not in Singapore on 30 April 1997. Obviously, Mr Harte was mistaken. Apart from this discrepancy, the rest of his complaint was generally consistent with the events described in his evidence-in-chief.

58. Dr Tan denied receiving a phone call from Mr Harte on the day following the surgery i.e. 29 April 1997. I prefer Mr Harte's testimony as it has a ring of truth to it. The swelling would have been considerable and unusual. A swelling of the size described would naturally cause considerable pain. Under the circumstances, I cannot imagine Mr Harte sitting still with the swelling he had and not seek immediate assurances from Dr Tan personally.

59. Dr Tan never asked Mr Harte to see him at his clinic. Believing Dr Tan's assurances, Mr Harte remained at home, in great pain, hoping and expecting the pain and swelling to subside. Over the next few days, the swelling increased instead until his scrotum was 5 to 6 times its normal size. As the swelling increased, his pain increased correspondingly.

30 April 1997 – Further phone calls

60. Mr Harte called Dr Tan's clinic again on 30 April 1997 because the pain and swelling was even greater than the previous day. He did not get to speak to Dr Tan but managed to speak to a nurse. He informed the nurse of the pain and swelling and said something was wrong. She told him to take the pills and come in on 2 May 1997. In sheer desperation, he had, in one of his telephone calls, asked the nurse whether he should apply ice to his scrotum. She told him not to.

61. According to Dr Tan, he was out of Singapore on a day trip to Kuala Lumpur on 30 April 1997. 1 May 1997 was a public holiday. Another urologist would normally cover him in his absence. Had Mr Harte called the nurse or the answering service and asked for Dr Tan on 30 April 1997, the nurse would have referred Mr Harte to the covering urologist. The clinic has an answering service after office hours. Dr Tan alleged that Mr Harte had not called the clinic on 30 April 1997. However, he failed to call all the material witnesses (e.g. all his staff who handled telephone calls in his clinic) to support what he said.

62. I accept Mr Harte's evidence that he called the clinic on 29 April and managed to speak with Dr Tan, and that he made further calls to Dr Tan's clinic on 30 April because the swelling and pain had increased further.

63. In my opinion, Dr Tan or his nurses should have asked Mr Harte to come to the clinic immediately. An issue thus arises whether there was any post-operative negligence. I shall deal with this in more detail later.

2 May 1997 – 1st consultation after surgery

64. By the 2 May 1997, Mr Harte's scrotum had grown to the size of a mango, about 7 inches in length and 5 inches across. The swelling was at its maximum on the 2 or 3 May 1997 before it began to subside slowly.

65. Due to the pain and swelling, it was agonising for him to walk down the hallway to Dr Tan's clinic even with the assistance of his wife.

66. When he saw Dr Tan, he testified that he **complained of** the swelling and the **intense pain**. After a short examination, Dr Tan simply repeated his earlier advice that he had no treatment other than some medication. Dr Tan explained that he had a **scrotal haematoma** and he would prescribe something to **ease the pain** and reduce the swelling. The swelling should subside in due course. If the swelling remained, he would **drain the blood from his scrotum**. (The significance of this evidence in bold will only be apparent later.)

67. Dr Tan prescribed **painkillers**, antibiotics and anti-inflammatory medication. He did not inform Mr and Mrs Harte that the haematoma could lead to irreparable damage to or atrophy of his testes. No ultrasound scan was done on the swollen scrotum.

68. The next appointment was set for 9 May 1997. However, Mr Harte was still worried about the intense swelling and the pain. He kept calling Dr Tan's clinic every day for advice. Despite his persistent calls, Dr Tan did not see him.

9 May 1997 – 2nd consultation after surgery

69. On 9th May 1997, Mr and Mrs Harte went to see Dr Tan. The swelling had by then begun to subside somewhat although his scrotum was quite hard or firm to the touch. He examined the plaintiff's scrotum with the ultrasound machine in his office.

70. Dr Tan said that it was not necessary to drain the blood because the swelling had gone down. He prescribed more pills.

71. In his medical report dated 24 May 1997, Dr Tan stated that he had reviewed Mr Harte on 2 May 1997 and noted that he had a complication of a 'scrotal haematoma'. Mr Harte was prescribed Zinnat, Danzen, Voltaren and Antacids. When he was last reviewed on 9 May 1997, the haematoma had begun to subside.

26 May 1997 – 3rd consultation and events thereafter

72. By the next consultation on 26 May 1997, the swelling had completely subsided. Dr Tan put Mr Harte on a 3 months' course of Povinorum, which is a drug containing a form of synthetic testosterone known as mesterolone. No explanation was given to the plaintiff why this drug was necessary. This did not seem proper to me. At the trial, Dr Tan's explanation was that the mesterolone was part of the usual therapy that he normally gives to varicocelectomy patients to improve their fertility after surgery.

73. The plaintiff called Dr Schlegel as an expert medical witness. Dr Schelegel is the Director at the Center for Male Reproductive Medicine & Microsurgery at the Cornell Institute for Reproductive Medicine, The New York Presbyterian Hospital-Weill Medical College of Cornell University, New York. He is also the Associate Professor of Urology and Vice-Chairman for Clinical Affairs, Department of Urology at the same establishment. Dr Schlegel testified that synthetic testosterones like mesterolone in fact inhibit sperm production. If what Dr Schlegel said is true, then Dr Tan's explanation does not appear credible to me. I am inclined to believe that Dr Tan already knew that the plaintiff's testes were damaged to the extent that he was not producing sufficient testosterone himself and he had to prescribe the synthetic testosterone. But he could not

bring himself to inform Mr Harte.

74. On 29 August 1997, Dr Tan ordered a seminal analysis at the plaintiff's request. This analysis revealed a marked reduction in the quantity and quality of his sperm. The count was less than 1 million sperm per millilitre compared with 29 million sperm per millilitre in April 1997, and 27.68 million in April 1996. A second seminal analysis a week later on 5 September 1997 showed no more sperm production.

75. Mr Harte saw Dr Tan again on 11 and 18 September. On both these occasions, he tried to explain that his sperm could be collected in locations other than his semen and he purportedly looked for blockage although prior to the surgery, there was no evidence of retrograde ejaculation or any blockage.

76. On 8 October 1997, Dr Tan noted some 5 months after the operation that the '*haematoma occurred 2 –3 days post operation, initially very gradual*.' This was hardly a contemporaneous record of the clinical findings. It was then that he began to consider the '*?mechanism of the haematoma*', which was what he wrote in his clinical notes.

77. On 9 October 1997, Mrs Harte called Dr Tan to find out the reasons for the disastrous seminal test results. Dr Tan explained that his testes were not functioning properly. Much dried blood was in his scrotum. There was a small 3mm epididymal cyst on his left side. Blood flow in his testes was not increased. There was no evidence of varicocele except for thrombosed varicoceles on the left side.

78. On 11 October 1997, Dr Tan broke the devastating news that it was very unlikely that he would ever father his own child. Mr and Mrs Harte were shocked and devastated.

79. On 17 October 1997, Mrs Harte queried Dr Tan again. Dr Tan told her that he did not encounter any problems during the surgery. He had removed as many as 18 veins in other patients without the swelling that Mr Harte had. In Mr Harte's case, he removed about one third to half of the veins in his scrotum and those left were 'small veins' less than 2 mm in size.

Dr Tan's reply to the Singapore Medical Council

80. As Dr Tan's contemporaneous clinical notes were brief, I have included his reply to the Singapore Medical Council dated 23 October 1998 where some of his observations were included. Dr Tan stated *inter alia* that:

> On review on 2 .5. 97, he complained of a scrotal hematoma that had developed 2 -3 days after the operation. The hematoma was non-tender. He was prescribed antibiotics prophylactically and Danzen to aid resolution of hematoma.
>
> He was reviewed again a week later on 9.5.97. The hematoma had subsided but he complained that the left testicle was harder than the right.
>
> The next review was on 26.5.97. The swelling had subsided almost completely but there was residual induration of the scrotal sac which was expected to take a longer period to subside.
>
> ...
>
> In my opinion the cause of his testicular hypofunction is difficult to determine at this time as it was highly unlikely that the arterial supply could have been cut off during the operation which was carefully done with magnification. Whether the fall and the subsequent hematocele had an effect on the outcome is again difficult to ascertain. However due care was taken to manage the hematocele

conservatively as it was not tender and was expected to subside spontaneously.

Mr Harte's condition

81. Due to the insufficient levels of testosterone in his blood, he became lethargic, had no stamina, lost his muscle mass and his sexual libido, and had difficulty in achieving an erection. He started to grow breasts and had tender nipples until he received testosterone replacement therapy.

82. After getting testosterone replacement, his libido improved and he did not feel as lethargic. The tenderness in his nipples subsided. However, his sex life was not the same as before.

83. It is not disputed that Mr Harte's right testis shrank in volume to 6 cc from what was previously 25 cc in April 1996. The left testis shrank to 8 cc from what was previously 15 cc. His testes had suffered irreparable and irreversible vascular damage. Having atrophied, they cannot now produce sperm or testosterone.

84. He feels ashamed of his body, in particular his testes, when he has to use changing rooms in clubs and gymnasiums. He is now flabby even with daily exercise.

85. Worst of all, he can no longer father a child through natural means because of the azoospermia i.e. total absence of sperm in his semen. This has devastated him and his wife emotionally and psychologically.

86. The testosterone replacement therapy of skin patches by Dr Walter Futterweit was ineffective due to the humid climate in Singapore. Dr Jimmy Beng prescribed testosterone supplement tablets in May 1999, which Mr Harte took on a daily basis instead. Testosterone replacement therapy would have to be continued for the rest of his life. The plaintiff alleged that the heavy dosage exposed him to increased risk of liver and prostrate cancer. Dr Clarence Lei Chang Moh testified that the risk of prostrate cancer was not increased though he agreed that some kinds of testosterone might stimulate the growth of small tumours in the liver.

87. On 11 September 1997, Mr Harte went for another CDUS scan of his testes. Dr Koay reported the following:

> EXAMS: ULTRASOUND TESTES
>
> COLOUR DOPPLER ULTRASOUND OF TESTES
>
> Both testes are of small volume with a patchy parenchymal echo pattern. There are at least two calcific foci in the left testis. Both heads of epididymis appear normal. There is a small 3 mm epididymal cyst on the left side. Tissue perfusion is not increased. No evidence of demonstrable varicocele is shown on valsalva manoeuvre. Thrombosed varicoceles are noted on the left side.
>
> COMMENT
>
> Patchy parenchymal echo pattern noted in both testes. There are a few calcific foci in the left testis. There is a small epididymal cyst on the left side. Thrombosed varicoceles are noted on the left side. Tissue perfusion is not increased.

88. Dr Koay explained that a normal testis is fairly homogeneous. Since he saw a patchy echo pattern, it might be due to inflammation, ischaemia or infarction. The patchiness showed that the tissue was not uniform and could indicate necrosis. The

'calcific foci' in the left testis indicated the presence of coagulated blood in the testes, an old haematoma, previous infection or infarcted testicular tissue. 'Tissue perfusion' meant that there was still some blood flow through the arteries through the spermatic cord to the testes and implied that not all the arterial blood supply had been cut. From his subjective assessment of the CDUS scan, the blood flow rate was either reduced or it could be normal. Hence, he wrote in his report that the tissue perfusion was not increased.

89. In October 1998, Mr Harte consulted Dr Dubin again for bilateral testicular biopsies. The pathology report showed that there was '***partial germinal tubules sclerosis and hyalinization***' and '***focal preserved germinal tubules showing decreased spermatogenesis with only few spermatids (less than 5 per germinal tubule)***' for the left testicle biopsy, and '***complete germinal tubules and interstitial sclerosis and hyalinization'*** and '***no spermatogenesis identified in this specimen'*** from the right testicle biopsy. Essentially this suggested that Mr Harte's left testis could not produce any mature sperms but just some spermatids (i.e. immature sperms) whereas his right testis was in a worse condition because no live sperm or spermatid was found.

90. Dr Schlegel advised that he might be able to extract sperm from his left testis and attempt the ICSI (intracytoplasmic sperm injection) procedure to fertilise one of Mrs Harte's eggs. Several tries might be necessary. Dr Schlegel was hopeful of achieving a pregnancy.

91. Hence Mr and Mrs Harte had no other alternative but to return to New York for the ICSI treatment by Dr Schlegel, which Mr Harte alleged was the only way for them to receive the treatment they needed on a frequent, closely monitored basis. Treatment 'long-distance' was not possible and was not ideal. Further, leaving Singapore allowed them to reduce to some degree, the emotional trauma and haunting memories of what had happened. Driven by their desire to achieve a pregnancy, Mr Harte decided to give up his position in Singapore thereby forgoing his expatriate package, perks and bonuses, and all prospects of promotion.

Broad Issues

92. Mr Harte alleged that Dr Tan had recommended an unnecessary surgery and further, that he negligently performed the operation producing the disastrous results that such operations were not known to produce. Dr Tan however blamed the atrophy on Mr Harte's fall in the toilet of the hospital soon after the surgery, which he said had caused both contusion and traction injuries. The hospital denied that they were liable at all as Dr Tan was not an employee of the hospital but a private and independent doctor from whom Mr Harte had sought treatment.

93. The main issues as characterised by the plaintiff may thus be summarised under 3 categories:

(a) Pre-operative

(1) Whether the 1st defendant acted in accordance with the minimum standard of care and skill expected of him in:

(a) Diagnosing that the plaintiff had bilateral varicocele as at April 1997;

(b) Concluding that his bilateral varicocele was the cause of his sub-fertility in April 1997;

(c) Advising the plaintiff to undergo a bilateral varicocelectomy in April 1997;

(d) Advising the plaintiff that a bilateral varicocelectomy was the only means

available to address his subfertility in April 1997; and/or

(e) Not specifically advising the plaintiff of the particular risks and consequences of a bilateral varicocelectomy and/or one involving a repeat left varicocelectomy.

For the reasons given earlier, I find that Dr Tan had not acted below the minimum standard of care and skill expected of him as a urologist in his pre-operative treatment, care and management of Mr Harte. The issues pertaining to the surgery and post-operative care are listed below:

(b) Surgery

(2) Whether the 1st defendant acted with the minimum standard of care and skill expected of him during his surgery;

(3) Whether the surgery caused the testicular atrophy;

(4) Whether the 1st defendant was the agent or servant of the hospital at the time of the surgery;

(5) Whether the hospital was vicariously liable for the acts and omissions of the 1st defendant; and

(6) Whether the defendants committed battery and trespass to the plaintiff.

(c) Post-operative

(5) Whether the fall from the toilet caused the testicular atrophy;

(6) Whether the 1st defendant acted in accordance with the minimum standard of care and skill expected of him in:

(a) seeing the plaintiff only on the 4th day after the operation despite the persistent complaints of pain and swelling; and

(b) not taking steps to drain the blood or operate to reduce the swelling at the time of examination.

Anatomy of the male reproductive system

94. Before I deal with the evidence, it is useful to have an understanding of the male reproductive system. I find the following medical literature to be most helpful:

(a) Exhibit P 18 -- Atlas of Surgical Management of Male Infertility edited by Thomas and Nagler. Chapter 2: Normal surgical anatomy contributed by Frank Hinman.

(b) Exhibit P 20 -- Color Atlas of Urologic Surgery by Cockett and Koshiba at page 306: The Scrotum and Its Contents.

(c) Exhibit P 46 -- Extracts from Gray's Anatomy by Williams, Warwick, Dyson and Bannister

(d) Exhibit P 6 -- A colour diagram of 'The Male Reproductive System'

95. It is appropriate that I give a brief description of the structure of the scrotum. The scrotum is divided by a septum into two compartments, each containing one testis. The skin of the scrotum is thin and elastic, and richly vascularized. Beneath the skin is the dartos muscle and connective tissue that contracts with the cold and relaxes with heat. Another layer of loose connective tissue lies between the dartos layer and the deeper layers. This connective tissue is loose and permits the scrotal contents to move freely. It also allows blood and other fluid and haematomas to accumulate and spread. The blood supply of the scrotal wall comes from the external and internal pudendal arteries. The scrotal wall is also supplied by a rich network of lymphatics.

96. There are 3 complete layers that envelop and protect the testis, epididymis and the spermatic cord: external spermatic fascia, cremasteric fascia and muscle layer, and the deepest is the internal spermatic fascia.

97. The testis is encased within a tough fibrous membrane called the tunica albuginea. It is loosely suspended by the spermatic cord and its investing tunic layers. The gubernaculum attaches the testis to the base of the scrotum. The epididymis is a long narrow body sitting at the posterior border of the testis with its enlarged head near the upper pole of the testis. The tail of the epididymis is attached to the testis at its lower end. This tail connects to the vas deferens, which is a tube for the passage of sperm.

98. The testis produces sperm within the seminiferous tubules and secretes androgen from the Legdig cells. The epididymis collects the immature sperms from the testes and allows them to mature.

99. The spermatic cord is a long, round bundle that begins at the internal inguinal ring and exits at the external inguinal ring near the groin region, and finally ends at the upper pole of the testis. Within it are the vas deferens, blood vessels, lymphatics, and nerve fibres to the cord and testis.

100. There are two main arteries in the spermatic cord: internal spermatic artery (also known as the testicular artery) and the artery to the vas deferens. Both supply blood to the testis and epididymis. They are connected together at their distal ends. Therefore, damage to any one of these two arteries will not compromise the testis. Additionally, the cremasteric artery (also known as the external spermatic artery) supplies blood to the coverings of the spermatic cord. D13 is a good diagram showing the free anastomosis between the three arteries supplying blood to the testis.

101. The veins of the testis and epididymis form the pampiniform plexus, part of which surrounds the spermatic artery (internal spermatic veins) and the other surrounds the vas deferens (vasal veins). As these veins ascend up the spermatic cord, they merge and decrease in number but increase in size. Finally, they converge into the spermatic vein at the internal inguinal ring. Arteries supply blood to the scrotum and testes, and the veins drain the blood away. The testis is also drained by the crematerial veins, external veins and the gubernacular veins as illustrated by Dr Tan in exhibit D28.

102. With this brief summary out of the way, I shall revert to the case at hand.

Plaintiff's case

103. The plaintiff relied heavily on the doctrine of *res ipsa loquitur* to establish his case because the surgery was entirely under the management and control of the 1st defendant, and vicariously, the 2nd defendants. It is not disputed that bilateral testicular atrophy is not likely to occur following a bilateral varicocelectomy unless the surgeon has not performed the surgery with sufficient care or is simply incompetent. According to Dr Notley, bilateral testicular infarction is not an acceptable complication of this surgical procedure. All the 1st defendant's medical experts generally agreed that it is exceedingly rare to

have testicular atrophy and azoospermia as a result of such an operation.

104. The various possibilities put forward on what might have gone wrong during the operation to cause such a disastrous outcome were:

> a) Inadvertent injury to or ligation of a testicular artery which is known to result in testicular atrophy or impaired spermatogenesis, or both;
>
> b) Compression of the venous structures within the spermatic cord;
>
> c) Venous hypertension from the division of an excessive number of veins.

105. Initially, the main thrust of the plaintiff's case was the division of an excessive number of veins by Dr Tan. In support of this, counsel for the plaintiff referred me to the following passage from Clinical Negligence by Powers and Harris 3rd Edition at p 1389 para 41.100:

> When the complaints are infertility and the testes are lost because of ischaemic necrosis after open correction of the varicoceles, patients are particularly aggrieved and likely to resort to litigation. Testicular death may follow venous congestion leading to increased testicular pressure which, in turn, results in arterial impairment leading to ischaemic necrosis. When the presenting complaints are of mild discomfort only and testicles are lost, patients are likely to resort to litigation.

106. However, this line was not pushed very hard during closing submissions by counsel for the plaintiff after Dr Schlegel himself categorically said that one could divide all the veins leaving the vasal vein intact both for a man with or without varicoceles, and no atrophy would result because there would still be sufficient venous drainage with the vasal vein intact.

107. Counsel for the plaintiff postulated that:

> (a) Dr Tan could have damaged the vas and its vessels when he clamped his Babcock forceps around the vas and spermatic cord, which would be larger in the case of an obese man like the plaintiff;
>
> (b) He could have caused traction injury to the spermatic cord when he tried to deliver it out through the layers of muscle and subcutaneous fat, and through the skin incision so that he could perform the surgery on the veins in and around the spermatic cord. The likelihood of such injury increased when the incision through the layers of muscle and fat was oblique or diagonally made to reach the spermatic cord i.e. the 'undermining technique', which is a more difficult surgical procedure;
>
> (c) Due to the insufficient spermatic cord length available at the subinguinal region, it was doubtful that Dr Tan could have pulled out the spermatic cord through the skin incision without causing traction injury to the delicate structures;
>
> (d) Dr Tan could have inadvertently ligated or divided the testicular artery (or even other arteries as well) because of inadequate optical magnification of 3 x power optical loupes when the medical literature recommended the use of 6 x power, thereby causing loss of or reduced blood supply. Similarly, inadvertent

> over-ligation of veins due to inadequate optical magnification increased the risk of venous congestion. [Whilst on this point, I note that Goldstein, Gilbert, Dicker, Dwosh and Gnecco in their paper, *Microsurgical Inguinal Varicocelectomy with Delivery of the Testis: An Artery and Lymphatic Sparing Techniques* published in December 1992, referred to the use of **2.5** x magnification loupes for varicocelectomies. By this standard, I do not think that Dr Tan had used insufficient magnification for his surgery.]

108. It was thus submitted that Dr Tan blundered in a similar fashion on both the left and right side of the varicocelectomy operation because:

> (a) the undermining would have led him to operate on the spermatic cord fairly closely to the left and right testes owing to the insufficient spermatic cord length, which meant that his was not a subinguinal approach but a scrotal approach, which carried with it a high risk of testicular atrophy;
>
> (b) the veins at that lower level of the spermatic cord near the testes would be more numerous and finer in the form of the pampiniform plexus; and
>
> (c) the scrotal varicocelectomies involving vein ligation in that region of the pampiniform plexus were no longer carried out, as such scrotal approaches were associated with the ligation of too many veins leading to ischaemic necrosis due to venous impairment.

Nurses' evidence

109. Nurse Tan Sang Eng examined Mr Harte immediately after the fall. He did not complain of any pain in the scrotum or penis area. She took a glance at the plaintiff's scrotal area when she removed the blanket covering Mr Harte to examine the skin incision sites (which were not at the scrotum but much higher up at the groin area: see photographs P25 to P 28). She did not see any swelling, bruising or discolouration. She maintained that she was sufficiently experienced to tell if the plaintiff's scrotum or penis was bruised or discoloured.

110. Nurse Looi Chai Hong examined the plaintiff at about 2.00 p.m. She also saw no bruising, swelling or discolouration at the scrotal area, but she did notice a slight bruise, **slight bluish in colour**, on the left side of the plaintiff's head. Her examination was about 1 hours after the fall. However by 7 p.m., Mr Harte could feel the swelling and discomfort because his scrotum touched his pants.

111. Dr Notley believed that swelling should begin about 2 hours after the fall. Consequently, the nurses might not notice any swelling at his scrotum. The nurses would probably also not see any difference or notice any colouration at that stage. Dr Notley's evidence demonstrates that the absence of swelling, bruising or discolouration at this time is not conclusive.

112. Dr Notley also said that a change of colour of the scrotum within 2 hours would be expected for a severe injury causing a severe haematoma. The obvious corollary is that a blunt trauma of a less serious nature may not involve a change of colour. This is not exceptional where there is minimal or no rupture of the blood vessels in the scrotal area external to the testes.

113. It is my opinion therefore that the nurses would probably have noticed the bruising and discolouration if it was a very serious blunt trauma that not only damaged the testes internally, but also severely ruptured the blood vessels in the extratesticular area of the scrotum. Where the blunt trauma was not as severe, the testes might still suffer contusion but there might not be a violent rupture (as opposed to a minimal rupture or rupture at the microlevel) of the extratesticular blood supply

to lead to an obvious scrotal haematoma. Nonetheless, the intratesticular contusion itself might still be serious enough to lead to atrophy. Thus the absence of discolouration and scrotal swelling observed by the nurses was inconclusive. In other words, one could not rule out internal testicular traction or contusion injury (or both) as a possible cause of the atrophy simply because those symptoms were absent.

Plaintiff's evidence

114. The plaintiff did not vomit or feel nausea after the fall. He only experienced 'minor swelling and discomfort' but no pain in his scrotum by the time of his discharge. The pain was only felt the following morning, when he woke up.

115. A/Prof Foo Keong Tatt said that if the pain was very severe, there would be severe nausea and the patient would vomit. Dr Schlegel also said that in cases of blunt trauma to the testicle causing damage, there would be a sudden and dramatic increase in pain accompanied normally by nausea and vomiting at that time because of its severity. From this evidence, it appears that severe pain is likely to cause vomiting and nausea. Since Mr Harte did not feel any intense pain, it is not inconsistent that he felt no nausea and did not vomit. But not all blunt traumas cause severe pain. Thus, the absence of severe pain, vomiting and nausea after the fall is again inconclusive.

116. Based on table 3 in the paper '*Blunt Scrotal Trauma: Emergency Department Evaluation and Management*' by Munter and Faleski, it was submitted that there should be symptoms of vomiting and nausea whenever there was testicular or scrotal contusion. The article itself referred to a case report of a 9- year-old boy who sustained a blunt straddle type injury about 24 hours before. He was brought for evaluation after persistent swelling, discolouration and right testicular pain. Despite this, there was **no nausea, vomiting or fever**. He had been comfortable in bed but had significant pain when walking which was in an exaggerated straddle type of gait. Here, an ultrasound showed that the left testicle was normal but the right testicle was moderately enlarged with intact tunica albuginia and epididymis. Within 36 hours, the pain and swelling resolved and the patient did not suffer atrophy. This example clearly drives home the point that not all trauma injuries must be accompanied by symptoms of nausea, vomiting or fever. It is overly simplistic to conclude that the plaintiff could not possibly have suffered any testicular or scrotal contusion because these symptoms were not manifested. As with all symptoms, I must emphasize that their absence or presence is not conclusive but may only be indicative of a particular medical condition.

117. The aforesaid table 3 shows that the risk of significant morbidity if misdiagnosed in the case of testicular or scrotal contusion is not high compared with torsion injury and testicular rupture or epididymal rupture. I understand the argument to be that testicular or scrotal contusion is thus not likely to result in any testicular atrophy. In my opinion, table 3 compares the risk of morbidity for contusion injury with the far more serious cases of (a) torsion at the spermatic cord level which can choke off all the blood supply to the testis; and (b) rupture of the testis or epididymis. It is going too far to infer from table 3 that morbidity in contusion injury is ***per se*** unlikely.

Mrs Harte's evidence

118. According to Mrs Harte, the plaintiff was '*alarmed at the swelling in his scrotum'* on the morning after the surgery. It was more than the swelling after the first varicocelectomy operation by Dr Dubin. The plaintiff's scrotum was '***bright*** *red initially when he woke up. Over the next few days it turned* ***reddish purplish***'. She never said his scrotum appeared blue-black, blackish or dark in colour. But clearly, she did notice a change of colour to a deeper or darker colour.

119. Reddish purplish is actually a mixture of red and blue, giving the purple hues. Colour perception is very subjective and it depends also on the kind of light under which she observed Mr Harte's swollen scrotum. In my mind, some reddish inflammation coupled with a blue-black bruising might well manifest in a colour combination, which appears darkish reddish purplish in colour. It is not possible to rule out scrotal haemorrhage being mixed with the inflammation. When the inflammation

resolves itself faster than the blue-black bruising, then a tinge of blue could well remain after several days. Thus it is not that simple and straightforward to determine or infer reliably from the colour changes the actual cause of the extratesticular swelling, whether it is exclusively the result of haemorrhage due to contusion or traction, or inflammation *per se*, or a combination of both inflammation and haemorrhage.

Dr Tan Eng Choon's and Dr Jimmy Beng's evidence

120. Counsel for the plaintiff referred me to the following testimony of Dr Tan Eng Choon, who treated Mr Harte in October 1997:

> A: From the history of the surgery and history of swelling after the operation, I suspect that a vascular event had probably taken place, which resulted in the atrophy of the testes. A vascular event means that his blood supply to the testes has been compromised meaning reduced, or the swelling could be compressing on the vital structures, which is the testes; and that resulted in the reduced blood flow, which caused the testes to undergo atrophy.
>
> Q: Did you think **at that time** that the atrophy would have been related to the surgery?
>
> A: I suspected very strongly.

121. However, when Dr Tan Eng Choon was asked if he had been told during the consultation that Mr Harte fell in the toilet soon after the operation, Dr Tan said he had no such information based on his record.

122. Dr Jimmy Beng who examined the plaintiff in May 1999 (some 19 months after Dr Tan Eng Choon) similarly took the position that '*in view of the history given that there was a severe swelling after the surgery… some damage had occurred to the testes, most likely of a vascular cause'*. He referred to the biopsy performed on Mr Harte's atrophic testes on 21 October 1998 and the pathology report, which mentioned '***germinal tubules sclerosis and hyalinization***'. Dr Jimmy Beng explained that 'sclerosis' meant scarring and 'hyalinization' was a change in the protein structure of the cells. These changes were not consistent with an infection and could be observed when an organ such as the testes was deprived of blood supply.

123. I remind myself of the various objections which I shall not go into, one of which was that both Dr Jimmy Beng and Dr Tan Eng Choon subpoenaed by the plaintiff were not willing to be expert witnesses. Hence, I limited their evidence to the medical findings reached by them at that time as part of their treatment of Mr Harte. They were merely witnesses of fact and no more.

124. It appears that the fall was not made known to Dr Tan Eng Choon and Dr Jimmy Beng. Not having the full history, it is not surprising that Dr Tan Eng Chuan readily suspected that the surgery was the sole cause of the atrophy since that was the only known event occurring earlier. But where there are two or more known events, then each must be separately evaluated to see if each can cause the same result. If some or all of them are capable of causing the same result, then one must thoroughly evaluate the causative factors in each 'capable event' and balance all the probabilities before coming to any conclusion as to which 'capable event', or even which combination of the several 'capable events' is the likely cause of all the vascular damage. This exercise of fact-finding is hardly an easy task where there is more than one 'capable event'.

125. Since Dr Jimmy Beng and Dr Tan Eng Choon did not have the benefit of performing this diagnostic exercise (unlike the medical expert witnesses called by the plaintiff and 1st defendant who were required to do this evaluation), their evidence would not have as much probative value in relation to the correct inference to be drawn for the proximate and probable cause of the vascular damage, although I do accept (a) their medical finding that vascular damage had indeed occurred, and (b) Dr Jimmy Beng's explanation for the presence of 'hyalinization' which he said could be seen when an organ was deprived of blood

supply.

126. In my opinion, a contusion injury to the testes due to a fall may cause internal haemorrhage within the tissues of the testes. The resulting testicular haematoma and buildup of pressure inside the testes can also shut down the blood supply leading to 'hyalinization'. Thus the presence of 'hyalinization' *per se* does not indicate that it must necessarily be due to the devascularisation flowing from some negligent surgical operation. Thus both events are equally capable of producing the same end result of 'hyalinization'. One cannot so readily infer from the presence of 'hyalinization' that the cause cannot be due to a physical injury from a fall.

127. That there is 'sclerosis' and 'hyalinisation' as found in the pathology report is not disputed. In fact, that the damage is vascular in nature is also not in dispute at all. The dispute is solely whether the deprivation of blood supply and the resultant vascular damage is more likely to be caused by the surgery or by the fall. I observe that no party has adopted any intermediate position that both events might well have contributed to the vascular damage, with one being the dominant cause and the other, the secondary cause. As such, I shall not address this point since the parties themselves have not regarded that as a reasonable possibility. All three parties pursued the 'all or nothing approach' i.e. either the surgery or the fall was the cause but not both.

Dr Schlegel's and Dr Notley's evidence

128. Dr Schlegel and Dr Notley testified that substandard surgery was the cause of the vascular damage. Dr Notley's belief was that the testicles were infarcted because Dr Tan operated on Mr Harte who no longer had varicoceles. The previous varicocelectomy had removed the varicocele on the left and there was no varicocele on his right. After the operation by Dr Tan, Mr Harte therefore ended up with an inadequate venous drainage, a serious venous congestion leading to reduced blood flow, a lowered oxygen saturation within the testicles and the deadly sequence of infarction.

129. Dr Notley considered Dr Koay's ultrasound report to be ambiguous. He believed that ultrasound was not a reliable tool of investigation. It would have to be backed up by the presence of a clinical varicocele.

130. In Dr Notley's opinion, a doctor should not operate on an assumption. If an assumption was made that a patient had a varicocele, which could not be demonstrated clinically or unequivocally on ultrasound, the number of veins taken should be limited very severely. Dr Notley's inclination was that no operation should be performed unless the doctor was completely certain. If he was not, then it was substandard management. In his view, Dr Tan had operated improperly on an assumption that Mr Harte had varicocele. His ligation of the veins subinguinally to deal with his infertility by further varicocele operations was therefore substandard. The fact that Mr Harte ended up with bilateral infarction flowed from Dr Tan's substandard management.

131. Dr Koay himself testified that there was in fact evidence of varicocele on the left side and mild varicocele on the right side. He mentioned 'intermittent' varicocele on the left side to mean 'mild to moderate' varicocele. 'Intermittent' meant that the varicocele appeared under stress. Without stress, it was not seen.

132. Dr Tan read Dr Koay's radiologist report and considered the description used by Dr Koay of 'intermittent' varicocele to mean the presence of varicocele. I accept that Dr Tan had not misinterpreted Dr Koay's report. To understand 'intermittence' in the context of a varicocele, an example may be appropriate. Clearly, there is no such thing as 'intermittent cancer'. Just as with cancer, a varicocele represents a medical condition. It is not as if the varicocele medical condition is an intermittent one. The 'intermittence' is actually a manifestation of a varicocele vein because the reflux of blood is seen as intermittent in the sense that the blood refluxes when pressure is applied. That description may have been confusing to Dr Notley, but not to Dr Koay and Dr Tan.

133. Further, Dr Tan testified that he found varicocele veins when he operated on Mr Harte. In that regard, Dr Koay's and Dr Tan's diagnosis based on the CDUS had been surgically confirmed to be correct.

134. In the circumstances, I have to reject Dr Notley's opinion that Dr Tan had operated merely on an assumption that Mr Harte had varicocele and therefore he had been substandard in his management.

135. Dr Notley made a clear distinction between operating on a patient with varicocele and on one without. For a patient with varicocele, it was unlikely that he would suffer from testicular infarction when the vas was isolated and not ligated. However if that was done for a normal person, there would be a much greater risk of infarction because a normal person would not have those extra veins, which a person suffering from varicocele would have. When counsel for the plaintiff referred Dr Notley to the medical literature showing that leaving one set of vasal veins was sufficient, Dr Notley explained that every one of those articles assumed that the operation was on a man with a clinically evident varicocele and therefore, leaving a set of vasal veins was sufficient for drainage.

136. However, Dr Schlegel contradicted Dr Notley's evidence. In his testimony, Dr Schlegel emphatically said that the vasal vein alone would be enough to drain the testes to avoid infarction. The rest of the internal veins and external veins could be tied up. This was true **both** for a man suffering from varicocele and one who was not. From his experience at Cornell where thousands of such operations had been performed based on such a procedure using the vasal veins as the only source of drainage, there had never been a case of testicular atrophy. Therefore, if Dr Tan had left one set of vasal veins behind for venous drainage as he said he had, his surgery would not have caused atrophy, regardless whether Mr Harte had varicocele, minimal varicocele or no varicocele. In my opinion, Dr Schlegel's evidence here is very damaging to the plaintiff's case.

137. In Dr Schlegel's view, inadvertent injury during surgery to the arteries supplying blood to the testicles could lead to devascularisation. As a matter of causation, this could not be disputed. Dr Tan agreed that if the internal wall of the artery was damaged (i.e. endothelial injury) then:

> a) platelets in the blood adhere to the injured sites on the endothelium (inner wall of the arteries) and clots would form;
>
> b) thrombosis might result; and
>
> c) this could lead to ischaemic injury to the testicle, resulting eventually in testicular atrophy.

138. Dr Tan maintained however that he did not injure any artery during the surgery. He had carefully lifted up the spermatic cord during the surgery by using a Babcock forceps to grip and lift the cord. He then slipped his finger under the cord to bring it up. I doubt that the arteries in the spermatic cord had sustained any endothelial damage from such handling by Dr Tan.

139. Since there are three arteries supplying blood to each testis, one needs to inadvertently damage more than one artery on each side before devascularisation can happen. Having evaluated this, I think it is unlikely for Dr Tan to make so many consecutive errors. In any event, if it is a shutdown of blood supply due to total arterial blockage, one should not see swelling. Since there was intratesticular swelling, I have to rule out this mode of causation.

140. Dr Notley then attributed the colour changes on the plaintiff's scrotum to infarction after surgery. He explained that the scrotum and testicles have a rich blood supply. The very dense subcutaneous plexus of vessels in the scrotum act as a radiator so that large amounts of heat can be lost. A scrotal haematoma is caused by rupture of one or more of the vessels in the scrotum and that results in bleeding into the tissues of the scrotum. Within the first hour, there is not much colour change. As the bleedings come to the surface, the scrotum looks a bit blue. Gradually, it becomes more and more dense. The scrotum will rapidly become black within two or three hours after the injury. By twelve hours, the scrotum is usually black and swollen for a **severe** haematoma. The haematoma is contained within the fascia planes but may spread to the penis, which is shared. The blackening will probably reach a peak in about 12 to 24 hours, stay black for two or three days, and then it will fade gradually, going yellowish, greenish and through 'all the colours of the rainbow'.

141. The colour change is due to the deoxygenation and the haemoglobin in the blood cells left within the scrotum. This

haemoglobin is broken down by the local activity of the inflamed cells in the scrotum that changes the pigment into the bile pigments, which are yellow and green. Hence, a black haematoma gradually goes through this varying change of colour through yellow and green. It takes about 10 to 12 days to subside. Dr Notley said that anti-inflammatory drugs would not make any difference to that natural process.

142. Dr Notley expected Mr Harte to have an obvious scrotal haematoma by the time he left the hospital if the fall had caused it. A scrotal haematoma usually takes several hours for the colouration to appear. **It depends on the severity of the injury, the nature of the injury, the size of the scrotum initially and a multiplicity of factors. It varies from individual to individual.**

143. I agree entirely with Dr Notley's opinion, which I have highlighted in bold. I observe that Dr Notley's evidence of a black scrotum was made in the context of an extremely severe haematoma or a very serious injury. I certainly do not think that remains invariably true of a less severe injury.

144. By way of contrast, the physical manifestations of an infarction according to Dr Notley are as follows::

> If a patient has an isolated testicular infarction, there will be initially no discolouration of the scrotum at all. … After about eight to ten to twelve hours, the reactionary swelling in the scrotum around the infracted testicle ... will become red and it will look as if it was inflamed. And it will do that because of the reaction of the scrotum to the infarcted testicle within it. It is the standard reaction of the body to anything that happens to it which is the classical changes of the inflammatory reaction which are redness, swelling, pain and tenderness, heat. …….. So for testicular infarction, after about three or four days, the patient will have a large firm scrotum which is this curious plumish colour discoloration, rather light plum colour, rather the colour of the chair, the leather in the chair in which you are sitting in, and it will gradually fade subsequently after that but it will not go black. ….. I believe that the appearances of a testicular haematoma are so dramatic, they really are very dramatic, it's black ..
>
> If it's an injury, it will be black, … I think within a few hours, 6 at the most. … I personally put it blackish blue.
>
> …. If we took Mr Harte's case, I would have expected him to have an obvious scrotal haematoma by the time he left hospital……. To start with, it would look a bit blue as the bleedings came to the surface and gradually, it would become more and more dense.
>
> … I'm absolutely confident that 12 hours after sufficient injury to produce a haematoma ..the scrotum will be black.

145. Dr Notley opined that testicular infarction not associated with any physical trauma would cause the scrotum to be bright red. Gradually the colour would become more dense: reddish brownish. That colour stays for about 4 to 5 days. Gradually, as the swelling subsides, the inflammatory reaction in the scrotum also diminishes. But the scrotum will not turn black, unlike a scrotal haematoma.

146. During cross-examination by counsel for the 1st defendant, Dr Notley explained that infection would make the situation worse for an infarcted testicle. The patient will run a temperature, and if the situation is not diagnosed, there will be an abscess because the tissue is dead. If the surgeon does not drain it, the abscess will burst and discharge itself. He agreed that infection might follow infarction. The scrotum will then be very bright red. When the pus comes to the surface, it will be yellow. Inflammation does not show up as purplish or bluish black, which can only be produced by a haematoma under the skin. Dr

Notley could not think of any mechanism that would produce a bright red coloured scrotum, which later became bluish purple or bluish black.

147. Where there is loss of blood supply, the testis turns pale in colour but the scrotum skin will probably retain its normal colour because the blood supply to the scrotum has not been interfered with. Dr Notley however did not think that Dr Tan tied up the arteries to Mr Harte's testes. It would have to be a very 'ham-fisted operation' and a very substandard operation if three arteries to each testis were tied up inadvertently. I share this view too.

148. Dr Notley disagreed that a 'tinge of blue' would describe a haematoma. Neither would that be an appearance from a contusion injury. If he had to offer an explanation for the 'tinge of blue' colour observed by Dr Tan on 2 May 1997, Dr Notley proffered the incredible explanation that perhaps Dr Tan was seeing the 'black testicles shining deeply within the scrotum' in the same way that our veins could be seen underneath the skin. I find Dr Notley's explanation unconvincing having regard to the number of tissue layers protecting the testes, which are buried deep within the scrotum.

149. When asked why the larger testicle ended up being smaller, Dr Notley said that the plaintiff had partial atrophy of his testicles from the histology of the subsequent biopsies. So one testicle underwent further atrophy. It did not make a difference whether it was due to contusion or infarction. In his opinion, no deductions could be made as to which mechanism or pathological process was operating.

150. Dr Notley conceded however that infarction of the testicles from trauma could occur (without an associated scrotal haematoma) if the trauma specifically damaged the blood vessels to the testicle in isolation without damaging the blood vessels in the scrotum. However, Dr Notley thought that it would be very difficult to damage the blood vessels within the testes without damaging the blood vessels in the scrotum at the same time.

151. Dr Notley explained infarction as the death of a piece of tissue due to the abolition of the blood supply to it. One could of course abolish the blood supply to a tissue by a blood clot, or by stuffing up the veins so that the blood could not flow. Contusion on the other hand might cause haemorrhage of the blood vessels. If there was haemorrhage in the testes with coagulation, Dr Notley agreed that the testicles could also infarct. Dr Notley said that if it was possible to have contusion of the testicular tissues without damaging the extratesticular tissues, the scrotum could still turn red because of the infarction in the testes. The undamaged arteries to the scrotum would dilate to produce the inflammatory red reaction, whereas spillage of blood from damaged arteries into a foreign place where it was no longer oxygenated would cause it to turn blue-black. The following testimony by Dr Notley is relevant:

> His Honour: ..The infarction is very simple, in a contusion, you contuse it, the vessels burst, and the vessels burst, the blood coagulates, and no blood supplies come from that, you get an infarcted testis.
>
> Witness: Yes, you certainly will but—
>
> His Honour: So therefore the reaction for both infarcted testes, whether – whatever means of causation, you land up with a reddish coloured looking scrotum. That is provided the skin is not damaged.
>
> Witness: But I'm making the point, and I'm sorry to keep going back to it---
>
> His Honour: Yes.
>
> Witness: -- as I do not believe you can contuse anything in that part of the world with—
>
> His Honour: I'll leave that aside, I'm coming to that—

Witness: --without creating a haematoma.

His Honour: Yes, I'm coming to that.

Witness: But if you could in some magical way do that without contusing the tissues outside artery, yes, you're absolutely right, I agree with you, if you could infarct the testicle in some way, you would end up with a situation of the bright red swollen scrotum 12 or so hours later. So that I would go along with that completely. I just put the caveat that I don't believe you could do it without producing a haematoma.

His Honour: So now, the test now really is, would passing through a smooth surface, smooth hard surface on one side only, produce damage to the skin? Because I don't expect the other side which touches his body will produce the skin trauma. The other side, which is smooth, well, it's a big question mark.

Witness: But what we're damaging with an injury to produce a haematoma is the blood vessels within the substance of the scrotum beneath the skin. There are no blood vessels of any size in the skin—

His Honour: So it's just sub-skin?

Witness: Underneath the skin, there is this rich plexus of vessels which I refer to, which is the radiator to keep the scrotum cool.

His Honour: OK, let me ask you this question. Because the scrotum is soft, right —

Witness: Yes.

His Honour: And if I were to press it, like you see here (demonstrates), OK, and release after less than a second, and because the vessels in the skin outside the testis is not packed into a balloon, will it burst these skin capillaries to create that blue-black situation?

Witness: Wouldn't be the capillaries, your Honour.

His Honour: Oh, those whatever blood vessels they may be, small arteries.

Witness: It would be the small blood vessels, veins and arteries—

His Honour: Veins and arteries.

Witness: -- underneath the skin. I believe they would, yes Sir. I don't believe you could create sufficient force onto a scrotum to rupture the blood vessels inside the testicle without rupturing the blood vessel outside the testicle. Blood vessels are the same whether they are inside or outside. It's nothing to do with being in a ---

His Honour: The difference is the tunica. That's what I'm concerned with. The difference is the tunica.

Witness: But the force you apply—

His Honour: If all of them don't have the tunica, then I will agree with you. My problem is that this one is in the tunica, so there lies a difference.

Witness: Well, I can't agree with you, Sir.

His Honour: You can't agree with me. OK, fine.

Witness: I'm sorry. I believe you would damage the blood vessels in the scrotum. Blood vessels shear under these circumstances, and if you're rolling this chap off the toilet seat between his weight which--- I don't know quite what those kilograms are but, if his weight is bearing down sufficiently to produce your theoretical 15 millimetres, that is going to produce a sufficient shearing force, as it's rolled over, which, if it damages the testicle itself, it will damage, to a lesser extent, I accept, the blood vessels in the scrotum, and that would produce a haematoma. I just do not believe the two things can be divorced. Doesn't make sense, it's not logical.

Q: Dr Notley, do you have any medical literature to back up what you have just said?

A: No, of course I don't. Why should I have?

His Honour: No, you see, Dr Notley, you see, if what you say is right, then under —the part of his body near the, or rather, between the anus and the suspended point of his testes which the testes is in contact, in the process of passing through, one would also expect a blue-black. Biologically, but one wouldn't—I don't think so.

Witness: No, I don't think you would, because the blue-black, the thing that is being crushed is the scrotum, not the—

His Honour: Yes, it's the pressure, it's the pressure because the pressure acts both ways, so this is the hard part of the toilet seat, this is the testis and on top is his body. So if you expect the skin here to be blue-black and the skin on the top of the scrotum is blue-black, I would expect at the same time that the skin underneath, on his body side, to be blue-black because they are all subjected to the same pressure.

Witness: But I sit on that piece of skin all my life, Sir, and I don't make haematomas.

His Honour: That's precisely the point, that's precisely the point. You sit on the skin all your life, there is no blue-black there.

Witness: But I don't sit on my balls.

His Honour: Ah-huh, that's what I mean by the difference, the difference is in the tunica. You sit on the skin, it's no problem.

Witness: Or I sit on my scrotum as well.

His Honour: The pressure could be very high, that's my point, ah. Can you explain? You're sitting on the skin for a short while there, the skin of the scrotum and not the skin of your flesh, no doubt. But there is no [haematoma.] Why?

Witness: I mean, I think this theory is not a tenable one because I just don't believe you could roll a large man across a toilet seat or a flat floor or a set of stones sufficiently to compress his scrotum and its internal contents, so that you'll get an intratesticular haematoma without shearing some of the blood vessels in the scrotum. It simply does not—it's not believable. I mean, the basis of injury is that blood vessels get hit, they tear, they produce bruises, there is no literature as far as I know about men falling off their scrotum and squashing their testicles.

His Honour: No, no, that's a different ball game, that's a physical problem which our engineers will be dealing with. I'm just interested in the physiological aspects of it. You carry on.

152. Counsel in re-examination asked Dr Notley whether there were differences between the scrotal skin and the skin of the buttocks for instance. He said:

The scrotal skin is lax and very elastic, it's not very thick, quite thin. The skin of the perineum, the area of the sit-upon which lies between the anus and the buttocks is much thicker, it's more securely fixed, so when you slide about on a chair or a bicycle saddle, it doesn't move, so you don't get shearing effects. The scrotal skin is, as I say, lax, and you can easily get shearing effects particularly if you roll the scrotum or move the scrotum in the way that has been described and if you roll yourself off a toilet seat in this way and shear your scrotum sufficiently badly to produce an intratesticular haematoma, I can entirely imagine how the shearing would produce haematoma, but I can't imagine that the slide of the smooth, much more supported skin of the back of the thighs and the back of the buttocks and the perineum would shift in the way to produce shearing and haematoma formation. Obviously, if it was a direct contusion, like a kick or a hammer, then, yes, you would certainly get a haematoma on the perineum or the back of the buttocks, but I don't think a slide of this shearing nature would produce that lesion in those areas.

Q: Mr Notley, what's the incidence of fat in the scrotal skin as opposed to, let's say, the skin of the perineum or the buttocks or even the abdomen?

A: There is very little fat in the scrotum, very little indeed, whereas there is more fat underneath those other areas, parts of skin that you mentioned. But there isn't much fat beneath the perennial skin which is what you sit on.

His Honour: How much pressure must there be? Let's assume the experiment is one whereby you just apply pressure, without any shearing, just press the scrotal skin and then let go, will you get a haematoma?

Witness: Well, you could do it you pressed it hard enough and for several minutes.

> His Honour: What sort of pressures are you talking about?
>
> Witness: I don't really know, but a common example is the love-bite that people produce in the neck, that's pressure on the skin over a period of time, that's a haematoma.

153. Dr Notley then concluded that it was not uncommon for injuries to result in scrotal haematomas and only very infrequently did they get testicular atrophy afterwards. A scrotal haematoma (i.e. extratesticular haematoma) *per se* does not cause atrophy of the testes. The published literature suggests that the scrotum is normally injured where testicular atrophy follows sufficiently severe trauma to the testicle. There is no published literature indicating that one can have the sort of catastrophic damage, which Mr Harte had, without physical damage to the scrotum in the form of a haematoma or abrasion of the skin. Many of these very severe traumas actually disrupt the scrotum. Dr Notley was not aware of any reported cases of testicular atrophy due to blunt trauma in which there had not been externally apparent injury to the scrotum in the form in its most minor way of a scrotal haematoma.

154. Dr Schlegel testified that a force applied directly to the testes sufficient to cause bilateral testicular atrophy would also be applied to the scrotum skin tissues. Fairly significant bruising and damage to these tissues would be expected as well.

1st defendant's experts: A/Prof Li, A/Prof Foo and Dr Lei

155. Professor Li Man Kay ('A/Prof Li') is the Senior Consultant and Head of the Division of Urology, Department of Surgery at the National University Hospital. He testified that a compression injury might give rise to a haematoma, which could be either extratesticular or intratesticular, or both.

156. Where the testes have been crushed internally, blood vessels within are ruptured. That causes congestion. An intratesticular haematoma develops. Whereas an extratesticular haematoma usually results from a direct hit on the scrotal skin. The ruptured blood vessels in the scrotal skin and adjacent dartos muscles leak out blood, thereby causing the extratesticular haematoma. Bleeding of the blood vessels in the external spermatic, cremasteric muscle and internal spermatic fascia will also cause an extratesticular haematoma although this is less likely because these tissues are more mobile than the dartos muscles. The chances of injuring something mobile is less in a direct hit or in blunt trauma. In either case, the scrotum will appear black in colour. A very big black scrotum the size of a mango definitely indicates substantial bleeding underneath the dartos muscle and in the subcutaneous area.

157. A/Prof Li further clarified that if there is no damage to the blood vessels within the dartos muscle, but 'blood is poured into the scrotal sac' from another source, the scrotum skin will also look bruised and black because of the blood permeating through and causing discolouration to appear through the scrotal skin.

158. A/Prof Li then elaborated on the kinds of scrotal injuries that he had seen viz kicks, hits, fights, bicycle and motorcycle accidents, falls into drains and falls suffered by construction workers. The scrotum bruising showed up as a blue-black scrotum all the time.

159. I asked A/Prof Li specifically whether the dartos muscle in the scrotum would bleed in a particular situation where there is a rolling over the toilet seat when a person falls off, which is a different situation from the case when the scrotum is hit [i.e. by strong impact forces or the like]. He answered in the positive but with a number of what I believe to be important qualifications. He said:

> His Honour: OK. So do you think, therefore, the rolling over a toilet seat will cause the dartos muscle to bleed because it is not the hitting, you know. If he falls off, let's assume that he rolls over.

Witness: The thing is, it really --- if you look at it this way, if we purposely, like your Honour was saying "I squeeze, and then I pay attention that I only squeeze the balls and I don't squeeze any other things", that was a scenario that it could happen but what I would say is, if somebody is rolling over, we don't know the speed. We know that there's a ---

His Honour: The speed, let's assume it's slow, not a speed of a car, let's say, this is slow roll over.

Witness: Yes, OK.

His Honour: Like a pancake, you know, you prepare a pancake, you roll over, you know, the pancake, you have this thing to roll.

Witness: **And we also don't know how much pressure is there and how much pinching and crushing and weight and so on, we don't really know but we only know that he's a big size chap.**

His Honour: Yes. And would the dartos muscle in that situation be caused to bleed?

Witness: Yes. The scenario, your Honour, that you gave was you avoid but as far as I'm concerned, clinically, I have seen a lot of patients who had scrotal injuries and so on, I have never seen the scrotum being spared, the skin being spared. It is always the black bruise, the whole thing is black.

160. Due to the various qualifications, I cannot reasonably construe A/Prof Li's answer to be that the plaintiff would necessarily have a black swollen scrotum if he had a contusion injury. Many unknown factors come into play. The examples given (from which A/Prof Li derived his opinion that a black bruise invariably appears), are those where the impact forces are sudden and of short duration viz kicks, hits, fights, bicycle and motorcycle accidents, falls into drains, falls suffered by construction workers. I can accept that the colour of the swollen scrotum may be black in these categories of trauma because of the strong and sudden impact force which is likely to cause severe rupture and fairly extensive bleeding of the blood vessels within the dartos muscle, which with time may develop into a black swollen scrotum as described by A/Prof Li.

161. However, the nature and magnitude of the contusion forces acting on the testes in this case during a roll off from a toilet seat is totally different (assuming that the testes in the scrotum were caught and pressed between the hard but smooth toilet seat surface on one side and the soft area in the plaintiff's perineum on the other side, and then sandwiched between the plaintiff's fleshy thighs on both sides and his flaccid penis in the front). Clearly, these contusion forces acting on the testes and scrotum skin tissues are not impact forces. I do not think that the expected result will necessarily be the same as that from impact forces.

162. The rate and extent of the buildup of pressures and forces on the testes and scrotum skin tissues arising from a slow roll over are far more gradual by comparison. It is wrong to think that the entire weight of 103.7 kg of the plaintiff was crushing the scrotum and testes at the point of roll over because the thighs (resting on the toilet seat) would still bear a substantial part of his torso weight. For this reason, there is a fair probability that the contusion was not as severe as one might think, although it remained sufficient to damage the internal of the testes. Hence, it is much too presumptive to conclude that the absence of a black scrotum automatically rules out the possibility of a moderate contusion injury to the testes.

163. A/Prof Li's evidence of a black scrotum must be made subject to the several qualifications that he himself had made earlier. My conclusion is supported by A/Prof Li's evidence that it would be quite unusual to see a black scrotum where there was no bruising on the skin. If there is crushing or rolling over by something smooth, then the skin may well be spared and

hence, there will be no extratesticular bleeding in the scrotal tissues between the scrotal skin and the tunica albuginea, and there will also be no black scrotum because the blood vessels in between the skin and tunica are not damaged or at least not extensively damaged. But there can still be an internally big intratesticular haematoma because of the damage to the blood vessels within the testes due to the crushing or rolling over by a smooth object.

164. Photograph D 51G shows the smooth and rounded edge of the same toilet seat that Mr Harte sat on when he fell off. This photograph shows the position of his scrotum when Mr Harte was probably sitting upright as in photograph D51F and hence, his scrotum was lifted upwards. When his torso fell towards his thighs (i.e. when he would be bending downward and forward in the final stages of the fall), his scrotum naturally drooped much lower into the toilet bowl. This effect can be seen in photographs D 51 D and E. In this position, the risk of his rather large scrotum (which I think prior to the fall, would be even larger than that seen in photographs D 51D and E because he had much larger and presumably heavier testicles then) being caught and squashed between his body and the top of the toilet seat during the fall is in my view much greater. A/Prof Li said that he would not expect any skin injury from abrasion. I agree with him that the injury if any to the testicles, the dartos or to any structure in that area is likely to be caused by pressure principally.

165. During cross-examination of the 1st defendant's experts, Mr Kronenburg went on enthusiastically to explore all kinds of imaginable errors that Dr Tan could commit during his operation. I shall cite some of them, which I find to be largely improbable upon closer examination.

166. The first is the inadvertent ligation and/or cutting of blood vessels. A/Prof Li testified that if the veins were cut without tying them up and then allowed to bleed, a haematoma the size of a big mango would not form because the veins would stop bleeding after a bit of pressure. Unlike an artery, the pressure of the blood oozing out of the veins would be relatively low. So it was unlikely to push to form a swelling the size of a mango. But where an artery was cut inadvertently, the blood outflow pressure would be high. The resulting extratesticular swelling should consequently be much larger. But A/Prof Li discounted the likelihood of an inadvertent snipping of an artery during the surgery because a cut artery would spurt out blood like a small fountain. The blood would obscure the surgical field. Dr Tan would immediately realise that he had accidentally cut an artery. The necessary measures to rectify the mistake could then be made.

167. Counsel then suggested the possibility of tying up the arteries inadvertently together with the varicose veins during the surgery. No blood would spurt out if he were to cut both the artery and vein together. While it is true that the surgeon is none the wiser, but there will be no extratesticular haematoma although there will be atrophy due to devascularisation. This does not fit the facts of Mr Harte's case.

168. The significance of A/Prof Li's evidence is this. If indeed the vein ligations done by Dr Tan had slipped, the veins would be oozing out blood into the extratesticular area but that could only cause a small extratesticular haematoma due to the low venous pressure. Since a large scrotum was presented, it suggested that the cause could not be attributed to a failure of any of the 18 x 2 sutures used to tie up the 18 ligated and divided varicose veins.

169. Counsel for the plaintiff then asked A/Prof Li whether slipping of the knots at the inadvertently divided artery would lead instead to a big extratesticular haematoma. He agreed it would, provided it was the knot at the pressure end and not the distal end that slipped. If the distal end slipped, it was all right. A/Prof Li added that slipping of knots is one of the problems of a new surgeon or a trainee.

170. A/Prof Li alluded to an interesting point that the pressure within the scrotum was not very high at the time of the maximum swelling because it was pear shaped as drawn by Mr Harte. As an example, the pressure within a pear shaped balloon would not be as high as in a round balloon. The pear shape suggested that it was not likely that an open artery was pouring blood into the scrotum.

171. After considering the number of things that must go wrong for this mode of negligence, I find it most unlikely having regard to the fact that Dr Tan is a very experienced surgeon. Further, I do not think he is at the age where he no longer has steady hands and good eyesight. In order for the bleak scenario painted by counsel to happen, the defendant would have to:

(a) Inadvertently ligate and divide the arteries to the left and right testes. Ligation of the testicular artery alone does not produce testicular infarction. More than the testicular artery must be inadvertently tied up and divided on each side. That accounts for two consecutive major errors.

(b) Slipping of knots must occur separately on both sides as the scrotum cavity is divided into two compartments by a septum and Mr Harte's scrotum swelled on both sides.

(c) The knot slippage must be on the pressure side of the divided artery and not on the distal side.

172. The likelihood of all these independent events happening at the same time sounds absolutely remote to me. I thus rule it out as a fanciful possibility. The final answer from A/Prof Li that the blood flowing out of the artery would be able to track back up and result in bloody incision at the skin ends this speculation because the skin incision and dressing on both sides were found to be clean and not stained with blood.

173. With an extratesticular haematoma, A/Prof Li believed that one would see bruising and a black discolouration. Upon palpitation, the doctor would find a thick and less mobile scrotal skin. Normal skin is thin and mobile.

174. A/Prof Li confirmed that the black blood clots in the testes with intratesticular bleeding could not be seen through the scrotal skin. I agree with him. The skin should appear normal in colour although the testicle is enlarged because of the several layers enveloping each testis. Any change in colour of the scrotal skin can only be due to blood in the extratesticular region appearing near the dartos muscles.

175. A/Prof Li said that he understood Dr Tan's description of 'scrotal haematoma' in his clinical notes to mean a bruised and black scrotum.

176. When A/Prof Li's attention was drawn to the evidence of Mrs Harte of a bright red scrotum later changing to reddish purplish, A/Prof Li thought that it was not possible. He felt that her observation of a reddish-purplish scrotum was inconsistent with a scrotal haematoma because the main constituent of a scrotal haematoma is blood in the scrotum and that never goes reddish-purplish. Like all blood clots inside the body, it is black.

177. When it is bright red, it means that there must be blood on or near the surface of the skin. But blood is not bright red, it is dull red in colour and blood never turns purple and neither will it turn reddish purple. A/Prof Li testified that the colour of a haematoma is blackish, like the colour of hair. Therefore, haemorrhaging within the dartos layer would not result in a red scrotum. Similarly a slipped knot resulting in bleeding into the scrotum would not produce a red coloured scrotum. Again it would be black. In the case of a fall, it was not inconceivable that the knots at the tied up veins might slip. A blood clot inside the body would also be black. In a case of bleeding from a slipped knot, A/Prof Li postulated that the blood would take about an hour to accumulate and the blood clot would be fully manifested say by 6 hours.

178. In the case of ligation of all the veins, the testes would be fully congested within about 10 to 20 minutes. A nagging pain would be felt. It would not be a sharp pain. The testes would bloat up to about double its size volumetrically by the time the operation was over and the patient had awakened. He should be able to see that his testes were larger. But the scrotum would be smaller than a mango. However, if not all the veins were tied up, the congestion would be proportionally less.

179. Dr Tan wrote the following in his medical notes:

9 May 1997

Haematoma subsided

Complained of hard testicles, left worse than right.
Ultrasound scan: organised clot around left testicle, small hydrocele
Wants medical report.

180. A/Prof Li interpreted the 'organised clot around the left testicle' as an internal blood clot within the testicle and not one underneath the scrotal skin. He reinforced his testimony by saying that a scrotal haematoma underneath the skin is normally very hard to find on ultrasound because it merges with the tissues. **From the 'small hydrocele', he concluded that there was intratesticular bleeding**. When the clot forms within a cavity, it pulls down into two separate layers. The top layer is the serum i.e. the hydrocele, and the lower layer is the clot and this hydrocele was found inside the testis..

181. I consider this evidence to be very significant. It confirms that there was internal bleeding in the testicle. If the veins were merely tied up or too many veins were tied up, I accept that devascularisation could occur but I do not think there would be any bleeding. There might simply be thrombosis of the blood vessels and capillaries but there would be no rupture of these vessels or bleeding to leave behind an organised clot and a small hydrocele. This critical evidence clearly swings the balance of probability more in the direction of the contusion trauma and injury of the testes with rupture of the blood vessels within the testes. This conclusion is confirmed by A/Prof Li that with an intratesticular haematoma, then there must be trauma or injury to the testicle.

182. A/Prof Li did not think that an immediate operation to drain the blood from the testes was necessarily a good idea. It was another general anesthesia. Drainage of a haematoma may introduce infection because blood is a very good medium for bacteria to grow and the scrotum is just next to the anus, which is a potentially contaminated area.

183. With regard to the intratesticular haematoma, A/Prof Li was of the opinion that Dr Tan would not be able to do anything to remedy the situation. If the testicle was to be saved from atrophy, action should be taken within the first twenty hours or so. Beyond that it was hopeless. Even for an operation done within twenty hours of injury, there was no guarantee that atrophy would not result. Therefore, there was the possibility of salvage if the haematoma could be detected by 6.00 pm on the day of the operation. Timing would be critical. In his opinion, it was too late to save anything by 2 May 1997 if there was in fact internal testicular haemorrhage from the fall.

184. A/Prof Li then went on to explain the mechanism of necrosis. When the blood supply to an organ is reduced to 5 or 10% of what it was before, ischaemic changes occur. The organ may shrink in size. For instance, when there is a decrease in the blood supply to the leg, there will be pain. Later, the big toe turns black. As a result of a chronic lack of blood supply to the leg, the toe can even drop off.

185. Although A/Prof Li was unable to say what would happen when the blood supply to the testicle dropped to 5 or 10%, he accepted that it was possible to get ischaemic necrosis through a decreased blood supply. As everything is possible in a biological human being, it is also possible to have a situation that the epididymis is alive and normal but not the testis, because the oxygen via the blood supply is somehow just sufficient for the smaller epididymis but insufficient for the relatively larger organ, the testis. But he thought that a situation like this was unlikely and remote.

186. When the radiology report from Cabrini Medical Centre dated 21 October 1998 was shown to A/Prof Li by Mr Kronenburg, he agreed that the reduced perfusion sufficient to support the head of the epididymis was sufficient to support some spermatogenesis because 'decreased spermatogenesis with only a few spermatids' was found in the left testis although in the right testis, 'no spermatozogenesis' was found.

187. Based on the ultrasound report of Dr Koay on 11 September 1997, A/Prof Li understood that the testes were of small volume with atrophy having set in and they were almost dead. However the presence of tissue perfusion indicated that there was still some blood supply through the testes based on the CDUS scan.

188. Further the fact that both heads of the epididymis appeared normal as stated in Dr Koay's ultrasound report confirmed this because the epididymis shared the same blood supply as the testis. If indeed Dr Tan had inadvertently ligated the artery or

the veins, or in other words, if it were purely a blood supply or a venous congestion problem, the epididymis would have disappeared too. One could not have the testis atrophying but not the epididymis. Based on logical deduction, this substantially rules out the theory that all the veins/arteries, or far too many veins/arteries were tied up because both the epididymis and testes should die or atrophy together. A/Prof Li believed that if the epididymis survived a blood supply reduction of say 50%, the testes would similarly survive. But whatever decrease there might be in the blood supply sufficient to cause death of the testis, it should also be sufficient to cause the epididymis to die. I fully agree. As an illustration, cutting the blood supply to the hand will also cut off the blood supply to the fingers. But with trauma, one can damage the fingers without damaging the hand because it depends on where the physical trauma happens to be inflicted.

189. According to A/Prof Li, the fact that there was severe damage to the testes without corresponding damage to the epididymis in Mr Harte's case indicated that it was more likely the result of trauma or contusion than a case of loss of or insufficient blood supply. This evidence lends substantial support to the defendant's fall theory and undermines the plaintiff's theory of a loss of or severe reduction of blood supply.

190. Following this, counsel for the plaintiff rightly explored the question whether both the testes and epididymis would suffer contusion injury given the nature of the fall. A/Prof Li explained that the epididymis is a very soft tissue. In a crushing or rolling situation, these soft tissues can yield to the contusion. The epididymis is located at the top posterior of the testis. Assuming a fall from a toilet seat where the pelvic bone presses down on the testis, there will be sandwiching of the epididymis between the pelvic bone and the tunica. But there is the possibility of a rotation of the testis because there is a lot of freedom and the scrotal skin can stretch to allow the rotation. The significance then is that the forces are not acting directly on the epididymis if the testis rotates. To my mind, A/Prof Li has given a reasonable explanation why a contusion injury to the testes may not necessarily damage the epididymis. Unlike the testis, which is encased within a tough unstretchable membrane called the tunica albuginea, the epididymis is located outside this membrane. Being softer and having greater elasticity, the epididymis can easily stretch and thus yield to the compression because it is not 'compacted' or 'constrained' so to speak within a fairly non-elastic container or tunica albuginea, which is being flattened. For this reason, I accept that the epididymis can withstand pressure or contusion better than the testis.

191. Interestingly, Dr Schlegel gave evidence in support of the fall theory when he said that an injury affecting directly the testicle from trauma would not usually affect the epididymis primarily either.

192. Even Dr Notley appeared to concur . He did not see any reason why the epididymis would not survive when the testes were squeezed. Whether it was infarction or contusion, the epididymis might survive as it is a '*pretty tough bit of equipment*'. Thus the absence of damage to the heads of both epididymis does not rule out contusion as a cause of the bilateral testicular atrophy.

193. Associate Professor Foo Keong Tatt ('A/Prof Foo'), the Senior Consultant Urologist and Head of the Department of Urology at the Singapore General Hospital, was called by the 1st defendant as his medical expert witness. He testified that one would find bruising, ecchymosis around the scrotum and possibly some abrasion on the scrotal skin and penis were a patient to suffer contusion injury to the scrotal and testes sufficient to result in almost total testicular atrophy. Similarly, Dr Clarence Lei Chang Moh, a consultant urologist at the Normah Medical Specialist Centre, Petra Jaya, Kuching, Sarawak, gave evidence that the scrotum would appear 'darkish, dark blue' or 'bruised and dark' four days after trauma to the scrotum. It would not be red anymore. Then Mr Lek asked Dr Lei for his diagnosis if the grapefruit sized swollen scrotum had a 'bluish tinge' instead. Dr Lei thought that if the testis had been normal before and there was a history of a fall, and the result was a dark, blue-black scrotum, then it was probably due to the fall. He did not directly answer Mr Lek's question.

194. Dr Lei then went on to deal with the other scenario when the testes were pressed and rolled over between a smooth hard surface and the patient's perineum (i.e. space between the anus and scrotum) and there was intratesticular injury as a result of that. He explained that there would probably not be injury to the scrotal skin. He explained that the testis is an organ held by a very tight tunica and it cannot expand. Accordingly, the slightest pressure on it can cause intratesticular damage. In response to questions from Mr Lek, Dr Clarence Lei said:

Q : … if there was intratesticular injury, OK, as a result of that motion [i.e. pressing and rolling over], OK, would you agree that it is likely that at the time, there would also be injury to the scrotal skin? Assuming there was intratesticular injury, would that at the same time be injury to the scrotal skin?

A : There may not be. In fact, I think probably there will not be because the testis is an organ that is held by a very tight tunica and it cannot expand. So **even the slightest pressure on it can cause intratesticular damage,** and I think you can easily find out by doing your own experiment. You just squeeze your testes a little, it's going to hurt a lot because of that nature. It's just like the brain where it's inside a very tight capsule. So, unfortunately, for the testes, no, but if you say about the muscle, for example, you know, you damaged the muscle, then there will be associated injury. If the muscle is damaged, then the skin will surely be damaged but not for the testes.

His Honour: It's a very delicate structure, is it?

Witness: Delicate structure with a tight capsule. So you can damage it without damaging the skin very much, unfortunately, that's what we are born with.

Q: But if there was eventual total intratesticular atrophy, that means not just merely injury but there was eventual total intratesticular atrophy, would you agree that it is likely that there would be some injury to the scrotal skin?

……

A: ….**But my opinion is that, that you can damage the testes without significant damage to the scrotal skin because of this special arrangement.**

On the same issue, A/Prof Foo's evidence was:

Q : Would you require the tunica to stretch before you could say atrophy would occur?

A : The atrophy would ---the tunica itself cannot stretch because it's a very tough membrane, and that is the reason why when it bleeds inside within the testicle, the pressure builds up, it's like your skull, cannot stretch.

Q : So, in other words ---

A : So intra cranial haematoma is very dangerous.

195. From the medical and bioengineering point of view, I find these answers of Dr Lei and A/Prof Foo to be very sound, although it is a bit of an exaggeration to say that the 'slightest pressure' can cause intratesticular damage. More likely it is moderate pressure that can cause intratesticular damage. I do not wish to be overly semantic about these things but what is important in my view is the mechanism of damage that Dr Lei and A/Foo had alluded to in their evidence, which must take account of the special biological construction or structure of the scrotum and testes.

196. All the while, the plaintiff and the 2nd defendants proceeded on the basis that there must necessarily be some skin abrasion and/or serious bruising of the scrotal skin whenever there is a rolling over or squeezing of the internal testes. Clearly,

there can be internal damage to the testes without the dark colour associated with scrotal skin bruising. Because each testis is contained in a tough, fairly non-elastic capsule, any flattening of the capsule thus reducing its volume will cause the internal pressure to build up very rapidly in the tissue contents. Even biological matter must obey the universal laws of physics. For instance, in a case involving a fixed mass of a gaseous substance maintained at the same temperature, the pressure is inversely proportional to its volume. This is Bolye's law. The smaller the volume, the higher will be the internal pressure throughout that fixed mass of gaseous substance, and vice versa. In the case of a fixed mass of a liquid substance, which is largely incompressible, the pressure in that liquid substance will increase very much more when the volume is reduced even slightly by compression. Where a fixed mass of liquid is contained within a flexible capsule, and an external force is applied over a small area of the capsule (i.e. an external pressure is applied on a small part of the external surface of the capsule), the pressure in the liquid throughout the capsule will rise such that it will be equal to that external pressure being applied. Therefore, if a man were to sit on his scrotum and thereby exert a strong external pressure on the tunica albuginea encasing his testes, that strong external pressure will be transmitted internally throughout the testicular tissue within the tunica albuginea, assuming that there is no rupture.

197. Thus by flattening or compressing the tunica albuginea, the corresponding increase in pressure throughout the testicular tissue encased within the tunica albuginea may rise to a level where it causes microvascular damage by two modes (a) direct damage to the tissue cells and (b) rupture or tearing of the delicate capillary vessels with resulting haemorrhage within the testes due to the elongation or strain from the flattening process. A reasonably good analogy will be the air (representing the testicular tissue contents) enclosed in a balloon (representing the tunica envelop). Flattening the balloon will raise the internal air pressure all round and the balloon will be forced to elongate.

198. Therefore, moderately intense squeezing of the testis is likely to cause microvascular damage to the delicate tissues encased within the tunica albuginea, which can subsequently damage the whole or part of the testis.

199. I find support from the evidence of Dr Tay Kah Phuan who confirmed the two ways in which compression pressure causes damage. One is contusion pressure on the cells themselves and the cells die. The direct contusion force can also disrupt the tissue and cause it to die. The second is microvascular rupture of blood vessels. Due to the rupture, there is a buildup of pressure that can subsequently infarct the testes. Sometimes, one cannot tell which comes first.

200. A/Prof Foo also clarified that there are no nerves within the actual tissues inside the testis. With the squeezing of the testis, it is the stretching of the tunica covering that causes the pain, not the damage to the internal tissues of the testes *per se*.

201. I then clarified with A/Prof Foo on the mechanism of the pain in the scrotal region:

> A: So, a pressure causing – a pressure that would cause the tunica to stretch would also cause pain?
>
> His Honour: No, you see, the cause of the pain is the stretching ---
>
> Mr Kronenburg: Yes.
>
> His Honour: So, if it is damage *per se* within the layer without stretching you won't feel pain. That's why I've to establish that. It's only after the thing stretches due to congestion and so on and coagulation, then the pain begins as it stretches. Because the stretching is where the nerve detects the stretching. That's where you get the pain. If the tunica doesn't stretch, you damage the inside, he says you won't feel the pain because there are no nerves inside.
>
> Witness: That's right, your Honour.
>
> .....

Q : Professor, if the Plaintiff had a squeezing contusion injury to the extent that atrophy resulted, would he feel pain – yes, maybe, or no? If the Plaintiff had a squeezing contusion injury to the extent that atrophy resulted, wouldn't he feel pain --

His Honour: When, the question is when.

Mr Kronenburg: Yes, I was going to ---

Witness : That's what I'm going to explain. Initially there may be pain.

Q: But subsequently?

A: Subsequently there may not be pain ---

Q: Immediately after?

A: Immediately after, there may be pain and then the pain will subside, but as the pressure builds up within the tunica ---

His Honour: Pain comes back again?

Witness: That's right, your Honour.

His Honour: All right.

Q: So there will be pain and the pain will disappear, then the pain will come back again?

A: That's right, your Honour.

Q: But you're certain there will be pain at the outset?

A: Initially. Yes.

His Honour: Which is for a short while, is it?

Witness: Yes, Your Honour.

202. Dr Notley in his rebuttal evidence substantially confirmed what A/Prof Foo had said was the pattern of the pain: "*And, of course, with the infarction as with the haematoma, the pain will subside over the next few days. And so by four days, it might not be so painful.*" Dr Notley explained that the pain in testicular contusion or injury would be felt instantly as the injury occurred. The pain intensity would probably be very high at the time of the injury and would last for a few minutes. But over the next few minutes, the severe pain would subside leaving the patient with a nasty and unpleasant ache. And if the testicle was injured, that nasty unpleasant ache would gradually increase over the next few hours because the full force of the patient's blood pressure would be pushing into the swollen testicle causing tension in the inelastic tunica albuginea. So the pain would come on within a few hours of the dull, unpleasant natured pain.

203. In contra-distinction, infarction would not be painful initially, no more painful than a patient who had excision of a varicocele which did not produce infarction. Because the pressure would be gradually rising within the testicle and within the tunica albuginea, this pain would be a dull pain, not a sharp acute pain. According to Dr Notley, the dull pain would come on gradually over a matter of hours, building up, starting probably as a mild ache a few hours afterwards and then getting worse

and worse, and probably peak ten to twelve hours later.

204. The apparently irreconcilable evidence is that by 7.00 p.m., Mr Harte still did not feel any pain except discomfort and a dull ache at his scrotum when he left the hospital. If there was any severe contusion injury, I think he should have felt the pain. Since there was no complaint of acute pain, it appears either that (a) there was no contusion injury, or (b) the contusion injury might only be moderate in magnitude. This calls for closer examination.

205. Since Mr Harte could feel the pain from the bump on his head, I have to rule out the possibility that the pain killer tablets consumed by Mr Harte after surgery (if any) and the remaining effects of the general anaesthesia were of such magnitude that they completely removed his ability to detect pain in his scrotum. I expect that Mr Harte would be more sensitive to pain in his testes than on his forehead. I also note that Mr Harte could feel discomfort from the minor swelling in his scrotum when he was recovering in the day ward after the surgery. This again suggests to me that the anaesthesia wore off fairly quickly. If indeed there was a severe and substantial blunt trauma to the testes, it is surprising that the acute pain would have escaped Mr Harte. But where the blunt trauma is moderate, then it is not beyond believability that Mr Harte could not, after regaining consciousness, detect the pain because it might not be as acute and that pain might have passed away or significantly reduced by the time he recovered from his syncope. It is not improbable that Mr Harte did not complain of the pain because he was already expecting discomfort, pain and swelling from the testicular congestion due to ligation of veins, which is inherent in a varicocele operation. As Dr Tan's operation on Mr Harte was bilateral in nature (as opposed to the unilateral varicocele operation on the left side by Dr Dubin), I suppose even greater pain and discomfort were to be expected. Thus, Mr Harte was content not to complain.

206. I find that the forces involved in a non-violent roll over and compression/squeezing of the testes are dissimilar in nature and are probably of lesser magnitude in comparison with those in the typical impact trauma situation involving for instance, a violent kick, or a motorcycle accident, where a sudden large impact force hits the scrotum and testes. In my considered opinion, the contusion in Mr Harte's case involved only a moderate non-impact force. It was more a gradual squeezing of the testes, as his heavy body rolled over his scrotum during the fall off the toilet seat. The duration of application of the force would be about a second or less. The resulting pain envisaged in this situation would probably not be acute and would have diminished quickly and not be noticeable by Mr Harte by the time he regained consciousness. The absence of a rupture of the tunica albuginea also suggests that the compressive force was not particularly severe. A/Prof Foo did not think that Mr Harte suffered any rupture of the tunica albuginea based on the medical evidence.

207. Nevertheless, the probability of internal microvascular damage cannot be excluded. If the damage is microvascular in nature within the testicular tissues (which I am made to understand do not have any nerves), I doubt very much that the microbleeding itself is going to cause much pain. When the microbleeding or haemorrhage feeding the growing intratesticular haematoma starts to swell and stretch the intact tunica albuginea and exert pressure on it, then the pain will begin. As the intratesticular swelling and pressure slowly increase, the pain will also gradually increase. Looked at from this angle, I find that the pain symptoms from moderate contusion and microvascular damage are largely consistent with the absence of clear signs of acute pain and the presence of a dull pain and some minor swelling when Mr Harte was in hospital waiting to be discharged. They are also consistent with the acute pain that Mr Harte felt only on the following morning after the surgery, when he experienced fairly substantial swelling.

208. The important outcome of the above analysis is that the absence of acute pain does not rule out contusion as a probable cause of the devascularisation damage in the testes. I believe that the absence of acute pain can only exclude severe impact injuries and severe contusions **but not those which are moderate in magnitude**.

209. A/Prof Foo was then asked specifically to describe the pain pattern for a case of trauma leading to intratesticular injury sufficient to lead to testicular atrophy. His view was that Mr Harte would slowly develop pain and a scrotal haematoma. Unless the pain was very severe, he might sometimes feel nausea. If there was severe nausea, the patient would vomit. After 4 days, there would be a big haematoma. It would be difficult to feel the testes, and therefore, it would be difficult to ascertain whether the haematoma was extratesticular or intratesticular. But A/Prof Foo thought that sometimes the pain would subside by the fourth day or the person might get used to the pain. The haematoma would harden.

210. As for venous congestion due to an inappropriate or negligent surgical procedure, A/Prof Foo agreed that it would take a few minutes for the pressure to build up in the testes after tying up the veins. However, it would take a few hours for the swelling to build up depending on the extent of the venous impairment or congestion. Interestingly, A/Prof Li said that the pain arising from venous congestion was not sudden but would build up slowly. With partial and not total impairment of venous flow, the congestion might take longer to build up. If this was the mechanism of devascularisation, then it would be similar to the discomfort and dull pain felt after the operation and over the next 12 hours.

211. I am persuaded by the preponderance of expert medical opinion to conclude that the absence of acute pain cannot exclude either venous impairment damage through careless surgery on the one hand, or microvascular bleeding and haemorrhage from contusion damage on the other. That also means that the absence of vomiting and nausea felt by Mr Harte after the fall does not exclude either a surgical cause or contusion damage.

Defence of 1st defendant

212. The 1st defendant, Dr Tan, attributed the testicular atrophy entirely to the fall in which the plaintiff's exposed scrotum was squashed between the toilet seat and his heavy torso, as his body collapsed forward and eventually slid off the toilet seat in an unconscious state with his head hitting the floor first.

213. On being told of the fall and the injury on the head, Dr Tan sent for a neurosurgeon, Dr Balagi, to examine Mr Harte. Dr Tan never asked for more information, and hence, he never received all the details about the fall. Dr Balagi saw Mr Harte twice and diagnosed "syncope" fainting, which Dr Tan said explained in part why the plaintiff did not feel pain in his scrotum area.

214. As I have said above, even if full details of the fall were provided, a reasonably careful and competent urologist might not visualise or realise that the testes could be contused by a fall in an unconscious state from a toilet seat. Being such an unusual and rare occurrence, I accept Dr Tan's defence that atrophic damage to testes arising from a fall from a toilet seat is not a reasonably foreseeable consequence.

215. As such, I find that no negligence arises from Dr Tan's failure to examine Mr Harte before his discharge from the hospital. Whether the reasons proffered by Dr Tan for not seeing him are reasonable or not need not concern me. Neither is it relevant whether his message to the plaintiff to wait for him was in fact conveyed to Mr Harte. It makes no difference as Dr Tan would have discharged Mr Harte even if Dr Tan did examine him personally. I doubt that Dr Tan would have been able to diagnose a potentially dangerous intratesticular haematoma developing at that stage, especially when Mr Harte had not complained of any serious pain and there were no obvious signs of bruising or swelling.

216. Dr Myint Soe stressed that the 1st defendant is a FRCS (General Surgeon) as well as a specialist FRCS (Urology). He is the only one in Singapore with a FRCS (Urology) qualification. He had received extensive training in hospitals in England and in Singapore before he set up his own private practice. Varicocelectomy is a minor operation involving only day surgery. He has done far more difficult operations like kidney transplants. Furthermore, he has done about 200 varicocelectomies including those via the subinguinal approach. So far, he has never encountered any disasters of any kind. Neither has he made any blunders in any of his more difficult operations. Basically, to have blundered twice in two consecutive simple operations causing atrophy on both sides must be so remote as to be statistically improbable having regard to his experience and his numerous previous successful operations.

217. I consider this statistical argument to be a point in favour of Dr Tan that he could not have botched up the simple operation twice. What is statistically significant and hardly to be ignored is the very strange coincidence that the allegedly consecutive substandard operation is accompanied by a fall. Without anything else, immediate suspicion will be cast on the fall as being the more likely cause than the alleged negligence of a competent and very experienced urologist. This statistical analysis will be considered in more detail subsequently.

218. The 1st defendant testified that he used the recognised subinguinal approach, which allows access to the external veins. His method of surgery using optical lubes is recognised. New medical studies have advocated an aggressive approach to ligate all the veins leaving just one set of veins to the vas intact and there should be no adverse consequences such as venous congestion. Dr Tan opined that he was in fact much far more conservative as he had left many other veins intact during his operation. First, he took care to ensure that the vas and the bundle of vessels were identified and kept out of the surgical field after opening the cremasteric fascia. This ensured that some veins and the vasal artery were kept intact. He was trained to do this and he followed this standard procedure. I find no reason to disbelieve him on this. Besides leaving the vasal veins untouched, he also spared the gubernacular veins thereby reserving more than adequate channels for venous drainage. Since he did not deliver the testes out during the surgery, he had not ligated or cut any of the gubernacular veins, which were at the bottom end of the testes. On these aspects of his operation, Dr Tan was not shaken in cross-examination and had steadfastly maintained that he had left the vasal artery and vasal veins intact together with the vas.

219. The 1st defendant explained that in a lattice like network of varicocele veins, the tributaries of the same dilated vein could be cut twice, counting as 2 cuts, which gave the wrong impression that an unduly large number of veins were ligated. Lower down at the subinguinal level, the number of veins increases due to branching as one gets closer to the pampiniform plexus and the scrotum level, where for instance, for each internal spermatic vein encountered higher up at the inguinal canal level, two to four tributaries may be encountered at the lower level of the plexus, the lattice like network of intercommunicating veins. This accounts for the larger number of veins (subcutaneous veins, the external veins and the cremasteric veins) ligated normally lower down at the subinguinal level as compared to an operation higher up at the inguinal level.

220. Dr Schlegel agreed that the 1st defendant's surgical procedure described by him would generally fit the standard of care for treatment of a varicocele. I cannot find any objective evidence from which to infer that Dr Tan could not have done what he said he did. In the absence of such evidence, I have no reason to disbelieve his testimony on how he performed the operation.

221. Dr Schlegel and Dr Goldstein in an article 'Anatomical Approach to Varicocelectomy' described their preferred inguinal approach to varicocelectomy, where they said:

> …All external spermatic veins are identified and doubly ligated .. and divided. The gubernaculum is inspected for the presence of veins exiting from the tunica vaginalis. These are either cauterized, doubly ligated or clipped, and divided. …. All internal spermatic veins, even very small veins should be ligated. On completion of the dissection, the spermatic cord is run over the index finger to confirm that all veins have been ligated…… At least one set of blood vessels around the vas deferens should be preserved because they allow the only egress of blood from the testis.

222. From the above passage, it can be seen that very aggressive ligation of as many veins as possible (including very small veins) was advocated by Dr Schlegel and Dr Goldstein. Dr Goldstein in Chapter 19: "*Mini-Incision Microsurgical Inguinal or Subinguinal Varicocelectomy with Delivery of the Testis*" in his book "*Surgery of Male Infertility*" stated at page 177 that:

> The vas deferens is always accompanied by two sets of vessels. *As long as at least one set of vasal veins remains intact, venous congestion will not occur.*

223. The above statement is also repeated in the joint paper by Goldstein, Gilbert, Dicker, Dwosh and Gnecco in "*Microsurgical Inguinal Varicocelectomy with Delivery of the Testis: An Artery and Lymphatic Sparing Technique*".

224. In essence, leaving untouched only the vasal vein at the vas deferens is sufficient to prevent vascular damage to the testes. This evidence representing the views of a number of responsible urologists, later confirmed by Dr Schlegel in his testimony, is **fatal** to the plaintiff's postulation of excessive division of veins by Dr Tan as the cause of the devascularisation.

225. I accept that Dr Tan had left all the arteries including the vasal artery intact. He did not ligate and divide the vasal vein. He also left some other veins behind as shown in D29. I find that there was adequate blood supply to and sufficient venous drainage from both his testes. As such, Dr Tan's operation did not cause the devascularisation, which led to Mr Harte's substantial testicular atrophy.

226. Having failed in this direction, counsel for the plaintiff spent a considerable amount of time taking issue with all imaginable ways that Dr Tan might have misused the Babcock forceps, one of which was that Dr Tan might have applied too much pressure on the spermatic cord when he used it to pull it out of the skin incision and the other was clamping the arteries between the rounded 'teeth' of the Babcock forceps, just to cite two examples. In my opinion, the foray into this area was not fruitful.

227. Counsel for the plaintiff invited Dr Notley to give a multitude of examples on how the misuse of the forceps would lead to devascularisation: closing the ends on the blood vessel and damaging the blood vessels, twisting the forceps through 180 degrees together with the vessels within it, undue traction on the Babcock forceps obstructing the flow of blood through the vessels thereby causing thrombosis, which is a very likely cause of devascularisation. Without some supporting evidence, these countless possibilities remain in the speculative realm and have little weight. Speculation on all sorts of possibilities is not going to get the plaintiff very far.

228. Dr Schlegel initially also asserted that the clamping and pulling of the vas deferens and blood vessels with the Babcock forceps could have actually damaged those blood vessels. I then note that Dr Schlegel uses it as well. This is an atraumatic instrument with curved rounded edges. Dr Tan maintained that he had held the sides of the spermatic cord where there were no major arteries to bring it out to the skin level as was recommended by Marmar and Kim. He did not encircle the spermatic cord within the Babcock forceps. At worst, if he had applied too much pressure momentarily say for 10 seconds as he pulled out the spermatic cord, devascularisation could not possibly happen. A period of 10 seconds is far too short for any cell death to occur through lack of oxygenated blood flow. Just for the sake of comparison, the medical evidence before me was that salvage of the testes must be done within 6 hours if the blood supply is choked off due to a torsion injury, which necessarily implies that a temporary shut off of blood supply to the testis for any reason during the 1 hour operation is not likely to cause any cell death or testicular death through devascularisation.

229. I thus accept the submission of counsel for Dr Tan that even if he had occluded the blood supply, it could not have been for a sufficient length of time to cause vascular damage of the kind seen.

230. Dr Tan testified that he used the Babcock forceps to isolate and keep the vas and its vessels out of harm's way. If he had inadvertently kinked the vessels with the Babcock forceps, the effects would be obvious because the veins would swell up or get engorged. Hence, I would imagine that he would have remedied the obvious problem immediately. Even if his use of the Babcock forceps had damaged any vasal artery, there were two other sets of arteries, viz the testicular and the cremasteric arteries to supply the blood to the testicles. Atrophy would not likely occur.

231. I also note that the plaintiff's expert witness on biomechanics, A/Prof Lim Chwee Teck had produced a document P54 from 'Data Book on Mechanical Properties of Living Cells, Tissues and Organs' verifying the degree to which arteries can be stretched. It appears from the yield strain data that arteries can tolerate stretching up to 34 %, which indicates that they are fairly flexible and can safely withstand a fair amount of stretching. This proves that arterial damage is unlikely when the arteries are a little stretched in the process of bringing the spermatic cord to the skin level. It is fanciful to think that these arteries are so extremely delicate when they are not. Fortunately, Mother Nature has provided a certain level of inbuilt tolerance and robustness in these arteries because they have to transport blood under pressure to all parts of the human body, and are subject to movements and stretching when a human being runs and exercises.

Defence of 2nd defendants

232. Mr Harte had of his own volition chosen to consult Dr Tan for his fertility treatment and varicocele operation.

233. The 2nd defendants' defence was that Dr Tan was not their employee, servant or agent. The 2nd defendants and Dr Tan never represented to the plaintiff that Dr Tan was ever an employee, servant or agent of the 2nd defendants. Dr Tan has his own private clinic. In legal parlance, Dr Tan is an independent contractor. He is merely accredited to use the 2nd defendants' hospital facilities and has been granted admitting privileges for his patients.

234. Mr Harte could not be mistaken that he was consulting a private specialist because the signboard outside Dr Tan's clinic would have clearly shown that his clinic was not part of the hospital establishment at all. Official receipts given to the plaintiff showed that they were issued by H H Tan Urology Centre Pte Ltd of #04-03 Gleneagles Medical Centre Singapore 258499.

235. Similarly, when he went to Sheng Yu Laboratory of #07-11 Specialists' Centre for his basic seminal analysis, for which he was separately issued a receipt, it must be clear to Mr Harte that the laboratory test was not done by the hospital but by a private agency.

236. Balanced against these were the direct payments to the hospital on the day of the surgery on admission. He paid an initial deposit of $300 to the hospital upon his admission for the surgery. He paid a total of $4,803.68 again to the hospital when he was discharged after the surgery. Both receipts represented that he was paying to 'Gleneagles Hospital Singapore, 6A Napier Road Singapore 258500'.

237. There was no breach of contract between the plaintiff and the 2nd defendants. The 2nd defendants' contract with the plaintiff was contained in the 2nd defendants' "Conditions of Service/Hospital Policies" dated 28 April 1997, which was for the provision by the 2nd defendants of hospitalisation services to the plaintiff in consideration of the plaintiff agreeing to pay the 2nd defendants all fees and expenses for the same. There was no allegation of any breach of this contract by the 2nd defendants.

238. There was no battery or trespass on the person of Mr Harte as all treatment, care and attention administered to him during his stay in the hospital was undertaken with his consent.

239. The 2nd defendants maintained that they were not negligent with regard to the fall in the toilet. There was also no allegation by the plaintiff that the fall was caused by the 2nd defendants' negligence. Having regard to the undisputed fact that it was Mr Harte who had, against the nurse's advice, insisted on going to the toilet, Mr Harte must accept full responsibility for his own fall whilst seated on the toilet bowl. If he did suffer any contusion of his testes as a result of the fall, he should not blame others for it. He could not say that the contusion was caused by the negligence of the hospital. I entirely agree.

The Law

240. There is no dispute as to what the law is in relation to the standard of care required in the area of medical negligence as it is fairly well settled. Judith Prakash J in a recent decision in *Jason Carlos Rancisco v Dr L. M. Thng & Anor* (Suit No 573 of 1998) had succinctly and clearly set out the law and I can do no better other than to quote paragraphs 106 and 107 of the learned judge's decision, which I most respectfully adopt:

> 106. The duty of the medical practitioner is to exercise reasonable skill and care in treating his patient and the burden lies on the plaintiff to satisfy the court that the defendant was in breach of his duty. Reasonable skill and care are assessed on the basis of the standard of the ordinary skilled man exercising and professing to have that special skill and not on the standard of the highest expert. This is what is known as the 'Bolam test' taken from the direction to the

jury of Mc Nair J in *Bolam v Friern Hospital Management Committee* [1957] 1 WLR 582. This test has been restated many times and has been approved by the House of Lords in cases like *White House v Jordan* [1981] 1 All ER 267 and *Sidaway v Board of Governors of the Bethlem Royal Hospital* [1985] 1 All ER 643.

107. The standard of care has been held to be a matter of medical judgment in *Sidaway* where Lord Scarman stated:

> 'The *Bolam* principle may be formulated as a rule but a doctor is not negligent if he acts in accordance with a practice accepted at the time as proper by a responsible body of medical opinion even though other doctors adopt a different practice. In short the law imposes a duty of care; but the standard of care is a matter of medical judgment'.

241. The learned judge also accepted the following formulation of the law by Lord Scarman in *Maynard v West Midlands Regional Health Authority* [1984] 1 WLR 634 at p 638-639:

> A case which is based on an allegation that a fully considered decision of two consultants in the field of their special skill was negligent clearly presents certain difficulties of proof. It is not enough to show that there is a body of competent professional opinion which considers that theirs was a wrong decision, if there also exists a body of professional opinion, equally competent, which supports the decision as reasonable in the circumstances. It is not enough to show that subsequent events show that the operation need never have been performed, if at the time the decision to operate was taken it was reasonable in the sense that a responsible body of medical opinion would have accepted it as proper …
>
> I would only add that a doctor who professes to exercise a special skill must exercise the ordinary skill of his speciality. Differences of opinion and practice exist, and will always exist, in the medical as in other professions. There is seldom any one answer exclusive of all others to problems of professional judgment. A court may prefer one body of opinion to the other: but that is no basis for a conclusion of negligence.
>
> …..
>
> ….I have to say that a judge's 'preference' for one body of distinguished professional opinion to another also professionally distinguished is not sufficient to establish negligence in a practitioner whose actions have received the seal of approval of those whose opinions, truthfully expressed, honestly held, were not preferred. …in the realm of diagnosis and treatment negligence is not established by preferring one respectable body of professional opinion to another. Failure to exercise the ordinary skill of a doctor (in the appropriate speciality, if he be a specialist) is necessary.'

242. Judith Prakash J. emphasized that the judge has to exercise his own critical faculties and not simply be swept along by the opinion of the medical experts. I agree with her completely as can be seen later in this case, I found myself unfortunately in complete disagreement with the medical experts called both by the plaintiff and the 1st defendant, particularly in relation to their expert opinion that the colour of a scrotal haematoma from physical trauma is necessarily 'as black as hair', 'black as a gown' or 'blue-black'.

243. Just as with the learned judge, I accept the authoritative proposition of law stated by Lord Browne-Wilkinson in *Bolitho v. City and Hackney H.A.* (H.L.(E.)) [1998] A.C.232, which clarifies the ambit of the *Bolam* test:

> ….in my view, the court is not bound to hold that a defendant doctor escapes liability for negligent treatment or diagnosis just because he leads evidence from a number of medical experts who are genuinely of opinion that the defendant's treatment or diagnosis accorded with sound medical practice.
>
> ……
>
> ….in cases of diagnosis and treatment there are cases where, despite a body of professional opinion sanctioning the defendant's conduct, the defendant can properly be held liable for negligence (I am not here considering questions of disclosure of risk). In my judgment that is because, in some cases, it cannot be demonstrated to the judge's satisfaction that the body of opinion relied upon is reasonable or responsible. In the vast majority of cases the fact that distinguished experts in the field are of a particular opinion will demonstrate the reasonableness of that opinion. In particular, where there are questions of assessment of the relative risks and benefits of adopting a particular medical practice, a reasonable view necessarily presupposes that the relative risks and benefits have been weighed by the experts in forming their opinions. But if, in a rare case, it can be demonstrated that the professional opinion is not capable of withstanding logical analysis, the judge is entitled to hold that the body of opinion is not reasonable or responsible.
>
> I emphasise that in my view it will very seldom be right for a judge to reach the conclusion that views genuinely held by a competent medical expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which a judge would not normally be able to make without expert evidence. As the quotation from Lord Scarman makes clear, it would be wrong to allow such assessment to deteriorate into seeking to persuade the judge to prefer one of two views both of which are capable of being logically supported. It is only where a judge can be satisfied that the body of expert opinion cannot be logically supported at all that such opinion will not provide the benchmark by reference to which the defendant's conduct falls to be assessed.

244. I applied the above test not only in relation to Dr Tan's medical advice to Mr Harte (as seen earlier), but also to Dr Tan's medical diagnosis of Mr Harte's varicocele condition, all aspects of his surgery and, not forgetting the no less important area, his post-operative patient care.

245. To prove that Dr Tan was negligent in his surgery, the plaintiff relied on the doctrine of *res ipsa loquitur*. His counsel referred me to the following passages from Halsbury's Laws of England, Vol 33: Negligence, para 664 –668:

> Under the doctrine *res ipsa loquitur*, a plaintiff establishes a prima facie case of negligence where (1) it is not possible for him to prove precisely what was the relevant act or omission which set in train the events leading to the accident and (2) on the evidence as it stands at the relevant time it is more likely than not that the effective cause of the accident was some act or omission of the defendant or of someone for whom the defendant is responsible, which act or omission constitutes a failure to take proper care for the plaintiff's safety. There must be reasonable evidence of negligence. However, when the thing which

> causes the accident is shown to be under the management of the defendant or his employees, and the accident is such as in the ordinary course of things does not happen if those who have the management use proper care, if affords reasonable evidence, in the absence of explanation by the defendant, that the accident arose from want of care.
>
> ..
>
> Even though the matter is not one of common experience, and where an unexplained accident occurs from a thing under the defendant's control, and medical or other expert evidence shows that such accidents would not happen if proper care were used, there is at least evidence of negligence for the court.
>
> ....
>
> Where the plaintiff successfully alleges *res ipsa locquitur* its effect is to furnish evidence of negligence on which a court is free to find for the plaintiff.

246. I do not think that the above maxim has any application here. The plaintiff's fall in the toilet unfortunately has crept into the equation and is capable *per se* of causing bilateral atrophy. The fall is something beyond the management and control of the 1st defendant. It is not for the 1st defendant to prove that the fall caused the damage. So long he shows that there are other probable causes (other than his surgery) for the atrophy, without proving which is in fact responsible for the damage, the principle of *res ipsa loquitur* can no longer help the plaintiff, who always has the legal burden to prove that Dr Tan had breached his duty of care towards him.

247. At this juncture, it bears remembering that an allegation of clinical negligence stemming from incompetence or malpractice amounts to an attack on the professional standing and reputation of a doctor. Hence, the charge of negligence against that doctor is necessarily of a serious nature. The standard of proof required for matters of such gravity has been laid down by the Court of Appeal decision in *Hucks v Cole* (1968) 112 Sol Jo 483 as follows:

> A charge of professional negligence against a medical man was a serious charge, on a different footing to a charge of negligence against a motorist or an employer. The reason is because the consequences for the professional man are far more grave. A finding of negligence affects his standing and reputation. It impairs the confidence which his clients have in him. The burden of proof is correspondingly greater. The principle applies that: 'In proportion as the charge is grave, so ought the proof to be clear': see *Hornal v Neubergger Products Ltd* [1957] 1 QB 247.
>
> Another difference lies in the fact that, with the best will in the world, things do sometimes go amiss in surgical operations or medical treatment. A striking illustration was *Roe v Minister of Health* [1954] 2 QB 66. So a doctor is not to be held negligent simply because something goes wrong. It is not right to invoke against him the maxim *res ipsa loquitur* save in an extreme case. He is not liable for mischance or misadventure. Nor is he liable for an error of judgment. He is not liable for choosing one course out of two which may be open to him, or for following one school of thought rather than another. He is liable only if he falls below the standard of a reasonably competent practitioner in his field – so much so that his conduct may fairly be held to be – I will not say deserving of censure, but, at any rate, inexcusable.

248. However, I think the above test laid down in *Hucks v Cole* does not apply to the initial fact-finding, or the proof of causal factors or causation, which ordinarily remains on the civil test of a balance of probability in any case. After establishing the causal factors, there is still the next step of proving the breach of the doctor's duty of care, which is the foundation of the charge against the doctor for his negligence. For this next step, I believe the principle stated in *Huck v Cole* must be kept in view: '*In proportion as the charge is grave, so ought* ***the proof to be clear****.'* Therefore the fact that something has gone wrong during surgery is not *per se* indicative of negligence. The mere fact of a complication occurring does not necessarily mean that the surgeon has fallen below the accepted standard of care of a reasonably competent surgeon. The question always has to be whether the proven facts are reasonably capable of an inference of negligence, where there is no direct evidence of negligence.

My Analysis

249. To begin with, it is wrong to think that both testes of the plaintiff have been completely infarcted or are now made up of dead tissue only. Dr Notley himself said that the biopsies done some months after the surgery showed that parts of the testes survived. He had no doubt that other parts of the testes had also survived up to a point. Dr Schlegel even said that Mr Harte's testes were probably one of the best in a man who has non-obstructive azoospermia with reference to the results of the testicular biopsy done on 21 October 1998. This clearly is a case of partial atrophy.

(a) Infection as a cause of atrophy

250. Subclinical infection (especially if it is inside the testis) is possible as infection is always a possibility after any surgery. Infection causing testicular failure can be a contributory cause. A swollen scrotum the size of a grapefruit can be the result of infection. But A/Prof Foo did not think that infection could cause testicular atrophy unless it was a very bad infection, in which case there would be pus and fever and the rest of the symptoms. Dr Jimmy Beng also concluded that the 'hyalinization' found was inconsistent with an infection. With no evidence of pus and fever and with evidence of 'hyalinization', I can safely rule out infection as a possible cause of the atrophy.

(b) Was the Scrotal approach adopted?

251. Counsel for the plaintiff submitted that Dr Tan had essentially used the scrotal approach in his operation on Mr Harte.

252. The old-fashioned scrotal approach normally involved incision at the scrotum itself. Dr Notley condemned the scrotal approach as being dangerous because it produced a high incidence of testicular infarction. Those using the scrotal approach would operate and ligate at the level of the pampiniform plexus network of veins before they coalesce into the vasal veins and other sets of veins. The surgeon might not know which were the important veins to leave behind and the minimum number that ought to be left behind unligated. Thus, the vasal veins and tributaries at the pampiniform plexus were likely to be divided as well. According to Dr Notley, the scrotal approach had since been abandoned because of these reasons.

253. However, Dr Notley's explanation of the dangers of the scrotal approach did not accord with the reasons given at page 1364 of 'Campbell's Urology' 7th Edition where it stated that:

> In the early 1900s, an open scrotal approach was employed, involving the mass ligation and excision of the varicosed plexus of veins. At the level of the scrotum, however, the pampiniform plexus of veins is intimately entwined with the coiled testicular artery. Therefore, scrotal operations are to be avoided because damage to the arterial supply of the testis frequently results in

testicular atrophy and further impairment of spermatogenesis and fertility.

254. I was thus given the wrong impression by Dr Notley that overligation of veins was the cause of the frequent incidence of atrophy in such scrotal operations when in fact it was the severance or damage of the arteries entwined with the veins ligated and excised at the scrotal level that led to the problem.

255. In any event, I find no evidence in this case that Dr Tan operated at the scrotum level using this old-fashioned method. Photographs P25 to 28 show the location of the incision at both sides, which were over the external inguinal ring. He did not deliver the testis out of the wound in both operations. He delivered up part of the spermatic cord to enable him to proceed with the vein ligation and division at the level consistent with the subinguinal approach. I find no evidence that the vein ligations and divisions were at the level of the pampiniform plexus network of veins. I believe there would be sufficient spermatic cord length above the pampiniform plexus network of veins for him to retrieve out of the skin incisions to perform the subinguinal varicocelectomy.

256. The fact that Dr Tan ligated a total of 11 and 7 veins on the right side and left side respectively does not indicate that he must have ligated at the pampiniform plexus. A/Prof Li said that at the subinguinal area, one might find on the average 15 to 20 internal veins already, and perhaps 5 or 6 external veins. There would also be some cremasteric veins. The medical literature tendered also recognises that there are more veins encountered with the subinguinal approach. So the total number of veins ligated by Dr Tan is not, in my view, inconsistent with an operation at the subinguinal level.

257. Neither were the two skin incisions too high or too low that it was not possible for Dr Tan to do a subinguinal surgery. Upon being shown the photographs of the skin incisions on Mr Harte, A/Prof Li said that with those skin cuts, one would definitely be able to do a subinguinal surgery because the skin was mobile and the patient was relatively fat. The skin incision could be easily moved to the area where the actual operation was intended. The operation in the deeper areas of the body need not be directly at the location where the skin incision had been made.

258. No evidence is led by the plaintiff to show that Dr Tan's skin incision at the level for Mr Harte is something that a reasonably competent urologist will not do for a subinguinal variocelectomy.

(c) The operation

259. In this case, there is no clear evidence proving that Dr Tan was negligent in his operation. As counsel for the 1st defendant rightly pointed out, there is no concrete evidence like a swab or an instrument left behind. The alleged negligence is based wholly on an inference that Dr Tan must have botched up the operation because atrophy has occurred.

260. The main theory advanced by counsel for the plaintiff initially was that Dr Tan had tied up too many veins. That theory was demolished by Dr Schlegel, the plaintiff's own witness. Dr Schlegel agreed that if Dr Tan had, at the level of the subinguinal surgery, cut 11 veins on the right side, and 7 on the left, leaving the vasal vein intact and with no inadvertent damage to other veins or arteries, then there would not be any vascular damage to the testes.

261. In fact, Dr Schlegel had in his professional papers suggested an even more aggressive varicocelectomy method where nearly all the veins except for the vasal veins are ligated. Dr Notley also stated that '*modern operations designed to excise all the spermatic veins, both internal and external, and even the gubernacular veins rarely create this disastrous complication [i.e. testicular infarction]. The crucial veins to be spared are those accompanying the vas.*' Since I accept Dr Tan's evidence that he had not ligated the vasal veins, it is thus most unlikely that he had infarcted Mr Harte's testicles.

262. Even if it were true that keeping just the set of vasal veins turned out to be inadequate for venous drainage in Mr Harte's case, and the testicles consequently infarcted leading to atrophy, I still cannot regard Dr Tan as having been negligent because operating in that manner is recognised by eminent and reputable urologists such as Dr Schlegel and Dr Goldstein, including Dr

Notley, as an acceptable practice. I would regard their views as representing a responsible body of professional opinion. I cannot see how Dr Tan could be said to have breached his duty of care to his patient by using such a widely recognised and accepted technique. A/Prof Li also said that the standard practice is to ligate as many veins as possible but leave the vasal veins intact. His is the view of yet another equally eminent and distinguished urologist.

263. Counsel for the plaintiff went on in his submission to analyse the countless ways in which Dr Tan could have misused the Babcock forceps and damaged the arteries or the vasal veins. There is also the added allegation of insufficient optical magnification by Dr Tan, implying that he did not see very well during his operation. As I have said before, they remain as theoretical possibilities, which do not carry much weight without some substantive evidence.

264. Mr Lek, counsel for the 2nd defendants, made a valiant attempt in his submission to explain the unusual, consecutive negligence of Dr Tan for the varicocelectomy operation on each side as follows:

> (a) Dr Tan made the skin incision at the same level as Dr Dubin who used the inguinal method. As Dr Tan opted for the subinguinal approach, he had to reach quite deep down (4 to 5 cm) in the case of the obese plaintiff in order to reach and pull out the spermatic cord.
>
> (b) A/Prof Li had said that it would be more difficult to deliver up the spermatic cord for a fat man and the surgeon would have to dig deeper to get to the cord.
>
> (c) At times, there might not be enough cord length to bring the whole spermatic cord up to the surface or the skin level and in that situation the surgery would have to be done within the wound.
>
> (d) In the process either of operating on the cord within the wound or in attempting the difficult procedure of delivering the spermatic cord out of the wound, he injured the testicular arteries, which led to ischaemic injury to the testicles.

265. Mr Lek thus submitted that since Dr Tan used the same technique on both sides, that was why testicular artery damage occurred on both sides resulting in atrophy in both testicles at the same time.

266. On closer examination of A/Prof Li's evidence, I find that the greater difficulty of retrieval of the spermatic cord was said in the context of a fat man versus a thinner man and the depth of retrieval. He did not say that there was real difficulty *per se*. On the contrary, A/Prof Li testified that actually there is no difficulty in pushing up the testis, through the inguinal area to deliver it out of the wound as the testis is mobile and there are no muscles in the way of the delivery. It is not at all traumatic on the spermatic cord to pull the whole testis out. However, the operation takes more time. Photographs in D44 show a varicocele operation where the entire testicle with part of its spermatic cord had been delivered completely out of the wound. Photographs in D43 show a typical varicocele operation on the spermatic cord where the testicle was not delivered out of the wound. A/Prof Li confirmed that there is also no difficulty pushing up the testis halfway without actually delivering the testis, in order to expose just the part of the spermatic cord that the surgeon intends to perform his varicocelectomy. Thus, the premise of Mr Lek's submission is not sound.

267. Mr Lek in his cross-examination elicited from A/Prof Li numerous possibilities on how accidental testicular artery injury can occur: clamping it, tying it, twisting it, pinching it, or burning it during diathermy. A/Prof Li accepted that these are the usual risks of surgery.

268. In his submissions, Mr Lek then referred me to the following articles to emphasize the importance of safeguarding the testicular artery, damage of which can cause atrophy:

(1) **Surgery of Male Infertility by Marc Goldstein, Chapter 18 - Varicocelectomy: General Considerations** - The incidence of testicular artery injury after inguinal varicocelectomy is unknown. Case reports, however, suggest that this complication may be more common than realized. It can result in testicular atrophy and, if the operation is performed bilaterally, azoospermia may ensue in a previously oligospermic man. Furthermore, Starzl and his transplant group reported a 14 per cent incidence of testicular atrophy and a 70 per cent incidence of testicular atrophy and a 70 percent incidence of hydrocele formation when the spermatic cord was divided and the vas and vasal vessels were preserved. (My Comment: In Mr Harte's case, there was no division of the spermatic cord, unlike the cases cited by Starzl which involved severance of the other arteries at the time of division of the whole spermatic cord where these arteries run alongside.)

(2) **Testicular Artery Ramification Within The Inguinal Canal by Jarow, Ogle, Kaspar and Hopkins -** Testicular arterial anatomy has great clinical importance due to the application of this knowledge to testicular surgery. The capability of collateral arterial blood supply through the deferential and cremasteric arteries to support the testis has been used in patients who require transection of the main testicular vessels during orchiopexy for crytorchidism. However, in some cases this collateral blood supply does not appear to be sufficient to maintain the testis and atrophy ensues. (My Comment: Again, this is not applicable because there is an intentional division by transection of the testicular artery during the operation, which did not take place in our case. Dr Tan had not cut any of the arteries during his operation.)

(3) **Microsurgical Inguinal Varicocelectomy with delivery of the Testis: An Artery and Lymphatic Sparing Technique -** Testicular atrophy and azoospermia following varicocelectomy are risks associated with ligation of the testicular artery. Preservation of the testicular artery is particularly critical when operating upon a solitary testis, or in children and adolescents on whom the operation is being performed prophylactically. (My Comment: This is also inapplicable because the article refers to ligation of the testicular artery, which did not happen here.)

(4) **Anatomical Approach to Varicocelcctomy: Schlegel and Goldstein -** Inadvertent injury to a testicular artery is easily possible with inadequate care or optical magnification.

269. In my view, Mr Lek tried to elevate the consequences of inadvertent injury to the testicular artery to that of a **deliberate severance (i.e. total damage)** of a testicular artery as a part of the operation, where the risk of atrophy would naturally be higher. Mr Lek appeared to be suggesting that Dr Tan accidentally damaged the testicular artery on both sides so severely that it amounted to a complete severance of a testicular artery first on one side followed later by the other side because of insufficient optical magnification. To my mind, this is not only remote but also highly speculative. Am I to believe that Dr Tan was operating almost blindfolded figuratively speaking, or he was so very heavy handed during both operations, tugging at the arteries more than he should, thereby causing severe traction injury to the arteries? I do not.

270. Whilst on this point, I tend to agree with A/Prof Foo that the traction force when using the Babcock forceps to grip and pull up the spermatic cord through the skin incision is not that rough and severe to cause injury to the artery. A/Prof Foo explained that the Babcock instrument is constructed in such a way that it does not cause severe injury to the tissue generally unless the surgeon is very rough or heavy handed and does things without care.

271. For the purpose of analysis, let me assume the worse that Dr Tan, despite all the care taken, had accidentally damaged the testicular artery fairly severely. Impairment of blood flow would then result. But I have to remind myself that there are three arteries (i.e. internal spermatic artery, artery of the vas deferens and cremasteric artery) supplying blood to each testicle. According to Dr Schlegel, the testicle will survive and in fact function pretty well where there is damage to just one artery. Exhibit D 13 confirms what Dr Schlegel said. The diagram there shows the free anastomosis between the three arteries supplying each testis. To accidentally and severely damage all three arteries which are not co-located together at one place on each testicle (i.e. making a total of six arteries to be damaged) to get to the disastrous stage of bilateral atrophy seems highly improbable.

272. This is also borne out by Dr Notley's observation that it is quite difficult to infarct the testicle because of its profuse arterial supply and the profuse anastomosis.

273. Furthermore, if the arteries or the veins were ligated, A/Prof Foo explained that there should not be any scrotal haematoma. I agree. A/Prof Foo's evidence makes logical sense because in that situation, there would be no blood flowing into the organ to create any haematoma, whether intratesticular or extratesticular. Whatever theory postulated must fit the facts. When it does not as in this case, the theory must be discarded. Since there was an intratesticular haematoma, the likelihood of inadvertent ligation of all the arteries or arterial damage resulting in their complete blockage has to be ruled out.

274. The CDUS result in September 1997 showing perfusion indicated that the arteries were still functioning to some extent, as there was still blood flow. This clearly dispels the hypothesis of inadvertent ligation or severe damage and blockage of the arteries.

(d) The operation technique used

275. In my judgment, the operation technique used by Dr Tan cannot be faulted. The subinguinal approach is recognised and supported by a number of medical papers tendered, which I find it unnecessary to enumerate. Dr Schlegel had agreed that if Dr Tan had done what he said he did, there would be no atrophy. Dr Schlegel accepted that the procedure used by Dr Tan as described in his affidavit generally fits the standard of care for treatment of a varicocele. Dr Schlegel did not say that the optical magnification used by Dr Tan during his surgery was insufficient or that too many veins were ligated.

276. All the experts agree that bilateral testicular atrophy after varicocelectomy is unheard of. Atrophy on one side has been reported but is extremely rare.

277. There is no credible evidence produced to persuade me that Dr Tan's surgery for this simple operation fell below the standard of care expected of any responsible and competent urologist surgeon.

(e) Traction injury

278. Dr Tan opined that Mr Harte suffered both contusion and traction injuries, the latter being more serious. In a varicocelectomy operation, the area of the spermatic cord cut open is mechanically weakened. When sudden traction force is applied, the resulting damage is thus even more severe than what it would otherwise be. See illustrations D 19, 22 and 23 by Dr Tan. His explanation for the mechanism of the traction injury was as follows:

> (a) The small veins external to the testicle will tear. Blood will leak into the scrotum and cause an extratesticular or scrotal haematoma and not an intratesticular haematoma. The rate of scrotal swelling depends on the size and number of veins damaged. The swelling may take up to a day in Mr Harte's case to be noticeable. A/Prof Foo also believes that traction injury will only cause an

extratesticular haematoma.

(b) Although arteries are mechanically stronger than veins, nevertheless excessive stretching can still tear the fragile inner wall lining of the arteries. The tearing causes spasm and contraction of the artery. Blood clots build up at the tear region, narrowing the arteries. Eventually that leads to thrombosis at the choke points. As blood flow slows down, the testes become ischaemic. If the blood supply is completely cut off, the testicle will devascularise but it will not lead to any swelling or intratesticular haematoma.

279. However, Dr Notley had not heard of a traction injury. He had not seen one. He did not think that it was recorded in the medical literature.

280. Dr Schlegel too had not come across a traction injury to the spermatic cord vessels during a varicocele surgery. In his opinion, as long as the muscles were around the spermatic cord, using the Babcock forceps to pull out the spermatic cord was not likely to cause enough injury to the blood vessels to damage them. After the blood vessels were separated from the muscles, then the exposed vessels would become vulnerable to avulsion or traction injuries.

281. A/Prof Foo also said that he was not aware of anything in the medical literature that supported a proposition that a traction injury to the spermatic cord would lead to eventual testicular atrophy. A/Prof Foo explained that an intratesticular haematoma caused by crushing of the testicles might be aggravated by traction injury to the arteries, which could at the same time compromise the blood supply to the testicles. Thus, the traction injury could contribute to the atrophy of the testes. However, in his opinion, the crushing injury was a more significant contributory than the traction injury.

282. A/Prof Li felt that one would need to pull extremely hard on the testes before there could be any traction injury to the spermatic cord. Not having come across this, he did not appear enamoured by this possibility. I share his sentiments.

283. Based on the opinions of the experts, I come to the conclusion that traction injury is not likely to cause any rupture of the capillary vessels within the testicular tissue and hence, internal intratesticular haemorrhage from traction injury is improbable. With continuous stretching according to A/Prof Foo, the internal spermatic fascia will be the first to give way, followed by the veins and lymphatics, then the arteries, the vas deferens and lastly the nerves (which are all within the spermatic cord). This shows that the arteries are much stronger than the internal spermatic fascia, veins and lymphatics. Hence, it is not likely for the arteries to give way so readily.

284. It is of course possible that a very large traction force can still sever or damage all 3 arteries to the testes on each side to cause bilateral atrophy through devascularisation. But I think that possibility is remote. The blood flowing out **under pressure** from the ruptured arteries will quickly distend the scrotum extratesticularly, thereby creating a big haematoma, which with time must cause some discolouration on his swollen scrotum. Mr Harte should perhaps be having a fairly dark coloured bluish black or reddish purplish scrotum with significant swelling by the time he left the hospital. But these were not the symptoms observed. There was no significant discolouration and/or swelling to move Mr Harte to raise an alarm before his discharge at 7.00 p.m. Neither was there evidence that the internal spermatic fascia was ripped or that the lymphatics were torn resulting in a substantial hydrocele formation. I thus rule out traction injury to the arteries as a cause of the devascularisation of the testes.

(f) Contusion injury

285. The plaintiff's main contentions against the contusion theory were:

(a) the absence of abrasion and bruising of the scrotal skin and penis;

(b) the absence of pain and swelling before discharge from the hospital;

(c) the absence of a black coloured scrotum;

(d) the presence of a bright red swollen scrotum turning to a reddish purplish colour on the morning after the fall;

(e) the extraordinary force needed to contuse the testes before one can get bilateral testicular atrophy.

286. Before dealing with the above contentions, I will closely examine how the mechanism of contusion can result in pain, scrotal swelling, intratesticular haematoma and finally atrophy.

i. Intratesticular haematoma and atrophy

287. With his schematic diagrams D18, D20 and D21, Dr Tan illustrated how the contusion injury could have occurred.

288. Dr Tan testified that contusion injury without rupture of testicles would lead to oedema (a condition characterized by an excess of watery fluid collecting in the cavities or tissues of the body), and also internal haemorrhage of the small capillary vessels within the testes. The accumulation of bleeding from the capillary vessels causes swelling, expansion and rise in pressure. An intratesticular haematoma forms within the tunica albuginea, which results in raised intratesticular pressure thereby shutting off the microcirculation. See illustrations D 9 and D 15 by Dr Tan. The rate at which oedema and swelling occur depends on how severe the contusion injury is and on how rapidly the blood flows into the testicles. I agree.

289. The post-operative ultrasound scan of the testicles showed micro-calcifications and a patchy appearance, which were not present before and could be due to the intratesticular haemorrhage or intratesticular haematoma. The presence of perfusion suggested that the arterial supply to the testes was not compromised.

290. A/Prof Foo was referred to an article: *Blunt Scrotal Trauma: Emergency Department Evaluation and Management by Munter and Faleski*. He was asked whether he would agree with the fact that a contusion without a testicular rupture seldom leads to morbidity. A/Prof Foo said that there were various degrees of contusion from mild to severe. But a mild one alone would not cause significant damage to the testes but a severe one combined with other factors would. He emphasized that in medicine one usually faces multiple causes and effects contributing to a final result. There is not just one factor to explain a phenomenon. I have to agree wholeheartedly. In his opinion, the severe contusion had contributed to the testicular atrophy.

291. A/Prof Foo testified that compressing a taut testis due to venous congestion or pressure buildup after the varicocele surgery would cause more damage. Intratesticular haematoma can also result from pressure damage to the testicular tissue within the testis itself.

292. In my opinion, the fact that the testicles became more congested after the operation due to the ligation of several varicocele veins was certainly a contributory factor. I fully agree that squeezing a congested taut testicle as opposed to an uncongested one is much more likely to cause internal microvascular and microstructural damage to the testicular tissue and capillary blood vessels. It is nothing more than pure common sense.

293. Apart from such direct microvascular and microstructural testicular tissue damage due to contusion, I am of the opinion that subsequent development of a significant haematoma, particularly an intratesticular haematoma, can also lead to atrophy due to the buildup of pressure that may compress the veins to an extent that the venous outflow of the testes becomes seriously impaired, and correspondingly the venous inflow too. I find support for this in P29: Clinical Negligence by Powers and Harris 3rd Edn at para 41.98 at p 1388.

ii. Pain

294. I generally accept the following explanation by A/Prof Foo on the mechanism of pain and the pain symptoms resulting from intratesticular haemorrhage:

> A: …let's assume that …bleeding starts in the testes but the bleeding is slow, you know, it accumulates slowly and at that point in time, within a few hours, they may not have any severe pain but only subsequently, it can cause serious damage to the testicles. It's the same in the head. We have seen patients with head injury, just a knock and then they fainted, so call, and then when they get up, they look very well. But sometimes they go to the A & E, they still look well but when they were sent home, you know, he can become unconscious and can even die from that because at the point of impact, bleeding occurs inside. But when the bleeding is minimal, it will not cause pressure on the brain but after a few hours, when it caused more pressure, patient becomes unconscious. It is a similar analogy in the testicles which the tissues inside cannot expand. So, initially, we might think that it is a minor injury. Even, you know, after a few days, it may not be detected. But only when it starts to manifest, you know, when patient becomes unconscious, then a scan or what may suspect there is intra-cranial haematoma. And it needs urgent operation to drain the haematoma to save the brain; similarly to the testes.
>
> His Honour: So are you saying that the pain comes along when the tunica is stretched?
>
> Witness: Yes, so initially there may not—
>
> His Honour: So if it is not stretched ---
>
> Witness: ---be much pain.
>
> His Honour: --but there is bleeding?
>
> Witness: Yes, if there is bleeding, it may not have much pain at that time.
>
> His Honour: It may not have pain. So the nerve detects the stretching, is it, or what?
>
> Witness: The nerve may—
>
> His Honour: How does the nerve mechanism and the pain mechanism work?
>
> Witness: The stretched nerve may numb the part a bit. So at the point of impact, he may not have the pain and subsequently, the pain may be due to stretching of the tunica covering the testicle. But this will take a few hours to develop. So when the patient goes home at that time, you know, the increased pressure in the testes may not be that great to cause him a lot of pain. But, as he himself said, after he went home, he started to develop more and more pain, and tried to contact Dr Tan and Dr Tan somehow he doesn't get the message

and—this can explain the whole theory.

Q: Doctor, this—what you've just described, is it consistent with the traction or contusion injury?

A: As I said, both, you see. The traction injury will injure the lymphatics, the veins, and possibly the artery and then the nerves. And that will explain the extratesticular haematoma. And that may explain why at the point of impact, you know, because of the nerve is injured, he may not feel severe pain at the point of impact. Now, the contusion injury within the testicle will not cause pain initially but it will cause pain subsequently, after he goes home. As the pressure builds up within the testicle, he will feel the pain.

295. A/Prof Foo agreed that pain generally increases with the size of the testicular swelling. But if the patient has continuous pain, the patient may become desensitised to it after a while. The nerves become slightly numb. The pain felt may be less but this varies from patient to patient.

296. I reiterate that the pain arising from moderate contusion may well be confused with the dull pain from the normal congestion that Mr Harte experienced in his previous varicocelectomy operation. The pain from the surgery itself may mask the pain arising from microvascular damage due to contusion. For all the reasons given earlier, I do not believe that Mr Harte could readily distinguish between them. I am therefore not surprised that he did not complain of any pain, although he experienced some swelling, discomfort and dull pain in his scrotum when he was leaving the hospital.

iii. Swelling – extratesticular and intratesticular

297. A/Prof Foo testified that if only one of the three arteries to the testis was ligated or one artery was damaged, the swelling would take longer to develop. Should there be a complete shutdown of blood supply due to ligation of all arteries, atrophy would occur but there would not be any swelling because there could never be any venous congestion without blood inflow.

298. However, if the veins leaving the testes were tied up instead, there would be venous congestion causing increased pressure and swelling in the testes. Should the tunica be stretched to its maximum, no more blood would ever flow in. This would similarly cause atrophy due to shutting down of the blood supply. Therefore, the rate of intratesticular swelling depends on the number of veins and/or arteries ligated.

299. As for extratesticular (i.e. scrotal) swelling, A/Prof Foo alluded to this other possibility of blood and fluid collecting in the extratesticular region. He explained the source of this blood and fluid as follows:

Witness: From the---if there is traction injury, the bleeding, that is, the lymphatic will be torn, the vein will be torn, and the bleeding from the traction injury damaging the lymphatic and vein will leak around the testicle. The crushing injury will cause bleeding inside the testicle, so you can expect both extratesticular haematoma as well as intratesticular haematoma.

300. A/Prof Foo agreed that swelling could also result from an infarcted testicle, howsoever caused (for instance by devascularisation, venous congestion, blunt trauma or torsion):

Q: .. Doctor, if a testicle is infarcted, OK, would there be swelling?

A: Depends on the time sequence. Initially, there may be swelling but

subsequently, you know, after a few weeks, a few months, it becomes in fact atrophied, becomes smaller.

Q: I see. So initially swelling, but after a few months, few weeks, it becomes smaller.

A: Yes.

Q: As the swelling grows, would there be pain?

A: Yes, your Honour.

Q: And this pain would increase until the swelling reached its peak?

A: Generally speaking, if the patient does not sort of get used to the pain, sometimes after a period you become refractory to the pain,…..

301. Dr Notley gave a comprehensive account of how physical damage can be caused within the testes from compression of the testicles with sufficient force to produce atrophy in the longer term. He said that the blood vessels in the scrotum are numerous and profuse and will be damaged in just the same way as the vessels within the testis. The vessels will be contused and probably torn and split. The only way the injury can produce testicular atrophy is by disruption of the blood vessels. The capillaries, arterials and venials haemorrhage because the testicle itself is crushed and so the vessels are broken, disrupted, torn, split or pulled apart. These are microscopic blood vessels. They are very fine and very delicate. If the testicle is distorted to pass through a small gap, those vessels are going to be subjected to shearing forces, which are going to tear them, pull them and break them. So as they bleed, one gets an intratesticular haematoma. I agree thus far with Dr Notley.

302. The scrotal skin also suffers from the same pressure. The 'very profuse subcutaneous vascular plexus in the scrotum' cannot escape damage as well, and the patient will then have an external as well as an internal haematoma. Dr Notley explained that extremely small damage to blood vessels within the scrotum could produce a spectacular haematoma. He said:

Witness: ..I mean, it's just not a reasonable assumption to say that you can have sufficient force supplied to the testicle to disrupt the testicle and it will not damage the testicular vessels. You only need …an **extremely small damage** to blood vessels within the scrotum **to produce a spectacular haematoma**. I know this from long experience, I do vasectomies through very small incisions, perhaps, half-centimetre incisions, and I've, from time to time, produced extremely major scrotal haematoma. So, you don't need very much. It spreads. ……. if I do the same sort of injury to a blood vessel in the scrotum, quite a small one, because it is free and elastic and floats in the breeze, a small bleed will not be confined ….It would tend to spread round the scrotum. So you will get a decent-size haematoma even with quite a small local injury. So I can't accept that you can pass a gentleman's testicles, ..whole scrotum, through a pair of rollers or a roller and a smooth rock, which is only 15 millimetres … apart , and sufficient to create a crush injury, a compression injury to the testicle, which I agree it's a rather special case, but I cannot accept that it would not damage the scrotal tissue sufficiently to produce a haematoma of some size or other which somebody would see, and that would happen, that's, haematoma will become visible within five or six hours, in my contention. I just do not believe you can do that sufficient to crush a testicle and not get a scrotal haematoma. It is inconsistent, to my knowledge of anatomy.

303. Therefore, it is not surprising at all if a contusion results in a large scrotal or extratesticular haematoma. In fact, that is usually an expected consequence of contusion. I can readily understand that. But I wanted to find a credible and reliable explanation for the large extratesticular haematoma if the cause was wholly surgical in nature. That was where I became most curious. So I asked Dr Notley to explain what one could expect to see when there was purely venous congestion of the testicles (assuming an overligation by Dr Tan during his operation) but with no physical injury sustained to the scrotum and testes (during the fall). Why was it that Mr Harte could develop such a spectacular swollen scrotum? It was most puzzling.

304. Based on the all the evidence before me, it became fairly clear that a large extratesticular or scrotal haematoma would not happen *per se* from the ligation or overligation and division of the veins or surgical damage to the arteries resulting in a shut down of the blood flow to the testes. That seemed to rule out the plaintiff's theory. If so, what reasonable biological explanation could there be for Mr Harte's large scrotal or extratesticular swelling, if indeed the plaintiff was right that there was no contusion injury after all.

305. Then Dr Notley explained:

> Witness: Well, if I may explain? The testicle swells but it probably doesn't swell to more than about twice its normal size because of the tight tunica albuginea. You know, that's what I think we all agree. But, by then, the scrotum has within it an acutely congested, abnormal, inflamed, angry, bad, dying testicle. The body reacts to any tissue like that by producing the classical inflammatory reaction which is the body's response to any injury, be it bacteria, be it viral, be it trauma, burns, hit it with a hammer, scrape it with a piece of barbed wire. The blood vessels locally will dilate, the capillaries, you remember the little tiny tubes that we have between the end of the arteries and before the veins, where all the activity in the tissues takes place, where all the chemical activity, the exchange of oxygen and carbon dioxides takes place, they become permeable as a result of the inflammatory reaction being produced by some hormones, which I'm afraid I cannot remember the name of, but they become permeable so that the fluid content of the blood leaks out into the tissues, that's the lymphatic fluid, but greatly exaggerated by the increased permeability of the capillaries. There is the, at the same time as this, a great increase in the blood supply coming into this as a result of the inflammatory response. So you get swelling as a result of the exudation that is going on, as part of the body's normal reaction to a piece of dead tissue within it. You get swelling due to the increase of blood flow through it, and because there's an increase in blood flow, you get an increase in heat of the area, so you feel hot. You also get an increase in the red colouration because it is oxygenated blood that is being passed through these dilated vessels.
>
> His Honour: But the tunica, isn't it a fairly impermeable layer?
>
> Witness: But we're not talking about the tunica albuginea.
>
> …..
>
> His Honour: But I'm concerned with how this swelling is spreading to the other parts, you know, the mechanism of it
>
> Witness: But it is the reaction of the scrotum to this happening within it, because within it is a testicle that has been injured, has swollen to twice the size, the tunica albuginea will, to a certain extent, be involved in the

inflammatory change of –

His Honour: You mean that would also swell? That would also be inflamed?

Witness: Yes, this will be thickened and exudate will form upon it as a result of the infarction, and this is what the scrotum itself will react to. So the chap will end up with a big red scrotum after twelve hours or so, which is acutely painful and tender to touch. ….After all, the tunica albuginea has all the blood vessels to the testicle passing through it. So it is, as it were, like---it's more like a pin cushion, isn't it, with lots of little holes in it where the blood, the arteries go in, the veins come out, so it's not an impermeable sheet like a polyethylene or damp-proof membrane you might care to put in your cellar to keep the water out. It's not like that at all. It's completely permeable to these things. And, particularly, if the blood vessels have been damaged and congested, you will have an exudation coming out into the scrotum which will produce this **local inflammatory reaction**.

His Honour: So that causes the extratesticular swelling?

Witness: **That causes the extratesticular swelling, the big scrotum**.

His Honour: All right.

Witness: **So the chap, the next morning, will have a big red, hot, painful, scrotum**.

305. Thus Dr Notley came up with this very attractive proposition that the resulting testicular infarction from devascularisation led to a severe inflammation in the morning. So Mr Harte would wake up finding himself with a big, red, hot, swollen scrotum. Dr Notley thus attributed the large red scrotal swelling **purely** to the inflammation, the cardinal signs of which would manifest within some hours of the infarction and progress over the next six to twelve hours. In its acute form, inflammation would be characterised by classical signs of pain (*dolor*), heat (*calor*), redness (*rubor*), swelling (*tumor*) and loss of function (*function laesa*). This reactive scrotal swelling with pain and reddening would increase the size of the scrotal swelling progressively over the next 24 hours or so. By then Mr Harte would have a firm, enlarged, red and painful scrotal swelling which would be tender to touch.

306. I have great difficulty accepting that the body's reaction to and rejection of a dead pair of testes alone can produce such a large scrotal swelling without **an extremely severe** inflammation, with infection and pus, and perhaps also accompanying fever of some sort from that infection, which Mr Harte never had. Further, I find it exceptional that just overnight, the testes would have died so rapidly, and the body would have reacted so violently and so quickly to the dead testes, that in just less than about 20 hours, Mr Harte would have developed a red, hot, very swollen, tender and inflamed scrotum adversely reacting to and rejecting the pair of dead testes within.

307. Although I do accept Dr Notley's explanation (which has also been confirmed by other expert witnesses) that a very large swelling in the nature of a scrotal haematoma can readily form from a minor haemorrhage of a small blood vessel, nevertheless I think it is quite unlikely for reactionary inflammation alone from a pair of infarcted dead testicles to produce a large swelling the size of a mango so quickly (without the aid of a haemorrhaging blood vessel of some kind).

308. There is no evidence of him complaining of a red, hot, swollen and inflamed scrotum. All the evidence I have is a bright red scrotum turning later into a reddish purplish colour, according to Mrs Harte. I also have the evidence of Mr Harte that he had a painful scrotum. However, he did not say that his red and hot scrotum was also tender to the touch, typically expected of badly inflamed or even septic scrotum. Why should an inflamed bright red scrotum turn to a deeper reddish purplish colour, and

after some 4 days later become a little bluish in colour as observed by Dr Tan, which is most incomprehensible and totally inconsistent with a bad reactionary inflammation. I find somehow that a deeper reddish purplish colour, changing to a bluish colour is more consistent with the appearance of an extratesticular/scrotal haematoma than a severe reactionary inflammation. Further, the absence of tenderness to touch typically reflects more of a haematoma than a septic and reactionary inflammation.

309. If it was indeed a reactionary inflammation, it is inconceivable that Mr and Mrs Harte should consider applying ice on his scrotum. Applying ice is more consistent with an attempt to reduce swelling from a haematoma than an inflammatory reaction in the scrotum against a pair of dead testes.

310. Mrs Harte's evidence was that her husband was in pain when Dr Tan prodded his scrotum area. After examining him, Dr Tan said that if the swelling did not go away, then he would have to draw blood from his scrotum. Dr Tan explained to them that Mr Harte most likely suffered from a scrotal haematoma. Dr Tan informed her that there was a blood clot and her impression was that the swelling was due to this. Again, there was nothing discussed about any severe reactionary inflammation.

311. The symptoms and evidence in my view do not fit Dr Notley's postulation that the entire reddish swollen scrotum the morning after the surgery was **purely** the result of a reactive scrotal inflammation. I cannot rule out that there may be **some** inflammation giving the reddish colour. Physical injury may also give rise to inflammation. Blood from ruptured capillary vessels slowly permeating to the tissues near the skin can also make the scrotum look 'reddish'. A taut scrotal skin may enhance the colouration. In fact, the subsequent change to a darker purplish red or reddish purplish colour indicates the presence of some scrotal haemorrhage. The tinge of blue colour observed by Dr Tan confirms this.

312. Dr Tan's contemporaneous notes of a '**scrotal haematoma**', and the fact that he told Mrs Harte of the presence of '**dried blood**' in his scrotum reinforce my opinion that there was in fact some extratesticular haemorrhage, which points towards the existence of a contusion injury. I shall be examining the evidence of an extratesticular hematoma in more detail later. For the moment, it suffices to state my view that the extratesticular haemorrhage with the red blood slowly permeating to the outer layers of the scrotum, and the inflammation arising from the contusion injury itself, additionally coupled perhaps with some minor inflammatory reaction at that stage to the gradually dying testes (damaged by a contusion process as explained earlier) constitute some of the multiple factors swelling Mr Harte's scrotum to the size of a mango, and colouring it bright red on the morning after the surgery.

313. An intratesticular haematoma (**without** an accompanying extratesticular haematoma) will obviously not result in a black or blue-black or dark reddish purplish coloured scrotum. I doubt very much that the colour of the haematoma within the testes can be seen through the scrotal skin. I reject Dr Notley's evidence of 'black testicles shinning deeply within the scrotum' to explain the 'tinge of blue' colour noted by Dr Tan when he examined Mr Harte on the fourth day. As the scrotal wall consists of 5 layers, I find it extremely difficult to accept that the dark coloured infarcted testes can be seen through all these layers to give it a tinge of blue colour.

314. In my opinion, the discolouration of the scrotum and the changes in colour seen by Mrs Harte and Dr Tan are likely to be associated with primarily an extratesticular haematoma and secondarily with some inflammation at the same time. I reject Dr Notley's evidence that the discolouration seen on the scrotal skin can possibly reflect the colour of the infarcted and dead pair of black testes. For the reasons given earlier, I also reject the contention that all contusion injury and resulting extratesticular haematomas, regardless of severity, must necessarily lead to a black coloured scrotum.

iv. Extraordinary force not needed

315. The plaintiff's experts relied heavily on the supposition that extraordinary force was needed before any blunt trauma to the testicles could occur. Dr Schlegel kept referring to the paper "*Bilateral Testicular Atrophy Following Blunt Trauma*" by MacDermott and Gray of the Department of Urology, Royal Infirmary, Bradford to demonstrate that a scrotal haematoma and bilateral testicular atrophy could only result when the impact forces reached the order of magnitude of a car falling with its

wheels landing on the testicles. To my mind, it is a gross exaggeration to say that the forces must reach such proportions before they can cause testicular contusion and bilateral atrophy.

316. In fact, I find that the comment of the authors of that article to be most interesting:

> In this case, there was **neither rupture of the tunica albuginea nor disruption of the macroscopic blood supply of either testis; indeed the testes appeared completely viable at operation.** The testes and scrotum were, however, caught under the revolving car wheel and subjected to a **compressive force**. This would have been sufficient to cause **microvascular damage to the testicular tissue leading to atrophy**. This case again illustrates the **importance of microstructural damage in testicular trauma**.

317. The doctors there concluded that both testes were viable after the accident because the tunica albuginea had not been breached and fresh bleeding was obtained through a short incision made in the tunica albuginea. Yet at the review 5 months later, both testes were found to have atrophied to pea-sized nodules. **Microvascular** damage and **microstructural** damage from trauma had led to the bilateral testicular atrophy. This shows how delicate the testes can be since damage at the microlevel can still lead to devastating results. I do not believe at all that extraordinary force is necessary in every case having regard to the fact that damage at the microlevel is already sufficient, given time, to produce testicular atrophy as that case demonstrates.

318. This supposition that maximum force is needed to cause testicular atrophy is debunked by Dr Tay Kah Phuan, a urologist practising at the Gleneagles Medical Centre and having his own private clinic called K P Tay Urology. He testified that 'very minimal trauma when applied very wrongly can result in very maximal injury' citing a recent case he treated. He accepted, quite rightly, that as a general rule, a minimal force would not cause a severe injury but one must never discount that a minimal force could also cause a maximal injury. This comment is with reference to the delicate tissues in the scrotum including the testes.

319. The brief facts of that recent case are as follows:

> (a) A 15 year old Indian boy was accidentally kicked on 7 June 2000 in the groin by his younger 13 year old brother whilst they were both asleep together on the bed. The boy woke up and noted that he was kicked. His younger brother was still asleep and unaware that he had kicked his older brother in the groin. The older boy went back to sleep again. In Dr Tay's opinion, a sleeping 13 year old boy obviously could not have kicked very hard.
>
> (b) On waking up the following morning, 8 June 2000, the boy had some pain and some minimal swelling. The pain was not sufficient for him to complain to anyone yet.
>
> (c) The swelling progressed. As the swelling increased, the pain increased. He also developed a low-grade fever. The father brought him to Dr Ravi, who brought the boy and his father to consult Dr Tay just before midnight on 9 June 2000.
>
> (d) Dr Tay examined him on 10 June 2000 (i.e. past midnight and in the early hours of the morning). The boy had some discomfort at the lower part of his abdomen. Dr Tay found that his scrotum and penis were swollen. His testes were not palpable on both sides. He had a scrotal haematoma the size of a small orange and it was 'dusky blue-black' in colour. This meant that there was a blood clot.

(e) Ultrasound revealed a normal right testis and a left intratesticular haematoma with possible left testicular rupture.

(f) He was operated on immediately at 0140 hours on 10 June 2000. Operative findings revealed a large scrotal haematoma as well as a haematoma on the left testis and epididymis with near rupture because of the swelling and the trauma, and haemorrhagic infarction of the left testis, epididymis and spermatic cord. He also had a haemorrhagic hydrocele. It was a collection of bloodstained fluid in the area outside the testis. His left testis was removed. The tunica was not ruptured.

(g) Histology report stated that there was reddish-black discolouration throughout the tissue of the testis. There was extensive congestion. Sections of the testis and epididymis under microscopic examination confirmed the presence of extensive haemorrhagic infarction consistent with the effects of haematoma induced by trauma.

(h) Dr Tay said that in blunt trauma like this, one would not usually see injury on the skin unless it were to be so extensive as to rupture the skin.

320. Dr Tay drew attention to the histology report which revealed that *'a few viable seminiferous tubules [were] seen focally, showing normal spermatogenesis'* despite the extensive damage to the boy's testis. The fever was reactionary and in response to inflammation. There was extensive inflammation. To all intents and purposes, the left testis was dead and he had to remove it. According to Dr Tay, tissue death must occur first before atrophy can set in. After the tissue dies, it undergoes fibrosis and scarring. Thereafter, it slowly shrinks with time.

321. He said that the boy would have some initial pain which woke him up but the pain was not severe enough to interrupt his sleep or for him to beat up his brother so to speak. But on the following day, the boy noted increasing pain which progressed. It progressed until the following day when he actually went to tell his father that he was kicked. The pain symptoms from the trauma to the boy's scrotum were remarkably similar to Mr Harte's.

322. In Dr Tay's opinion, it was highly unlikely that he could have saved the testis even if the boy had come to see him as early as 2 hours after the accident. If it was tissue death because of a crushing injury, it would take time for the tissue to die, but it would die. In his view, there was nothing much that the doctor could do. Unlike torsion injury interrupting the blood supply, the surgery to untwist the testis to restore the blood supply should be done within 6 hours in order to save it.

323. Dr Tay also gave an instance of another patient with much delayed scrotal swelling and pain after trauma. This was a cyclist wearing tight lycra pants. When he pulled the bottom of the pants to urinate, his lycra pants slipped and snapped back, hitting his right testis. He had very little pain and could cycle home. He noticed pain on his right testis only at night, followed by swelling in the testis a day later. The pain and swelling subsided but recurred 2 days before seeing Dr Tay. When Dr Tay examined him a week after the accident, his testis was swollen to nearly double its size. The right epididymis was thickened and very tender. There was a small hydrocele, a collection of fluid in the testis. The skin was perfectly fine with no scrotal haematoma. The ultrasound report revealed a 'right-sided epididymitis' (meaning inflammation of the right epididymis with evidence of 'marked hyperaemic changes') and 'mild orchitis' (meaning infection of the testis). This was a relatively minor trauma according to Dr Tay. It was fortunate that he did not lose his testis nor suffer atrophy.

324. Another interesting example given by Dr Tay was a case he treated many years ago, where a Thai worker had swelling of his testes. There was no history of trauma that he could tell Dr Tay. He had infection of the testes and infection of the epididymis. He had spiking fever that went up and down. He was given injections and antibiotics. About 5 days or a week later, the testes actually eroded through the scrotum and discharged pus. This anecdote is important, as we shall see later, as it helps to establish if it was only the intratesticular haematoma plus very severe reactionary inflammation (without any accompanying

scrotal or extratesticular haematoma) that caused the scrotum to swell to the size it was on the morning after and to continue to swell to the size of a mango by the 4th day. To my mind, that inflammation must be extremely severe for the reactionary swelling to be the main contributory of the mango sized scrotum.

325. I am inclined to believe that it was the intratesticular haematoma, plus the extratesticular haematoma plus some degree of inflammation, which together caused the extensive swelling seen in Mr Harte's scrotum. One has to bear in mind that the plaintiff never had any fever, which would naturally be expected for a serious reactionary inflammation. Dr Tay had explained very succinctly that trauma *per se* would not cause infection. Trauma *per se* causes inflammation. With an inflamed tissue, there is going to be swelling in that tissue. Then it can become secondarily infected but it will take some time. In the case of the Indian boy for instance, trauma remains as the primary injury although there can be secondary reactive inflammation with associated fever.

326. I admitted such anecdotal evidence under s 48 of the Evidence Act, being evidence which might be consistent or inconsistent with the opinion of the different medical experts including Dr Tay himself in relation to the rate of swelling, degree of pain felt, severity of the trauma and the extent of testicular damage. Just as with the other case studies cited in the medical literature admitted into court, these first hand case studies of Dr Tay are relevant evidence.

327. Dr Myint Soe referred me to *C v C* reported in Butterworths: Personal Injury Litigation Service (Issue 31) where a tanker driver used a toilet with a defective seat. The seat came loose, trapping his left testicle. Later, his left testicle had to be removed. Again, this shows that the force needed to cause severe injury need not be very large.

328. When the example of the Indian boy was put to Prof Schlegel, he attributed the damage to 'torsion' of the testicle, which he said commonly occurs in young boys. He said that it was not pathologically possible to tell the difference between torsion and trauma and the results were equally consistent with both.

329. Based on the totality of the evidence, I think that the infarction in that Indian boy's left testicle was due to trauma and not torsion. There was no sharp pain felt during the night because the boy fell asleep after the kick. A sharp pain is normally associated with torsion injury. Dr Tay, who examined the boy and operated on him to remove the dead testicle, never diagnosed a torsion injury after considering the history related by the patient, the ultrasound report, his operative findings and the histology report. His conclusion was affirmatively that of trauma to the testicle due to the kick. Without any question, this case clearly shows how delicate the testes really are.

v. Evidence of a Scrotal Haematoma

330. I do not consider the absence of a black coloured scrotum to be evidence fatal to Dr Tan's postulation of a contusion injury sufficiently severe to cause atrophy. Mr Harte's reddish purplish scrotum (as described by Mrs Harte), depicting a dark coloured scrotum, might well show up by the fourth day as a tinge of blue as described by Dr Tan, depending on how the inflammation and bruising were resolving themselves.

331. In my opinion, a blue-black coloured scrotum might well be conclusive that there was a scrotal haematoma but the absence of the blue-black colour is not necessarily conclusive that there could never be any trauma or physical contusion to the testes, and hence there could not be any scrotal haematoma.

332. Prof Li Man Kay quoted from medical literature that 50 Kg is required to cause testicular atrophy. I believe that Prof Li must be referring to the paper Wasko and Goldstein: *Traumatic rupture of the testicle*, where the authors have estimated that a force of 50 kg of pressure is needed to rupture the tunica albuginea in blunt scrotal trauma.

333. Although none of the experts have suggested that Mr Harte had sustained any rupture of the tunica albuginea covering his testes, nevertheless it is still enlightening to find out what are the signs and symptoms expected in cases of blunt trauma

involving a **rupture of the tunica albuginea**. Fournier, Laing and McAninch stated the following at page 382 of their paper: *Scrotal Ultrasonograph and the Management of Testicular Trauma*:

> **Signs and Symptoms**
>
> Rupture of a testis secondary to blunt trauma is a violent and immediately painful event. Wasko and Goldstein have estimated that a force of 50 kg of pressure is needed to rupture the tunica albuginea in blunt scrotal trauma. Nausea and vomiting are frequently associated, as occasionally is syncope. The amount of scrotal swelling and ecchymosis **can differ**. Rupture of the tunica albuginea behind the tunica vaginalis can be associated with **no ecchymosis** and **minimal** to moderate testicular enlargement because bleeding and the extruded seminiferous tubules will be contained by the tunica vaginalis. On the other hand, if subalbugineal arterioles are involved, the hematocele can be large and may compress the parenchyma, eventually leading to parenchymal atrophy. If the rupture involved the junction of the tunica albuginea and tunica vaginalis in the area of the epididymis, the bleeding will extend into the scrotal sac proper and lead to a scrotal hematoma.
>
> Findings on physical examination are **various**. Occasionally, the testis is displaced to the inguinal canal and not palpable in the scrotum. The most consistent sign is exquisite pain with palpation. Scrotal ecchymosis and swelling are also frequent, although **their absence does not rule out a rupture**. The differential diagnosis includes simple hematocele without rupture, torsion of the testis or one of the appendices, testis tumor, epididymis, reactive hydrocele, and spermatic cord. An accurate history should be taken if possible to ascertain the magnitude of the force causing the injury. Cassie has reported that an associated neoplasm may predispose to rupture after apparently minor injuries. In this case, **testicular rupture was manifested by swelling and pain some 16 hours after a fall down a flight of stairs**. Patients with normal testes will report scrotal trauma that is much more violent and directly localized and they usually experience pain and swelling immediately; alternatively, they may have been in an accident resulting in multiple injuries.

334. It is my opinion after considering all the evidence presented to me, that rupture of the small blood capillaries at the microscopic level does not require extraordinary trauma. The proposition that only extraordinary force can cause testicular damage is simply untrue. The above passage confirms that physical symptoms are indeed very variable. Even for testicular rupture, which is more serious, the pain and swelling manifested only some 16 hours after the fall. It was not immediate. The authors went on to say that the absence of scrotal ecchymosis (i.e. a small haemorrhagic area in the skin or mucous membrane either blue or purplish in colour) and swelling do not rule out an injury as serious as a rupture of the tunica albuginea.

335. I also disagree with the medical expert opinion presented on both sides that a bruise must necessarily appear black or as 'black as a gown'. It seems to me that in a very severe case of bruising that might well be the colour. But bruising from a less substantial trauma involving microvascular damage cannot to my mind, become as black as that.

336. In cases of soft tissue trauma, e.g. a heavy slap on the cheeks or thighs may cause swelling and redness but not necessarily a black or blue-black bruise. Whereas a punch landing on an area of the body which is near a bony structure e.g. the forehead or cheekbone will more likely result in a denser blue-black bruise. Even then, it may not necessarily be of that colour as can be seen from nurse Ms Tan Sang Eng's testimony that she observed some **redness** (not blackness) at the plaintiff's left forehead and left cheek after he had hit his head on the hard toilet floor during his fall from the toilet seat. Another nurse Ms Looi Chai Hang, who saw the bruise on Mr Harte some 1 hours later described it as having a very slight bluish colour. All this

clearly shows that there is no certainty that a bruising injury must show up as a black or blue-black swelling. It is a fallacy. As can be seen from the evidence of these two nurses, a bruise from trauma can start off with a reddish colour and later change to a slight bluish colour. The colour changes observed on Mr Harte's swollen scrotum are remarkably similar.

337. The swelling reactions and colour of the bruising depend on the magnitude of the force applied, whether it is an impact or non-impact force, the kind of body tissue involved, whether the tissue is against a bone or against another soft part of the body, whether there is a rich supply of blood vessels in that area, and the degree of rupture or haemorrhage of these vessels at the traumatised area. In my opinion, there can be internal testicular haemorrhage without obvious signs appearing on the scrotum skin immediately or at all. The reddish, bluish, purplish reddish, blue-black or even black colour may surface later on as the blood from the ruptured vessels in the scrotum gradually permeates to the outer skin layers, and various complex, biological, chemical and inflammatory reactions subsequently take place, giving the various possible colours.

338. Thus, it is far too dogmatic to think that all scrotal bruising must be black and if there is no black colour seen, there is no bruising and therefore no trauma of any kind, or that redness seen on the scrotum must exclude an extratesticular haematoma. Not only is it simplistic, it is also unrealistic and completely ignores the fact that multiple factors come into play.

339. I think that a more balanced view is that expressed in "*All New Family Medical Guide to Health and Prevention*" by Martin and Powell, 1995 Edn where it simply states:

> A bruise is nothing more than blood under the skin. If blood leaks out of a blood vessel due to any cause, a bruise forms. It may be **red, yellow, orange, or blue**. Blue usually indicates a deeper bruise; the color is caused by the blood pigment, hemoglobin, reflected through skin tissue.

340. I happened to check with the Dorland's Illustrated Medical Dictionary and am gladly reassured that the '***reddish purplish***' coloured scrotum seen by Mrs Harte is entirely consistent with the presence of a scrotal haematoma. Dorland's Illustrated Medical Dictionary 20th Edn gives the following definitions:

> Ecchymosis : a small **hemorrhagic spot**, larger than a petechia, in the skin or mucous membrane forming a nonelevated rounded or irregular, **blue or purplish** patch.
>
> Petechia : a pinpoint, nonraised, perfectly round, **purplish red** spot caused by intradermal or submucous **hemorrhage**.
>
> Intradermal : 1. Within the dermis. 2. Intracutaneous.

341. Naturally, a collection of round purplish red spots caused by intradermal or submucous haemorrhage in the scrotum will show up as a 'reddish purplish' scrotum.

342. I note that Dr Schlegel had medically examined Mr Harte on 12 January 2000. Being an experienced and eminent urologist, Dr Schlegel must have taken a detailed history from Mr Harte. Yet in his report given as late as 22 March 2000, Dr Schlegel stated his view that Mr Harte had developed a **scrotal haematoma** subsequent to the varicocelectomy. Nothing was mentioned in Dr Schlegel's report that Mr Harte had developed an inflamed red scrotum (without a scrotal haematoma), which Dr Notley postulated had happened. I would imagine that Dr Schlegel knew the difference between a scrotal haematoma and an inflammatory swelling.

343. Similarly, Dr Dubin who also saw Mr Harte on 21 October 1999 wrote to Mr Harte on 27 October 1999 stating that he had **haematoma** of the scrotum after his operation in Singapore. Again, I imagine that Mr Harte would probably have told Dr Dubin about the signs and symptoms he felt after his surgery and would have given Dr Dubin the whole history. Yet Dr Dubin did not mention anything about a large red, hot scrotal swelling arising from a severe reactionary inflammation, which would be quite

different from a haematoma.

344. Dr Tan Hun Hoe testified that the haematoma he saw some 4 days later was the size of a large orange and it was **'a tinge of blue'** which in his opinion was due to the 'altered blood clots'. Dr Tan explained that 'altered' meant that the blood there was no longer functioning having lost its oxygen, and it was beginning to denature i.e. to change its biological and chemical properties.

345. Dr Tan gave Mr Harte a medical report dated 24 May 1997 stating that:

> He was reviewed on 2nd May '97 and was noted to have the complication of a **scrotal haematoma**. He was prescribed Zinnat, Danzen, Voltaren and Antacids and when last reviewed on 9th May '97 the **haematoma** had begun to subside.

346. I presume that Dr Tan based his medical report on his contemporaneous medical notes on 2 May 1997 where he wrote, "*Had **scrotal haematoma***". On 9 May 1997, he recorded, "***Haematoma subsided.*** *Complained of hard testicles, left worse than right. Ultrasound scan: organised clot around left testicle, small hydrocele.*"

347. The mention of an organised clot around the left testicle also suggests to me that there was a haematoma rather than an adverse reactionary inflammation to a pair of dead infarcted testes.

348. Dr Tan in his report to the Singapore Medical Council dated 23 October 1998 stated that:

> On review on 2.5.97, he complained of a scrotal hematoma that had developed 2 – 3 days after the operation. The hematoma was non-tender. He was prescribed antibiotics prophylactically and Danzen to aid resolution of hematoma.
>
> He was reviewed again a week later on 9.5.97. The hematoma had subsided but he complained that the left testicle was harder than the right.
>
> The next review was on 26.5.97. The swelling had subsided almost completely but there was residual induration of the scrotal sac which was expected to take a longer period to subside.
>
> …..
> …..
>
> In my opinion the cause of his testicular hypofunction is difficult to determine at this time as it was highly unlikely that the arterial supply could have been cut off during the operation which was carefully done with magnification. Whether the fall and the subsequent hematocele had an effect on the outcome is again difficult to ascertain. However due care was taken to manage the hematocele conservatively as it was not tender and was expected to subside spontaneously.

349. I observe that there was mention in the contemporaneous medical notes for 26 May 1997 that "*Induration subsided but can still be felt. Swelling subsided.*"

350. The fact of induration of the scrotal sac suggests the presence of some hardening due to dried blood. That was also what Dr Tan had told Mrs Harte, which Mrs Harte had scribbled down in her notes exhibited at ABD 171.

351. Further, the fact that the scrotum was non-tender does not suggest a severe reactionary inflammation to me. I expect a reactionary inflammation giving rise to swelling the size of a mango to be very tender.

352. The later notes taken by Dr Tan for 8 October 1997 had the following remarks showing Dr Tan's puzzlement:

> Discussed possibility of Retrograde ejaculation, possibility of 'page' kidney effect of haematoma on testicles. **? mechanism of haematoma.**
>
> Haematoma occurred 2 – 3 days post operation, **initially very gradual**."

To complete the picture, his notes for 17 October 1997 were:

> Mrs Harte called up.
>
> Wanted clarification on operation findings and procedure
>
> Past cases and results
>
> Discussed haematoma
>
> **Patient's fall in toilet on front? Cause of haematoma**

353. After the defendants closed their case, counsel for the plaintiff recalled Dr Notley who threw in at the last minute an explanation of a reactive inflammation for the bright red coloured swollen scrotum appearing on the morning after the surgery. This red herring admittedly threw me off balance in my analysis of the cause of the bilateral testicular atrophy.

354. The sworn complaint by Mr Harte himself to the Singapore Medical Council dated 2 September 1998 puts to rest whether he had a severe reactionary inflammation or a haematoma. Mr Harte wrote:

> After the surgery I had a very painful **scrotal hematoma**. I consulted Dr Tan two days after the surgery and he advised that the swelling should go down in a few days and everything would be normal. When the swelling persisted, I made an appointment to see him. The soonest he could see me was on May 2, 1997. He said that if the swelling did not subside he would have to **drain the blood from my scrotum**. He didn't seem concerned and asked that I return in one week for another check-up. The swelling did eventually subside, but, until it did I called Dr. Tan on a daily basis for advice. I asked if I should **put ice on it** and was told not to by one of his assistants.

355. Again why should there be a need to consider draining of blood from his scrotum if there was no haematoma but a severe reactionary inflammation? Further, why was there a need to consider putting ice on the scrotum if it was a severe reactionary inflammation? My own understanding is that an ice pack will help reduce swelling in a haematoma and is not quite appropriate for a reactionary inflammation of a septic nature. From all the above evidence, I am convinced that Mr Harte did suffer from an extratesticular (or scrotal) haematoma besides an internal haematoma. This conclusion points towards physical contusion rather than surgery as the cause of both kinds of swelling.

<u>vi. Absence of abrasion and bruising on the scrotum and penis</u>

356. A/Prof Foo held the view that it was not necessary to have a blue-black penis in the fall from a toilet seat. I agree with him. It depends on the nature of the force and how the force is applied. It is possible for the penis skin to be scratched when falling off a toilet seat. But the fact that scratch marks are absent cannot exclude that event. Where there is the same degree of contusion to the testis and flaccid penis, contusion injury is more likely to surface in the testis than in the penis because the

testis is hard and taut whereas the penis is flaccid. A flaccid penis moulds to the force acting on it but not an erect penis, which can therefore suffer from a contusion injury. Like the erect penis, the firm testes can succumb to contusion injury.

357. The inner rim of the toilet seat is not a sharp object. The inner edge is contoured and smooth as can be seen in photograph D51G of the actual toilet seat in question. Given the smoothness and rounded contour, the absence of obvious abrasion or bruising on the skin of the penis or the scrotum is hardly surprising, regardless whether there was any contusion injury. Dr Notley also did not think that there would be bruises on the penis if the patient slid over the front edge of the toilet seat.

358. I believe that the soft, flexible, stretchable scrotal skin and the underlying dartos muscle can withstand a fair amount of pressure and stretching without sustaining bruising. Dr Notley aptly observed that one could sit on the skin of the buttocks without experiencing bruising. But with the testis itself, which is encased in a tough tunica envelope, I think it is a completely different matter. Compressing an egg-like testis encased in a fairly tough, inelastic membrane generates a considerable buildup of pressure within it, which may cause tissue damage and haemorrhagic ruptures internally without any significant observable bruising to the scrotal skin.

vii. Uneven damage to testicles

359. None of the plaintiff's experts could provide a plausible reason based on their own theories why the testicle on the right side originally much bigger at 25 cc had ended up being smaller at 6 cc whereas the left smaller testicle of 15 cc had ended up being larger at 8 cc. This seems to contradict their position that the left operation being a repeat operation was more risky than the right. Yet the damage on the right turns out to be far more severe than the left.

360. Dr Tan offered a logical explanation that where a compressive force acts on two unequal sized testicles at the same time, the larger one is naturally more traumatised. Second, the right testicle is usually attached by a shorter cord and therefore, it will sustain more of the traction injury when both testicles are equally trapped at the inner rim of the toilet seat before being pulled through and compressed. For this reason, the right testicle will suffer greater traction injury. See the illustrations D21 and D22 by Dr Tan. A/Prof Ng Wan Sing from the School of Mechanical and Production Engineering, Nanyang Technological University confirmed this aspect of Dr Tan's evidence from an engineering point of view.

361. Dr Tan testified that a force of 50 kg would be more than enough to rupture the tunica albugenia. The corollary would be that a force less than 50 Kg might not cause rupture but it could still be sufficient to cause contusion injury.

362. That the right testis has been more severely atrophied is also consistent with what Munter and Faleski stated in *Blunt Scrotal Trauma: Emergency Department Evaluation and Management*:

> Testicular, scrotal, and epididymal hematomas and contusions may occur as a direct result of a blow. Because of the shape of the testicle, its mobility within the scrotum, and the protection of the tunica albuginia, testicular rupture occurs when the testicle is trapped between the striking object and a hard surface, typically the public bone or thigh. ….
>
> The **right testicle is more often injured**. This is postulated to be caused by the **higher position** of the right testicle, with a resultant **greater chance to be caught against the pubic or ischial bone.**

363. The higher position of the right testicle mentioned by Munter and Faleski confirms what Dr Tan had said that the right testicle is usually attached by a shorter spermatic cord.

(g) Biomechanical analysis

364. Dr Tan's principal defence was that the fall of Mr Harte from the toilet seat was the sole cause of his bilateral testicular atrophy ('fall theory'). The plaintiff weighed about 103 kg at that time. He was indeed heavy. By comparison, a force of 50 kg would be sufficient to cause rupture and very severe damage to the testes. Having heard evidence from two engineers, I shall now scrutinise Dr Tan's 'fall theory'.

365. Mrs Harte described how Mr Harte fell:

> There were 3 nurses may be 4 nurses were holding him and walking him to the toilet and I was following behind. They sat him on the toilet and then they left the toilet. I remained inside. He was looking a little pale at that time and was sweating. So I turned around to the sink to get a paper towel. When I turned round back to Denis, his eyes were rolling back in his head and I could see that he was about to pass out. I moved towards him to hold him up but he was already falling forward. So I grabbed him to try to break the fall and that's when he hit his head on the floor.
>
> When he was falling, I grabbed his body at his chest and back, but he was so heavy that he continued to go forward and his head hit the floor first, followed on by the rest of his body. He was lying eventually on his left side with his left leg on the floor and the right crossed over his left leg and his hand above his head. As he was falling, his buttocks lifted upwards up from the toilet seat. At the time of falling, both his feet were at the axle point of the roll.
>
> As his head hit the floor, his body was in a couched position with his body fairly close to his thighs, when he rolled onto the floor hitting his head.

366. According to Mrs Harte, his body was so heavy that as he fell forward, his buttocks automatically rose upwards. His head hit the floor first. He eventually landed on his left side with his right leg crossed. His knee was protecting his groin and scrotal area. She maintained that his scrotum was **not crushed**.

367. This part of her evidence seems contrived because I do not think she was in a position to see whether his scrotum was ever sandwiched between his body and the floor. I also doubt that she could have in that split second observed that Mr Harte's scrotum was not crushed by the toilet seat, especially when her attention was focussed on preventing Mr Harte from falling. One has to bear in mind that Mr Harte was in hospital attire at that time which would have obscured much of her view in any case. As with all oral evidence, there is always a possibility of wrong recollections and even biased embellishments. For instance, Mrs Harte's evidence that 3 or 4 nurses held and walked Mr Harte to the toilet with herself following behind was contradicted by nurse Tan Sang Eng who said that only she and Mrs Harte helped Mr Harte into the toilet and seated him.

368. After Mr Harte fell, Mrs Harte ran to get help. The nurses came. Mrs Harte and the nurses could not lift him up from the floor because he was so heavy. After Mr Harte regained consciousness, he got up himself with some assistance from them.

369. A/Prof Ng Wan Sing from the School of Mechanical and Production Engineering, Nanyang Technological University, was called by Dr Tan to substantiate his fall theory. A/Prof Ng's report is at D 40.

370. A/Prof Ng basically applied a static analysis to what was clearly a dynamic situation. In my opinion, his qualitative analysis is a little simplistic. The results of A/Prof Ng's analysis can easily mislead if the limitations of his static model are not fully appreciated. A/Prof Ng conceded that his static analysis had not taken into account the dynamic nature of the forces,

motions and accelerations. His static model projected a shifting pivoting point about the toilet seat with toppling and sliding occurring thereafter. As Mr Harte slid off the edge of the toilet seat under the full weight of his body and given the closeness of his scrotum to the inner rim of the toilet seat, A/Prof Ng concluded that it was '*very possible that the scrotum pressed against the seat and rim during the roll over and sliding*'. With only a qualitative analysis, Prof Ng could not determine the amount of clearance, if any, between the toilet seat and Mr Harte's perineum region as Mr Harte slid off. Neither could Prof Ng establish the magnitude of the compressive force.

371. I find that A/Prof Lim Chwee Teck from the Department of Mechanical Engineering, National University of Singapore, presented a far more sophisticated quantitative finite element analysis using the computer software called 'ABAQUS/Explicit finite element software.' The computer simulation models the dynamics of the plaintiff falling off the toilet seat when he lost consciousness. From the trajectories, one can study whether Mr Harte's scrotum would be caught underneath the toilet seat or pressed between his body and the toilet seat during the fall. A/Prof Lim was tasked to analyse the dynamic nature of the free fall due to gravity assuming different initial torso angles of Mr Harte just before his torso started collapsing to his thighs due to syncope.

372. I agree that A/Prof Lim's dynamic analysis better predicts the trajectory of the fall than A/Prof Ng's static analysis. A/Prof Lim's report is at P50.

373. The approximations and assumptions applied in A/Prof Lim's model must be fully appreciated. He approximated the mass distribution, centre of mass and radius of gyration, and then calculated the moment of inertia of the various segments of Mr Harte's body relying on the '*Dempster's Body Segment Parameter Data for 2-Dimensional Studies*' shown at Appendix B of his report. The coefficient of friction between the plaintiff's thigh and toilet seat was obtained from a paper '*Friction behaviour of human skin'* by Sasada, Ide and Kawakami. The plaintiff's body is modelled as 4 segments viz (a) head, trunk and arms; (b) thighs and pelvis; (c) legs; and (d) feet. Each is modelled as a rigid segment connected to the other segments using frictionless joints.

374. As Mr Harte fell in an unconscious state, it is assumed that he lost muscular control instantaneously. Therefore, all the major joints in his body are approximated to function as loose frictionless joints. Mr Harte's body is assumed to be a deadweight falling under the influence of gravity. The model simply treats Mr Harte's non-rigid 3-Dimensional body as a rigid 2-Dimensional body with frictionless joints and with certain maximum limits of collapse angles at the knees and ankles based on actual measurements taken from Mr Harte himself.

375. In his report, A/Prof Lim described his engineering findings on the dynamics of the fall as follows:

> (a) The upper torso first pivots about the pelvis until it hits the thighs.
>
> (b) The upper torso and thigh then pivot as one mass about the knee with some simultaneous pivoting of the leg about the ankle.
>
> (c) The knee then starts to collapse under the weight of the body and reaches the minimum angle achieved by the knee.
>
> (d) The head and upper torso continue to move forward and downward with the head hitting the floor first followed by the rest of the body.
>
> (e) There is no sliding of the thighs on the toilet seat due to the great inertial torque arising from the substantial deceleration upon the rigid body impact.
>
> (f) The buttocks lift off the toilet seat.
>
> (g) There is noticeable clearance between the scrotum and toilet seat based on

a hanging distance of the scrotum of 6 cm.

(h) The closest the scrotum gets to the toilet seat during the fall is approximated to be about 4 cm, 3 cm and 2 cm for the fall from 80 , 70 and 60 positions respectively.

376. I accept that finite element analysis using software packages and computers are often used to perform complicated, engineering calculations and to solve complex engineering problems. In this case, the modelling of the fall (assuming different starting angles of a human body with the physical characteristics of Mr Harte and with certain general parameters obtained from biomechanical data about the distribution of body mass, radius of gyration and the position of the centre of mass for each of the body segments modelled), gives a better insight and understanding of the motions and trajectories of a fall off a toilet seat under the influence of gravity. Short of replicating either with Mr Harte on the toilet seat and subjecting him to anaesthesia to create the unconsciousness, or with an actual 3-Dimensional model built to mimic the physical characteristics of Mr Harte, the 2-Dimensional computer simulation is the next best and most practical solution. Although it is helpful as a guide, nevertheless one must remember its limitations and its inability to fully simulate the human body, which is flesh and blood.

377. The most useful part of the finite element dynamic analysis as opposed to the common sense and more readily understood static analysis of A/Prof Ng is to demonstrate the hitherto unforeseen effect of rotation of the torso about the knees before collapsing because of the forward and angular momentum of the torso in the course of the fall. Where there is no slip between the feet and the toilet floor, the tibia and fibula bones of the lower part of the leg act as a strong strut and they can transmit forces along its length through the frictionless pivot at the knee joint, to allow the upper torso to use it momentarily as a strut, over which the torso could continue to rotate about the knee pivot in the course of falling. The initial torso rotation about the strut at the knee pivot is the main reason for the 'lift' of the buttocks off the toilet seat or the 'flying' motion as described by Dr Myint Soe in his submissions. This fall trajectory cannot be appreciated without the dynamic analysis. The computer program is at exhibit P55 with full instructions for its operation.

378. A/Prof Ng in my view had no engineering basis for asserting that the ankles of the plaintiff would buckle from the onset of the fall and that the tibia and fibula would not act as a 'strut' and therefore, Mr Harte's torso could only rotate about the toilet seat as he fell off. Of course, if the feet of the plaintiff had already slipped (for instance where the floor was slippery), then the tibia and fibula would not function properly as a strut, and there might not be any pivoting about the knee at all in the course of the fall. The body would then have fallen as if the obstruction (in the way of the fall) from the lower leg were not there at all.

379. However, as the angle of the lower leg in relation to the floor was close to 90 , and as the floor was not exactly slippery, I take the view that the friction force was sufficient to prevent any slippage at the feet at least at the initial part of the torso rotation about the knee. I find support for this in the evidence of Mrs Harte that '*at the time of falling, both his feet were at the axle point of the roll*.'

380. During the later stages of the fall and as the angle of the lower leg to the floor became more acute, it might well be that the friction forces between the feet and the floor were overcome at some stage and both feet would begin to slip backwards. However, these later stages of the fall were not material in my analysis whether contusion was in fact possible. The effect of the feet slippage is relevant only when one needs to consider how much closer the final rest position of the body and buttocks after the fall was going to be. Logically, the earlier the slippage of the feet during the fall, the nearer would be the final position of the body to the toilet bowl.

381. If Mr Harte had fallen from a starting position with his torso completely resting on his thighs, then the results of A/Prof Lim's analysis and A/Prof Ng's analysis would tend to converge. In other words, the dynamic model predicts much of the outcome of the static model when the initial fall angle is set at the minimum of 25 , which essentially transforms the dynamic model into a static model. At 25 , there will no longer be any angular rotation nor angular momentum left in the torso just prior to hitting the thighs.

382. A/Prof Lim gave a quantitative analysis for the whole spectrum of fall angles between 80 and 30 whereas A/Prof Ng's

qualitative analysis related only to the far end of that spectrum with the fall angle at 25 . The real answer is probably somewhere in between the two ends of the spectrum. But which end of the spectrum is the actual fall going to be nearer? If the torso angle is closer to the low end of the spectrum (i.e. nearer the 25 angle which is the equivalent of the static model), then there is a higher probability of contusion. If the torso angle is towards the high end of the spectrum, then there is a higher probability that the scrotum will clear the toilet seat and not be contused at all.

383. Based purely on common sense (which I must caution myself may well be wrong), if the torso starts to fall from an angle closer to 25 (which essentially is the static model), I reason that the buttocks will slide first before toppling over the edge of the toilet seat. If there is any lifting of the buttocks in the course of the fall, it will be minimal. More likely, the lift will not be sufficient to enable the scrotum to clear the rim of the toilet seat. As such, it is probable for the scrotum and the then much larger testicles (prior to his atrophy) to be squashed by his heavy torso in the course of sliding off the toilet seat as he fell. Can this common sense reasoning of mine be confirmed more scientifically by the dynamic finite element model of A/Prof Lim? I shall now proceed to examine this.

384. I note that there are many important unknown parameters which affect the outcome predicted by the simulation: (a) elasticity and energy absorption characteristics of the stomach and thighs at the point of impact; (b) how Mrs Harte had held the plaintiff, the amount of force she applied to prevent the fall and the amount of energy she expended in doing so; (c) the rate at which Mr Harte lost consciousness; and (d) the energy loss in the process of stretching the strong back muscles of the torso and the skin as the body fell.

385. Since these parameters were not known or readily available, A/Prof Lim recognised that he had totally ignored them to make the modelling easier. He readily acknowledged that if these unknown parameters were taken into account, they would affect the clearance between the toilet seat and the scrotum.

386. What is clear in my mind is that these unknown parameters will negate the dynamic effects from the falling torso, reduce the angular velocity and momentum of the torso just before the impact on the thighs. Hence the 'lift effect' due to the rotation about the knee will be substantially reduced. Consequently, the clearance between the toilet seat and scrotum diminishes. It is conceivable based on the dynamic finite element model that the plaintiff's body might have crashed down after being lifted slightly, and thereafter squashed or crushed his scrotum on the toilet seat before he finally fell to the floor.

387. One way to demonstrate the effects of these unknown parameters is to treat the plaintiff as having fallen from a lower torso angle position to compensate for these unknown parameters (which I shall call 'interventions'). Simulating lower torso angles indirectly allows the finite element computation of the fall dynamics to factor in all the interventions. To enable me to have some understanding of the trajectories when these unknown parameters are taken into consideration, I therefore directed A/Prof Lim to perform further computer simulations for lower angles from 50 down to 30 . In other words, an 80 fall, after factoring all the interventions, may be approximated by a 50 simulation. Similarly, a 70 fall after accounting for the effects from all the interventions may be represented by a 40 simulation. A fall from 70 with the interventions taken into consideration may be the equivalent of a simulated fall from an assumed torso angle of 30.

i. Interventions

388. I shall now elaborate on these interventions, which collectively would reduce the angular velocities, angular momentum and rate of fall of the collapsing torso:

> (a) Mrs Harte's attempt to stop Mr Harte's fall in my opinion is a significant intervention. She managed to catch him at his chest to prevent him from falling. She must have caught him fairly early in the fall because it was not likely for her to catch him at his chest level if Mr Harte had already fallen close to his thighs. Further, she had some time to anticipate as she saw his eyeballs rolling and

thus, would have time to place herself in a position to grab him. Faced with that situation, I imagine Mrs Harte as a dutiful wife would have mustered all her strength to stop his fall. She might even have used her body to push him back. Certainly, it could not be a half-hearted attempt. Being a well-built Caucasian woman, I think she would be quite strong. Her considerable intervention slowed down the rate of fall and hence, the ability of the torso to pick up momentum. The energy in the falling torso, manifested in its linear and angular velocities, would be correspondingly reduced. Not satisfied with merely a qualitative reasoning (which I accept can be erroneous), I looked for some confirmation by some quantitative analysis and modelling. For this, I have asked for and I have the benefit of A/Prof Lim's programme written on Microsoft Excel (P55 file) computing (a) the potential energy released as a result of the fall of the heavy torso of Mr Harte for various fall angles, and (b) the energy reduction caused by Mrs Harte's intervention assuming a range of angles for her intervention at different levels of force applied by her to prevent the fall. After using the programme and going through the various possibilities (see P55A for the computations I have done using the programme and Annex A for the results), I estimate that it is likely that her intervention could have reduced the energy in Mr Harte's fall by between 30 to 50 %. I therefore conclude with a fair degree of confidence that Mrs Harte's intervention is a very significant factor in reducing the velocity and energy of Mr Harte's fall.

(b) The energy absorbed by the elastic tissues and muscles during the compression of the large stomach and the fleshy thighs of Mr Harte would also slow down the momentum of the fall. In a simulated fall impact experiment on trochanteric soft tissues, a total energy of 140 J was applied in each impact. The tissue energy absorption ranged from 8.4 to 81.6 J. (which corresponds to energy absorption of between 8% to 58%). Increased tissue thickness correlated strongly with increased tissue energy absorption capacity. See the paper on '*Force attenuation in trochanteric soft tissues during impact from a fall*' by J Orthop in the Journal of Orthopaedic Research Nov 1995. Since Mr Harte had substantial fatty padding around his stomach, pelvis and thighs, they must account for a fair amount of energy absorption.

(c) The back muscles are huge muscle groups with strong ligaments. They must be stretched when the torso bends during the fall. The skin of the back also stretches, including the intervertebral discs and tissues at the spine. Together they would reduce the energy and momentum of the fall and the impact of Mr Harte's torso on his thighs.

(d) Loss of consciousness is not instantaneous. The body gradually loses its muscle tone and then collapses. Consequently, the fall might actually start from a lower angle than what would ordinarily be Mr Harte's normal position when seated on a toilet bowl. The momentum generated from a fall from a lower angle is necessarily less.

(e) The rigid segment analysis ignores the fact that the plaintiff's body is made of multiple joints and a considerable amount of soft tissue padding. For a start, there are 26 joints alone between the head and the hip. But they are simulated by one rigid segment in the finite element programme. When a fall occurs, the 26 joints move together at different rates. The whole body of an unconscious man

is much more wobbly than a rigid segment of a few joints. There will be shifts of body weights and forces during the descent, which is quite unlike a rigid model.

ii. Inferences drawn from Computer Simulation

389. In my judgment, the above interventions will substantially reduce the momentum and energy of the falling torso. Hence, the so called 'lift' of the buttocks upwards in a circular arc about the knee and later about the ankle, as observed in the computer simulation for 80 to 60 is significantly reduced.

390. As an indication of the effects of such reductions, I studied in slow motion the fall characteristics as predicted by A/Prof Lim's dynamic finite element analysis. For fall angles of 50 to 30 , the simulation predicts that the scrotum will actually impact on the toilet seat for the 40 and 30 fall angles, whereas the available clearance (i.e. gap between the toilet seat and Mr Harte's perineum) in the case of the fall angle of 50 has become fairly small. These lower fall angles give a good indication of what the actual fall dynamics are going to be, when the fall energy has been reduced by the intervention of Mrs Harte, absorbed by the back muscles, abdomen, thighs and skin during stretching and so on. The sum total of these multiple interventions is that the 'dynamic lift effect' seen in the fall angles from 80 to 60 in fact may not be sufficient to lift Mr Harte's scrotum clear of the toilet seat to avoid any contusion. The common sense analysis is to that extent corroborated by the dynamic model of A/Prof Lim.

391. I am reinforced in my conclusion by the undisputed evidence below that the energy and momentum in the fall is not as great as that demonstrated for the fall angles from 80 to 60 and is in fact much closer to the other end of the spectrum represented by A/Prof Ng's static analysis:

(a) The final positions predicted by the dynamic analysis for 80 to 60 angles appear to be too far away from the toilet seat. The model predicts a lively forward movement that thrust Mr Harte's head so far forward as to be below the toilet sink with his buttocks a good half a metre away from the far edge of the toilet bowl. The final position of Mr Harte's buttocks as enacted by Mr Daniel Xu based on the evidence of nurse Tan Sang Eng (who tried her best to reconstruct the final position of Mr Harte after the fall in the toilet) shows that Mr Harte's buttocks were in fact much nearer the edge of the toilet seat. See photographs P40 A to C showing what would be the final position of Mr Harte viz a viz the toilet seat and the other wall structures in the toilet which were set up to scale in court. The toilet bowl was the actual one used by Mr Harte, which Gleneagles Hospital managed to preserve when the ward was renovated. The fact that the buttocks were fairly near the edge of the toilet seat suggests to me that the rate of fall must have been considerably slower than that predicted by the 80 to 60 fall angles. As expected, the fall angles of 80 to 60 divorced from all the effects of the abovementioned interventions have wrongly postulated the position where Mr Harte landed on the floor. The true position is in my view more realistically predicted by fall angles of 50 to 30 , which do take account of the effects of the interventions and which give results approaching that of A/Prof Ng's static model analysis. In short, after taking everything into consideration, I infer from the finite element modelling done by A/Prof Lim that there was a high probability of a contusion of the scrotum on the edge of the toilet seat rather than a trajectory that allowed an unconscious Mr Harte to 'fly over' the edge of the toilet seat in the course of the fall. An unconscious man cannot be expected to use his leg muscles to lift himself and hence, lift his scrotum clear of and away from the toilet seat to avoid any impact or contact with it.

(b) Most critically, if indeed the momentum and velocities of the fall were as great as predicted by the model for the fall angles of 80 to 60 with his head hitting the floor first, most probably Mr Harte's skull would have been cracked. However he escaped with a small reddish minor bruise and no swelling on his forehead, which later turned into a slight bluish bruise also with no swelling, and thus required no treatment whatsoever. This strongly suggests that his was a very low velocity impact. He did not crash onto the hard tiled floor in the toilet. It further suggests a '**crumbling**' fall and a slow sliding off the toilet seat. This minor injury thus falls into its proper place. It is obvious to me that the 80 to 60 finite element modelling gives a wrong result and must be rejected. Based on the evidence of the minor bruising on the forehead, I infer that the appropriate model should be much closer to the 30 to 50 finite element models, where the effects of the interventions are not ignored. Not surprisingly, A/Prof Lim's model, though it is only a guide, has predicted a contusion of the scrotum on the toilet seat, which is exactly what I think is likely to have happened.

iii. Position of scrotum viz a viz the toilet seat

392. Mr Hart was indeed very cooperative and had allowed himself to be photographed without his clothes on and seated on the very same toilet seat from which he fell. With Mr Harte seated on the toilet seat, there was hardly any gap between his penis and the inner edge of the toilet seat. This is understandable as the actual toilet seat he used in Gleneagles Hospital was small relative to his large size and build. Photograph D 51B shows the position of his penis when Mr Harte was seated normally on the toilet seat. It was estimated from the various scans taken that his scrotum would be hanging down some 8 cm or more beneath the toilet seat after bending over fully with his torso touching his thighs, which would have been the final position from which he fell off the toilet seat. In that position, his scrotum should be most vulnerable as it would be drooping deepest beneath the level of the toilet seat. Photographs D51D and D51E give an idea of the extent by which the plaintiff's scrotum was hanging.

393. Consideration must be given to the fact that Mr Harte's atrophied testes were already much smaller and presumably lighter in weight at the time the photographs were taken. Common sense tells me that his scrotum just before the fall, containing two much larger and heavier testes and aggravated by the usual swelling after a varicocelectomy, would necessarily droop even deeper into the toilet bowl. Obviously, a larger scrotum hanging lower beneath the toilet seat is far less likely to escape contusion in a fall off a toilet seat in an unconscious state when the legs no longer retained any muscle control to lift the body, and consequently also the scrotum safely clear of the toilet seat.

394. Since Mr Harte fell from a most vulnerable position when his slightly swollen scrotum after the operation was hanging deepest in the toilet bowl, the forward movement of the body during the fall would push his penis and scrotum to be jammed between the perineum and pelvic bone on the top, and the hard but smooth upper surface of the toilet seat at the bottom, whilst hemmed in at the same time by the inner thighs on both sides. The resultant effect is a fairly hard squeezing of the testicles on all sides for a short duration when his heavy body moved forward and fell eventually, pulling along with it and crushing the scrotum and testes as they passed over the smooth edged toilet seat, which consequently resulted in the contusion injury. A close analogy is the extraction of sugar cane juice by pulling sugar cane between two horizontal rollers set at a gap smaller than the thickness of the sugar cane. The compression of the sugar cane between the rollers causes the juice to be separated or squeezed out from the sugar cane fibres.

Conclusion

395. I fully sympathise with the condition of Mr Harte. It was a most unfortunate accident. Regrettably, I come to the

conclusion based on the evidence that the mechanics of Mr Harte's fall were in fact closer to that put forward by A/Prof Ng than that suggested by A/Prof Lim.

396. For all the reasons given, I find on a balance of probability that both testes suffered contusion injury from the fall. The contusion was not especially severe but it was sufficient to cause intratesticular microvascular damage and haemorrhage, which eventually resulted in patchy and non-homogeneous damage to the smaller testis on the left and much more severe damage to the larger testis on the right as would be expected when both testes were contused at the same time. This easily explains the bilateral nature of the damage which, had the cause been surgical in nature, would not be that readily explainable.

397. The contusion not only caused an intratesticular haematoma, it also led to an extratesticular haematoma, which developed slowly as there was similarly microvascular and microbleeding in the extratesticular tissues in the scrotum on both sides of the septum. As the scrotal swelling increased, it also pushed the scrotum to the size of a mango and added some more pressure on the already congested testes labouring under their own separate intratesticular haematomas. The combined pressure aggravated the whole situation, by forcing a serious but not total venous shutdown within the testes, which led unfortunately to the high degree of vascular and atrophic damage seen eventually on Mr Harte's testes.

398. I must record my appreciation to A/Prof Lim for his extremely helpful computer modelling and the short program written on the Microsoft Excel which enabled me to feed into it different parameters to study the overall effect of Mrs Harte's intervention. His dynamic analysis using the finite element modelling with data from Dempster's Tables helped me to better envisage the likely trajectories that an unconscious man falling off a toilet seat might take. With A/Prof Lim's simulation, I am reinforced in my view that on a balance of probability, it was the fall that contused and damaged his testes and not Dr Tan's surgery.

399. Exhibit P55 has a Zip Drive/CD-ROM containing the computer simulation results. It also contains the Microsoft Excel Program to compute the energy input from Mrs Harte for various degrees of intervention by her.

Statistical analysis

400. My conclusion also matches up with my statistical analysis, which I shall elaborate below.

401. If indeed the operation by Dr Tan had caused the disastrous results, that would represent 2 negligent operations (because he botched up twice, one on the left varicocelectomy and the other on the right) out of 200 previously successful varicocele operations on other patients or an estimated average of 1/100 chance of negligence. Dr Tan, a qualified and practising surgeon of many years, had in fact performed other far more complicated operations without any reports of negligence. For the purpose of this analysis, I shall ignore them. (If these other operations are included, the average statistical probability of negligence on the part of Dr Tan will be further diminished.)

402. Clearly, the bilateral varicocelectomy operation on Mr Harte comprised two separate and independent operations. To have botched up on both sides means that Dr Tan was negligent in two consecutive varicocele operations. Prima facie, that is statistically very remote because the mathematical probability is 1/100 multiplied by 1/100 giving a 1/10,000 chance of it happening consecutively. As Dr Notley very rightly conceded, it is rare to have infarction and resulting testicular atrophy after a varicocelectomy. It is even rarer for a bilateral testicular atrophy to take place after a bilateral varicocele operation.

403. Why must it be the case then that on the very rare occasion (1 out of 10,000) when Dr Tan had botched up his two operations consecutively, the patient was also found to have sustained a fall, an undisputed fact before me? Just by statistical probabilities alone, the fact that the plaintiff's fall had to coincide with an extremely remote case of an alleged consecutive negligence renders it statistically most unlikely that the operation was the cause of the bilateral testicular atrophy. It would be far more likely for the fall to have caused the atrophy.

404. This statistical evaluation based on pure probabilities and statistical reasoning is certainly evidence that has some

probative value and is part of the overall circumstantial evidence from which one may infer that it was the fall and not the operation that had caused the damage. Even without support from this statistical analysis, I am already satisfied on a balance of probability that the fall was the cause of the atrophy.

405. From the totality of the evidence in this case, the plaintiff has failed to discharge the requisite burden placed on him by law to prove on a balance of probability that Dr Tan had not discharged his duty of care to Mr Harte in his operation. The legal burden of proof remains always with the plaintiff. It is not for the 1st defendant to satisfy the court that he was not negligent. The 1st defendant only has a 'persuasive burden' to ensure that the balance of probability is not tilted against him.

406. However, Dr Tan adduced so much evidence that he managed to tilt the balance against the plaintiff and establish on a balance of probability that the fall did cause the resultant damage.

407. If I am wrong in this finding, I continue to hold the view that Dr Tan has furnished more than sufficient evidence to discharge his persuasive burden and rebut the plaintiff's case sufficiently to the extent that the plaintiff has not been able to prove to my satisfaction that Dr Tan was negligent in his operation.

408. The matter does not end here, as I have to be certain that Dr Tan was also fulfilling his duty of care to his patient after the surgery, which I shall refer to as his post-operative treatment, management and care of his patient.

Post-operative negligence

(a) 1st day after surgery.

409. Ms Magdalene Taye, the then clinic assistant employed by Dr Tan, averred in her affidavit of evidence-in-chief that she did not receive any telephone call from the plaintiff (a) complaining of scrotum pain or swelling, or (b) requesting for an early appointment. If she did, she would make sure that Dr Tan was informed. Nothing could be found in the clinical notes to indicate that Mr Harte had telephoned Dr Tan or the clinic. According to Ms Taye, it was the standard practice to write down telephone messages for Dr Tan and to provide Dr Tan with the case notes in relation to the caller. She was affirmative in her evidence that had she received any complaints from Mr Harte on the telephone, she would definitely let Dr Tan know. If there was anything else, she would definitely ask him to come back to see Dr Tan.

410. There was a staff nurse, Ms Louise Lee Hui San, who also worked for Dr Tan at that time. She was not called to testify. Ms Kamisah, another of Dr Tan's staff, also did not testify. It was not clear if they were the ones who received Mr Harte's telephone calls on the morning following the surgery when Mr Harte desperately tried to contact Dr Tan.

411. Dr Tan's case was that Mr Harte never called at all. I find that extremely hard to accept given the pain and swelling that Mr Harte was experiencing the morning after the surgery. I accept Mr Harte's evidence that he managed eventually to speak to Dr Tan about his post-operative condition.

412. When a doctor hears his patient making such complaints on the morning after the surgery, what must he do? A/Prof Foo answering for himself said that he would see the patient at the earliest convenient time. He might see the patient the next morning if it was not too urgent and the complaint was received after office hours.

413. A/Prof Foo opined that it was not reasonable post-operative care for a doctor to ignore the urgency and postpone seeing the patient after learning that his patient had complained of pain and scrotal swelling. He should see the patient. I agree entirely. Dr Tan should have asked Mr Harte to see him immediately. I see no good excuse for his failure.

414. Even if the telephone call from Mr Harte were to be received by a nurse and that message was not brought to the attention of the doctor, the doctor would still be vicariously responsible for the negligent act of his own nurse. The doctor must be

responsible for the management of his own clinic and ensure that there is a system where important messages are promptly brought to his immediate attention. Sometimes it may make the difference between life and death.

415. In my judgment, Dr Tan had failed in his duty of care when he did not arrange for Mr Harte to be examined as soon as possible, after having been told of the extent of his unusual pain and swelling. Dr Tan was clearly negligent in his post-operative care of Mr Harte. Even if he did not receive the messages from Mr Harte because none of his nurses brought them to his attention, he would still be vicariously liable nonetheless.

(b) 4th day after surgery

416. It is undisputed that the swelling was severe, whether one goes by Mr Harte's description of the size of a mango or that of a grapefruit by Dr Tan. That severity of swelling must be of immediate concern and was certainly abnormal for a bilateral subinguinal operation.

417. Dr Tan testified that he could not palpate the testicles through the swollen scrotum on 2 May 1997, which was the 1st consultation 4 days after the surgery. Why did he not use the ultrasound machine in his own office or the more sophisticated hospital CDUS facilities to investigate the condition of Mr Harte's testicles? This was very puzzling. Instead he prescribed more medicine and sent Mr Harte home after reassuring him that the swelling would subside. Strangely, he used his own ultrasound machine to examine the plaintiff's scrotum several days later on 9 May 1997 after the swelling had subsided and the testes were palpable, when one would have expected him at least to have used his own ultrasound machine on 2 May 1997 for the very reason that Mr Harte's testes were not palpable then.

418. A/Prof Foo opined that it was **dangerous** to send the plaintiff home with some medication under those circumstances and that an ultrasound should have been done first. If the ultrasound results show a pair of big swollen testes, A/Prof Foo said, "*Yes, we might want to* ***explore and drain the haematoma****, your Honour .... To reduce the pressure on the testes*." In his opinion, it might be too late after 4 days, but some testicular tissue might still be saved. He would have immediately drained the testis by cutting the tunica, and let the blood and blood clots flow off. He would not have just prescribed medicine and sent him home. According to A/Prof Foo, this was the "***minimum standard he would expect of any other urologist in the same position***".

419. A/Prof Li also agreed that he would use an ultrasound machine to check the swollen and non-palpable scrotum. If done, I think the CDUS would probably show up a dangerous and severe intratesticular swelling in both testes requiring emergency treatment and drainage.

420. I would regard the views of A/Prof Foo and A/Prof Li as being representative of a body of responsible, reasonable and respectable urologists as to what ought to or should have been done. I believe what was said is also equally applicable in relation to the proper treatment that should have been given on 29 April 1997, had Dr Tan acted responsibly and examined Mr Harte on that very morning after the surgery when he received complaints of unusual pain and swelling.

421. An opinion that there is no necessity to explore and drain the haematoma after discovering the dangerous and fairly substantial intratesticular haematoma in both testes is to my mind not a reasonable or responsible one that any respectable and competent urologist could honestly hold. It does not make any sense not to try to do something to save the situation but let the dangerous and severe bilateral intratesticular haematoma take its normal course, with the foreseeable and likely result of bilateral atrophy if the dangerous intratesticular haematoma is not alleviated immediately. Whether or not, the whole set of testes can be saved is irrelevant. So long as there exists a reasonable chance of saving some portion of it, it justifies and mandates the operative step to surgically explore and drain the intratesticular haematomas, unless of course the patient himself, after being properly informed, decides for himself that there is no necessity for such an invasive operative procedure.

422. In a 1989 paper "*Blunt Scrotal Trauma: Emergency Department Evaluation and Management*", Munter and Faleski

stated that:

> Blunt scrotal trauma can cause significant injuries which if not correctly diagnosed and treated may result in profound morbidity, including loss of a testicle…. Ultrasonography recently has become widely reported as a useful modality in the evaluation of blunt scrotal trauma…

423. I would have thought therefore that any reasonably competent urologist should have realised the importance of using ultrasound to investigate a severe scrotal swelling particularly where the testes could not be palpated. It is a breach of his duty of care simply to assume that there would not be any intratesticular haematoma, and hence, dispense with an ultrasound. I would regard that omission as one which any responsible and reasonably competent urologist in the shoes of Dr Tan would not omit to do in the face of a scrotal swelling the size of a mango, with the testes not palpable. Clearly, it was a poor exercise of medical and clinical judgment, which I do not expect of a responsible and competent urologist.

424. When the testes were not palpable through the scrotal haematoma, there is all the more reason to order an ultrasound to check for intratesticular haematoma, which Dr Tan as an experienced and competent urologist should have known can be dangerous. His admission that he did palpate for the testicles to my mind indicates that he wanted to check for intratesticular haematoma. Otherwise, why palpate for the testicles in the first place? Dr Tan could not be heard to say that he did not at that time realise the importance of checking for intratesticular haematoma.

425. In my opinion, the clinical examination, investigation and treatment by Dr Tan on 2 May 1997 were clearly not up to the appropriate standard of post-operative treatment and care expected of a reasonably competent specialist urologist. I also observe that Dr Tan failed to record in his clinical notes the colour, shape or the size of the scrotal haematoma although he admitted that the size of the swelling, its colour, his inability to palpate the testicles, tenderness of the scrotum should all have been recorded. A lackadaisical attitude seems to have been taken by Dr Tan over contemporaneous recording of important and significant details of clinical observations and findings. I note also the extremely brief operation notes kept by Dr Tan. Reliance on pure memory for such matters is certainly not good medical practice.

<u>Record of 'No pain'</u>

426. Looking at the drugs prescribed, Dr Tan appears to have given the plaintiff strong drugs to resolve the inflammation. Dr Tan was questioned why he had prescribed painkillers (i.e. Voltaren) when he asserted that the plaintiff had not experienced any pain, as stated in his entry 'No pain' in his clinical notes dated 2 May 1997. Interestingly in his affidavit of evidence-in-chief, Dr Tan left out mentioning Voltaren from the list of medication he prescribed on 2 May 1997.

427. Dr Tan maintained that he gave Voltaren for inflammation, thereby suggesting that it was not prescribed because of the pain. When confronted with the fact that he also prescribed Danzen, which is an anti-inflammatory drug, he explained that he was using a 'combination' of anti-inflammatory drugs.

428. As a result of some exchange of letters between the plaintiff's and 1st defendant's solicitors over the question whether the words 'No pain' in his clinical notes for 2 May 1997 were written contemporaneously because of the different ink colour, Dr Tan conceded that the entry was likely to be filled in later but he could not remember exactly when. Amazingly, Dr Tan said that it was even possible that he could have inserted the words 'No pain' just before he submitted his report to the Singapore Medical Council but this would be about 1 year and 3 to 4 months after his medical examination on 2 May 1997!

429. If so, this must have been inserted as an afterthought. It could hardly be regarded as anything close to a contemporaneous record of his clinical findings on 2 May 1997.

430. When his counsel asked him whether the plaintiff suffered any pain on 2 May 1997, his evidence was:

A: Under oath, I cannot be sure that he specifically said that he was not in pain. But I am sure that he did not say that he was in pain.

…..

Ct : Did you ask Mr Harte if he felt any pain on 2 May '97?

A : I believe I did.

Ct : What was his answer?

A : He must have said that it was not severe or nothing much at all. The reason I say this is that as I mentioned earlier that the unusual things or the abnormal things, or the specific things, or points raised to me, are things which I would record as soon as I can. In general, normal things or non-events, are not recorded, and this you can see from the rest of the notes which generally do not record normal findings.

Ct : So therefore 'No pain' is not an abnormal finding?

A : 'No pain' is what I consider a non-event. … 'No pain' is a finding in relation to the haematoma, which should have been put in, including other details like size, colour, any inability to palpate the testicles through the haematoma and whether it was tender to palpate and the ensuing discussion on what to do next and the treatment plan for haematoma. All this in retrospect should have been included.

431. If it was a non-event, then why did he take all the trouble later to insert 'No pain' to his clinical notes subsequently? If he felt that the size, colour and inability to palpate as well as 'No pain' should have been put in because they were all in relation to the haematoma, it is remarkable that he should decide to insert only the words 'No pain' in his clinical notes but leave out the rest of the relevant details of the haematoma. Clearly, it was selective insertion of relevant details by Dr Tan at work here.

432. What is most telling was the way he tried to explain away the rather unusual phenomenon of swelling without pain. He said that in his experience, he had seen several scrotal haematomas following operations like herniagraphy, vasectomy, orchidectomy, varicocelectomy where there is usually no pain in spite of the scrotal haematoma because these haematomas stretch the scrotal skin and they look more impressive than they are actually painful. His diagnosis at that time was that Mr Harte had a scrotal haematoma but he did not suspect that he had intratesticular haematoma. Dr Tan then said:

A: … In retrospect, after asking myself what happened to him, I believe he had both types of haematoma [i.e. (a) scrotal haematoma or extratesticular haematoma and (b) intratesticular haematoma].

Ct : In which case would he have pain?

A :Yes. He should have felt pain for the intratesticular haematomas.

Ct : So I ask you again, did the patient answer you that he felt pain…., you did ask him the question if he felt any pain on 2 May '97?

A : I am relying on what I recollect on that day, and as I mentioned earlier I can remember him walking through the door, still able to joke about his condition – by saying he has got big balls. And as would be an automatic standard question, I

would have asked him about pain. And I remember that pain on that day was not an issue. All I can recall is that he did not indicate to me that he was in severe pain.

Ct : So he indicated to you that he was in some pain?

A : No. He allowed me to examine him and I palpated the haematoma, and it was not tender and I have no reason to suspect that what he had was nothing more than the normal scrotal haematoma.

Ct : For the last time, when you asked him whether he felt any pain, what was his answer?

A : His answer was 'No'.

Ct : So did you believe him?

A : I have no reason not to believe him.

433. During this part of his evidence, Dr Tan was particularly evasive. From the above evidence, I am inclined to believe that Dr Tan was not truthful in his testimony. Obviously, Mr Harte must be suffering from a substantial intratesticular haematoma in both testes in order to suffer from bilateral testicular atrophy in the first place. That intratesticular haematoma stretching the tunica albuginea must be painful for Mr Harte. It is inherently incredible that Mr Harte, upon being questioned by Dr Tan whether he felt any pain, would have answered the opposite of what he was feeling at that time. I find as a fact that Mr Harte had complained of pain in his scrotum to Dr Tan on 2 May 1997.

434. The fact that Voltaren, a painkiller, had been prescribed corroborates the plaintiff's evidence that he was in pain. In my judgment, Dr Tan had lied that the plaintiff was not in pain on 2 May 1997. The words 'No pain' were a fabrication inserted by Dr Tan to his clinical notes for reasons best known to himself. Plaintiff's counsel had submitted that the words were inserted just before Dr Tan's reply to the Singapore Medical Council to explain the mishap.

435. Because of this false insertion of 'No pain', Dr Tan had to tally up with his other story that he did not receive any phone calls from Mr Harte prior to 2nd May 1997 complaining of the pain and swelling. I did not believe Dr Tan that he had not received these calls. This brings me to my other finding that his failure to instruct Mr Harte to come immediately to see him upon hearing the plaintiff's painful condition and swelling, knowing full well that a surgery had been performed and that the substantial swelling was extremely unusual, was something that a responsible and reasonably competent urologist surgeon would not have done. Plainly, Dr Tan had not taken sufficient care by asking the plaintiff to come for a medical check-up and he was dismissive of what the plaintiff had told him. He was overly confident that his surgery had gone on well and hence, there was no need to call Mr Harte in to be seen immediately despite all the complaints by Mr Harte on the telephone to him on the day after the surgery.

436. Counsel for the plaintiff submitted that Dr Tan, having suspected that the operation had gone wrong, tried to cover up his tracks when he finally saw Mr Harte on the 4th day after the surgery. Counsel for the plaintiff mentioned several instances of Dr Tan's conduct from which a cover-up by Dr Tan could be inferred, but I do not find it necessary to deal specifically with them.

Trespass or battery

437. Having regard to my finding that the surgery was not the cause of the damage, I need not address the question of trespass and battery. In my opinion, this head of claim is unmeritorious and in any event, it was not pursued with any vigour by

counsel for the plaintiff. The plaintiff had in fact signed a 'Consent for operation' form for his bilateral varicocelectomy on 28 April 1997. Once there is consent in law, which I find there was for the operation, the operation itself cannot constitute trespass or battery.

Vicarious liability of the 2nd defendants

438. The plaintiff did not dispute that Dr Tan runs a private medical practice at his own clinic. He was never employed by the hospital. In the course of the trial, counsel for the plaintiff clarified that the vicarious liability of the 2nd defendants did not arise from employment but arose purely from an agency arrangement.

439. As I have found that Dr Tan's negligence during the surgery is not proven, the question of vicarious liability for the surgery performed in the hospital does not even arise. The plaintiff's claim against the hospital must necessarily fail. Since the hospital has been dragged in purely on that basis, it may well have an impact on the question of legal costs, in particular whether Dr Tan must bear any part of the hospital's costs in defending the plaintiff's suit. For this reason, I will state my finding on vicarious liability.

440. Without going into the detailed reasons, it suffices for me to say that Dr Tan was not an agent of the hospital. Neither had the hospital held out Dr Tan as its agent for the purpose of the surgery. The hospital merely supplies hospital facilities and certain nursing care services to independent surgeons (including Dr Tan) who are free to choose any particular hospital where they have admitting privileges to perform their surgery and treat their private patients. Gleneagles Hospital has no control over how these independent surgeons privately treat and manage their own patients, even when the hospital facilities are being used.

441. I agree with counsel for the 2nd defendants that the hospital is not vicariously liable to the plaintiff for any alleged negligent acts or omissions of Dr Tan, which have nothing to do with the hospital facilities and nursing care services provided. Mr Harte sought out and contracted with Dr Tan directly to treat his infertility and operate on him. He never sought those services from the hospital. Since the hospital never provided the services of a specialist surgeon to operate on Mr Harte, the hospital was not under any duty in law to see that the specialist surgeon engaged by Mr Harte himself had exercised due care. That appears to me to be determinative of the issue of vicarious liability. If indeed there was any alleged principal and agency relationship, the principal party treating, managing and operating on Mr Harte appears to me to be Dr Tan himself and not the hospital.

Assessment of Damages

442. First, I shall determine the damages on the assumption that Dr Tan is liable for all the damage suffered by Mr Harte. Second, I shall assess the chance, as best as I can, of Dr Tan saving some parts of his testes had he asked Mr Harte to see him immediately on being told of the unusual swelling and pain on 29 April 1997 and treated him appropriately. The damages will then be discounted accordingly on the basis that the degree of damage might have been far less catastrophic had Dr Tan not been negligent post-operatively.

(a) General Damages for pain and suffering

The general damages for pain and suffering are assessed as follows:

(a) Large haematoma untreated for 4 days	- $ 2000

(b) Complete loss of fertility for a 36 year old man who has been sub-fertile for 9 years and has no children yet	- $20,000
(c) Partial penile impotence and impaired libido	- $13,000
(d) Atrophy and shrinkage of both testicles	- $10,000
(e) Increased risk of liver cancer from long term testosterone therapy and an awareness of a shortened life span	- $5,000
TOTAL	- **S$50,000**

(b) Special Damages

Expenses already incurred may be divided into 4 categories:

(a) Medical expenses incurred before the operation

(b) Medical expenses incurred for the operation itself

(c) Local medical expenses incurred after the operation

(d) Overseas medical expenses incurred after the operation.

443. With my finding that Dr Tan was not negligent in his operation, the medical expenses under (a) and (b) cannot be allowed. From the evidence, it appears that Mr Harte was reimbursed for most of these expenses by his insurance company in New York.

444. Items (c) and (d) will be allowed in principle as they comprise expenses incurred after the post-operative negligence of Dr Tan. Since Mr Harte was on paid home leave, I consider it reasonable to seek advice and a second opinion from the doctors in New York, and in particular Dr Dubin who had treated him previously.

445. However, these expenses must be properly proved. The legal burden is on the plaintiff to prove his special damages. But no supporting documentary evidence was tendered in court for most of his expenses listed in his statement of claim. It is also unclear whether he has obtained full reimbursement. He said '*I have to check all my records. The way health insurance works in U.S., not everything is paid 100%. I certainly submitted the bills in the form of health claims and I was certainly reimbursed for those costs.*' When probed further, the plaintiff clarified that he had received reimbursement of 30 to 50 % of his total medical bill because there were deductions. From the statement of special damages served pursuant to Order 18 Rule 12 (1A)(b) of the Rules of Court 1997, it appears that items (c) and (d) total S$ 4992.70 and US $4982.08.

446. Mr Lek for the 2nd defendants argued that where there was reimbursement of those medical expenses by Mr Harte's health insurance provided either by the company or the Health Care Plan in the USA, Mr Harte suffered no loss. As such, he could not submit a claim for damages in this suit as that would be making a double claim. I disagree.

i. Claims with reimbursement

447. A defendant tortfeasor has no valid defence to a claim of damages from an insured and reimbursed employee plaintiff. Such an insured and reimbursed employee plaintiff has all the rights intact against the defendant tortfeasor. The defendant tortfeasor's liability to pay the plaintiff is not affected by any reimbursement that the plaintiff has obtained from his employer or insurance. After the plaintiff has received payment of the damages, it is a matter between him and his employer or insurance whether the monies earlier paid by his employer or insurance are to be recovered. The right of subrogation is separate from the

issue before me whether the defendant tortfeasor must pay for the damages suffered by the plaintiff. It need not concern the court here.

448. Medical expenses or health insurance policies are normally regarded as indemnity policies, and thus attract the right of subrogation. Subrogation applies to all insurance contracts, which are contracts of indemnity. The doctrine of subrogation is of course, that the insured cannot make a profit from his loss and that for any profit he does make, he is accountable in equity to his insurer.

449. In *Napier v Hunter* [1993] 2 WLR 42 the House of Lords was unanimous in holding that insurers have an equitable interest in money received by the insured. The opinions contained reviews of the history of the doctrine of subrogation, which indicate that at the very least, it developed in equity as well as at common law. The principles establish that the insurers have an enforceable equitable interest in the damages payable by the tortfeasor.

450. The equitable interest of the insurers is to be satisfied by saying that they have a lien or charge over the money in question, rather than by saying that the money was impressed with a trust. Thus, if the insured who has received the money goes bankrupt, the insurers can recover the money in priority to the claims of other unsecured creditors; *Re Miller, Gibb & Co* [1957] 1 WLR 703, approved by the House of Lords in *Napier v Hunter*.

451. As there is a clear listing of the post-operative medical expenses with full particulars given (see statement of special damages), I do not think that there can be much dispute that they were in fact incurred although the supporting receipts were not produced. I do not think that I should penalise the plaintiff and disallow his claim here simply because he had not produced the receipts perhaps because they were given to the insurance company or his company to obtain reimbursement, and hence were no longer available for production before me.

452. I therefore allow in full the expenses under items (c) and (d) incurred after the operation, which total **S$ 4,992.70** **and** **US $4,982.08** respectively.

ii. Air Fares for overseas treatment

453. No loss was suffered under items 1.3.1, 1.3.2 and 1.5.1 in the statement of special damages because the plaintiff never incurred any expenses for the return airfares. His employer paid for the airfares between New York and Singapore for his April and October 1998 trips, including his third trip in 1999, where he took advantage of his home leave and a business trip to London, to seek medical consultation in New York. These return trips were part of his perquisites under his employment contract and were consumed principally either as his home leave or in the course of work for the company, and not because of the medical treatment which I regard as merely incidental. Clearly, medical consultation was not the primary reason for those trips back home to New York. I therefore disallow Mr Harte's claim for airfares amounting to a total of US$23,100, which should not be mounted at all as they stand on a completely different footing from the claim for his post-operative medical expenses for which he had been partially reimbursed.

(c) Future Expenses

I classify the future expenses into the following:

> (a) Cost of fertility treatment comprising two procedures viz TESE (testicular sperm extraction) and ICSI (intracytoplasmic sperm injection) at Cornell Medical Centre, New York. Microdissection TESE is the surgical removal of sperm from the testes or epididymis under optical magnification. ICSI encompasses the removal

of ova (eggs) from the wife and then the laboratory injection of sperm into each ovum for fertilisation.

(b) Loss of earnings having to return to a lower paying job in New York because of the fertility treatment.

(c) Cost of hormone replacement therapy

454. I accept the principle that the plaintiff is not allowed to claim any expense that would have been incurred regardless of the tort of negligence committed against him: *Cutler v Vauxhall Motors Ltd* [1970] 2 All ER 56. The plaintiff there was not entitled to recover damages for the surgery, as there was a reasonable probability that even if he had not suffered the accident, he would not have avoided the surgery for the severe varicose veins present in his legs even before the accident. The court is therefore required to have regard to contingencies and their probabilities when assessing the future expenses flowing from the tortuous act.

i. Cost of fertility treatment

455. The plaintiff had a pre-existing condition of poor sperm quality even before he consulted Dr Tan. Despite trying for 3 years, the wife could not conceive naturally. By then, she was 36 years of age. Apparently the poor fertility of Mr Harte was compounded by some medical problems on Mrs Harte's part. She herself had a small cyst in her left ovary and it was explained to her by Dr Jones that she would have irregular menses.

456. Dr Dubin himself advised the couple to consider IVF should his left varicocelectomy prove to be unsuccessful. Dr Dubin's left varicocelectomy operation did not help much. After the atrophy, Dr Dubin advised Mr Harte in April 1998 to consider other options like donor insemination or adoption. He repeated that advice on 27 October 1999. These to me are more realistic options.

457. A/Prof Li Man Kay testified that ICSI - 'Intracytoplasmic Sperm Insertion' would have been the recommended method of fertility treatment for the couple, **even before** the bilateral varicocelectomy because the plaintiff had weak sperm and the chances that his sperm would be able to penetrate the egg would be very low. Artificial insemination by injection of sperm into the vagina and uterus and the test-tube baby method (where the egg would be mixed with the sperms in a test-tube) were both not viable in Mr Harte's case.

458. Dr Schlegel said that he would have recommended intrauterine insemination or in vitro fertilisation (although he later said that he would only have recommended sperm washing with medication for stimulation of the female partner to produce eggs). This procedure of sperm washing and stimulation of the woman would cost between US$300 to US$2000. It would take typically 2 to 3 attempts for a couple to achieve a pregnancy, therefore costing a total between US$600 to US$9,000. For in vitro fertilisation, Dr Schlegel said that the cost would be approximately US $13,000 per attempt. Whereas in the case of the TESE and ICSI, the total cost for each attempt is US$28,000, of which US$16,000 is for the ICSI and all portions of the in vitro fertilisation, US$4,000 is for hospital fees, $1,000 for anaesthesia and approximately US$7,000 is for testicular sperm extraction or the TESE component.

459. Dr Notley also said that if he had been Mr Harte, he would have tried intracytoplasmic sperm injection (i.e. ICSI).

460. Based on the above evidence, it is most probable that Mrs Harte will have to undergo an ICSI procedure in any case. Even with sperm washing, his weak sperms will not likely fertilize his wife's eggs. His inability all these years to impregnate his wife is testimony to the fact that his sperms simply have no ability to do what they are supposed to do. Any concentration by centrifuging the sperms and making them more concentrated is not going to make much difference in my opinion. Hence, the ICSI procedure will still be needed even if Mr Harte did not suffer any testicular atrophy.

461. Hence, based on the principle stated in *Cutler v Vauxhall Motors Ltd,* I do not think that the cost of his wife's ICSI procedure should be included as part of the damages as it is not an expense that could be avoided at all.

462. As Mr Harte has returned to his hometown in New York, it seems reasonable that he seeks treatment from Dr Schlegel, who now advocates the microdissection TESE and ICSI procedure. There is some scepticism whether the microdissection procedure is more effective than the standard TESE/ICSI procedure. However, the couple firmly believes that Mr Harte's condition necessitates the special treatment by Dr Schlegel. I do not think they can be faulted if they pin their hopes desperately on him. Given these circumstances, the comparatively cheaper cost of alternative or similar treatment in Singapore is beside the point.

463. Dr Fong Yang had testified that frozen sperm could be stored indefinitely without any degradation in the sperm quality. This also avoids the problem of degradation of sperm quality with age and the risk of multiple testicular operations to remove testicular tissue to obtain fresh sperm each time.

464. In the paper '*Physiological consequences of testicular sperm extraction'* by Peter Schlegel and Li-Ming Su, the risk of permanent devascularisation of the testicle whenever a TESE surgery is performed is recognised. The authors said:

> In addition to the transient effects of a biopsy on spermatogenesis, permanent devascularization of testis may occur after TESE procedures. …we postulate that multiple incisions in the tunica albuginea used to retrieve spermatozoa may result in interruption of a sufficient proportion of testicular arteries to devascularize the testis. Therefore, **avoiding multiple incisions in the tunica albuginea as well as minimizing repeated TESE attempts is important to avoid the risk of permanent ischaemic testicular injury from TESE**.
>
> …….
>
> We also recommend that at least 6 months elapse before a repeat TESE procedure is performed. **The banking of frozen testicular tissue or spermatozoa retrieved during TESE procedures is advised to decrease the need for repeat TESE attempts and minimize injury to the testis.**"

465. However, the plaintiff inflated his claim by computing damages based on 10 cycles of TESE/ICSI. In answer to the request for further and better particulars, the following reasons were given for requiring as many as 10 attempts:

> (a) The treatment involves the extraction of sperm material from the left testis for storage of the same and subsequent use of the same to fertilise an egg to be extracted from the plaintiff's wife.
>
> (b) Dr Schlegel and his medical team utilise a surgical technique that enables multiple attempts to be made at extracting sperm material from the left testis.
>
> a. Even though viable sperm material may be extracted successfully on each attempt, the said sperm material may not successfully fertilise the egg obtained from the plaintiff's wife. In such an instance, the extraction process will have to be repeated --
>
> b. Until a successful fertilisation takes place or
>
> c. Until no further sperm material can be found or

d. Until no further eggs can be extracted from the plaintiff's wife.

466. Nowhere was it stated that the 10 cycles were required because Mr and Mrs Harte wanted to have a big family because they liked children very much. To my mind, that evidence in court was an afterthought to bolster their evidence that 10 cycles were needed. With Mrs Harte already at 38 years of age, I believe that there is a limit to the number of pregnancies that she can safely have. There is also a risk that multiple incisions on the tunica albuginea as well as repeated TESE attempts may cause permanent ischaemic testicular injury.

467. Considerable difficulties lay ahead for the couple as shown by their first attempt in June 2000 in the Cornell University Centre for Reproductive Medicine and Infertility. Mrs Harte failed to produce sufficient mature eggs. She produced only 4 follicles (immature eggs). The whole procedure had to be called off. Whether this expensive method is going to work at all for Mr and Mrs Harte is unclear.

468. Dr Schlegel has testified that if he fails on his first attempt to find any sperm, he will not make any further attempts. Dr Dubin's own opinion is extremely guarded for the procedure advocated by Dr Goldstein and Dr Schlegel.

469. Given these problems, I am already very generous to allow 3 cycles of the invasive TESE procedure to obtain sperm for the ICSI procedure, which is as far as I can reasonably go. Unused sperms should be frozen for future use as stated by Dr Schlegel in his own paper instead of resorting to too many expensive TESE operations with their attendant and not inconsiderable risks.

470. I will use Dr Schlegel's estimate that the TESE component of his advocated procedure costs approximately US$7,000. 3 cycles thus add up to US$21,000 plus an estimate of US $4,500 for the use of frozen-thawed sperms for any attempts in between or thereafter. Since there will be further pain and suffering and inconvenience from these 3 operations, I award a further US$1,500 x 3 for Mr Harte's pain, suffering and inconvenience during the TESE operations. Total award here is thus **US$30,000.**

ii. Loss of Earnings

471. Mr Harte testified that he gave up his job in Singapore and returned to New York strictly for medical treatment. They decided to leave Singapore after they found out about Dr Schlegel and his team. After some research, they believed that they would have a reasonable chance of having children of their own using the microdissection TESE–ICSI method practised by Dr Schlegel. Clearly, Mr Harte wanted to portray an impression that he was forced to leave his well paid job behind and leave Singapore because they badly wanted to get treatment from the best medical centre for the TESE-ICSI procedure at Cornell University Medical Centre in order to have their children of their own. So they decided to return to New York.

472. Mr Lek rightly pointed out that the plaintiff did not act on any reliable medical advice before deciding to undergo fertility treatment in New York. The decision to leave Singapore was made **even before** any consultation with Dr Schlegel.

473. Mrs Harte testified that they decided to leave Singapore after they obtained the biopsy result of 21 October 1998 from the Cabrini Medical Centre. She did not relate that decision to the need to seek medical treatment in New York. In her affidavit, she said that the full extent of her husband's injury only became apparent in October 1997. It left her sorely depressed. She found herself crying uncontrollably every day without warning. She started to hate Singapore and anything associated with it. She rarely left her house. She also admitted that she wanted to leave Singapore. Her bad feelings about Singapore would have set in by September/October 1997. In fact, Dr Tan informed them that Mr Harte would not be able to father a child on 11 October 1997. Given her strong feelings against staying on in Singapore and that the plaintiff was a person who would respect and give consideration to her wishes, it is not likely in my opinion that the plaintiff would have extended his contract to stay on in Singapore.

474. In all probability, the plaintiff and his wife had already decided to leave Singapore because they felt that Dr Tan's surgery was solely responsible for the bilateral atrophy. As a consequence of my finding, the basis for that belief has turned out to be erroneous. Should I award damages founded on a course of action flowing from the wrong basis? Perhaps I should not.

475. Most importantly, they decided to leave Singapore even before consulting Dr Schlegel. They had not even planned or decided how they were going to afford the fertility treatments.

476. The documents of E D & F Man show that the plaintiff was 'assigned' or 'seconded' by his employers, E D &F Man Inc, to E D & F Man Cocoa (Singapore) Pte Ltd for only a period of 3 years commencing on 1 August 1996 and after which period, it was stipulated that the Plaintiff would have to return to the US. Mr Trevor Johnson could not recall if there was any documentation showing that the plaintiff was to be employed in Singapore for longer than 3 years.

477. Mr Johnson confirmed that he never made any formal recommendation to the management committee of the cocoa division that the plaintiff was to succeed him.

478. If as the plaintiff claimed that they 'only decided to leave Singapore after [they] found out about Dr Schlegel and his team', this would have been at the earliest sometime after Dr Dubin's letter to the plaintiff dated 27 October 1999, wherein Dr Dubin suggested that Mr Harte might wish to consult with Drs. Goldstein and Schlegel at the New York Hospital, who had reported being able to remove sperm such as his and together with ICSI, achieve embryos and pregnancies.

479. There should have been some documentary evidence in E D & F Man of an extension of the plaintiff's contract to stay on but there was none. His 3 year term ended on 31 July 1999. Instead, Mr Johnson said that he had already recruited the plaintiff's replacement (Mr Paul Hutchinson) from London, who joined E D & F Man (Singapore) in July 1999, which meant that he would have looked for a replacement even before that date. The plaintiff was obviously lying in this regard.

480. At the end of his 3 years' secondment (plus a few more months to help his successor acquaint himself with his job), the plaintiff left for home as was the arrangement. A Form IR21 dated 15 November 1999, which E D & F Man Asia Pte Ltd submitted to the Comptroller of Income Tax (P12), stated the "Reason for Cessation/Departure" as "CONTRACT EXPIRED". It is pertinent to note that his employer E D & F Man was also having some ownership changes and restructuring at that time. The divestment exercise of the agricultural part of the business resulted in some headcount reduction in the sugar trading office in Singapore although Mr Harte maintained that there was no reduction in the cocoa trading office where he worked.

481. If indeed his company wanted to extend his contract, he could easily have stayed on in Singapore and still have his TESE done in New York. I do not think his work would suffer on account of perhaps a short trip of about a week a year to New York just for his TESE. It could also be timed to coincide with his home leave, which could be extended by a week if Mr Harte were to be so calculative about his leave. Given the speed and ready availability of air travel today, I do not think that it would be so inconvenient to undergo day surgery treatment in New York for his part of the TESE, and thereafter to fly back.

482. Clearly the fertility treatment sought in New York would not require him and his wife to be stationed permanently in New York. Dr Fong Yang and A/Prof Li confirmed that both the TESE and ICSI are day surgeries. The male patient's presence is required for only 1 day and the female patient is required for a period of 3 to 4 days according to Dr Fong Yang. The pre-operative medicine obviously can be taken in Singapore if Mr Harte were to be based in Singapore.

483. According to Dr Schlegel, the plaintiff would be required in New York for only about a week (of which he would have to be available one or two days beforehand because of the uncertainty of the timing of the ovulation, then he would stay in hospital for approximately one day for the actual TESE procedure and another three or four days for convalescence). Mrs Harte on the other hand would have to be in New York for about 3 to 4 weeks for the ICSI procedure. She would be treated with hormones for about 2 weeks and monitored for multiple egg production. After retrieval of her eggs, the sperm extracted from Mr Harte using the TESE procedure would then be injected into them. 3 days later, the fertilised embryos would be transferred back to her and she would need to stay in the hospital for several days to a week for additional monitoring. All this would add up to about 3 to 4 weeks in New York for her. But Mrs Harte, who was not working in Singapore at that time, should have no difficulty

staying 3 to 4 weeks in New York if the need arose. I did not consider the time needed for the TESE-ICSI procedure to be so extravagant that Mr Harte could not hold his job in Singapore whilst undergoing this fertility treatment by Dr Schlegel in New York.

484. Given the risks of the procedure, Dr Schlegel further said that the procedure should only be repeated not more than once every 6 months. Since I am allowing only 3 procedures as a reasonable number, it is certainly not going to be disruptive at all. Surely the day surgery TESE procedure for Mr Harte can be fitted within his annual home leave of 25 days per year (and extended if necessary) if he really wants to continue working in Singapore.

485. I do not believe at all that the need for fertility treatment by Dr Schlegel was his sole or main reason to return to New York. He has not proved on a balance of probability that he would have been able to stay on in Singapore for the long term but for his need for fertility treatment in New York.

486. In conclusion, I cannot accept that the TESE treatment at Cornell would be so disruptive that Mr Harte is forced to give up his job in Singapore. I do not find any cogent reason to justify the drastic measure by Mr Harte of leaving a well paid job in Singapore for a much lower paying job in New York. Some inconvenience there may be, having to go to New York each time for the TESE. But that is not a sufficient reason. The plaintiff always has a duty to mitigate his loss. Instead of trying to mitigate the loss, the plaintiff here increased his alleged loss without good reason, if indeed the decision to resign from his position in Singapore was wholly his (which I do not believe was the case in any event), and not because his services in Singapore were no longer needed by the company after the expiry of his contract (which I believe was more likely the case).

487. In fact, I regard his resignation to take up a lower paying job in New York so that he could go for a few day surgeries (spread out perhaps over 2 or 3 years) as most unreasonable. I cannot allow his loss of salary to be borne by the 1st defendant. The plaintiff has to live with his own decision.

488. I thus reject Mr Harte's claim for his alleged loss of earning amounting to S$2.4 million.

iii. Cost of hormone replacement therapy

489. Mr Harte further claimed the cost of hormone replacement therapy in New York at US$525 per month for 45 years based on a projected life span of 78 years. Dr Futterwie did not testify to these expenses, which the plaintiff computed at US$283,500.

490. The cost of US$525 includes consultation and blood test every month, presumably to monitor for any development of liver cancer as a result of the hormone therapy. The actual cost of the 'testoderm' drug is US$118.49 per month.

491. Dr Jimmy Beng stated that Mr Harte would have to be closely monitored with liver function tests but there was no hard and fast rule. In my opinion, that monthly consultation (US$246.60 per consultation) and blood test (US$160 per test) is extravagant. Two blood tests and consultations a year will be more than sufficient for close monitoring having regard to the low incidence of liver cancer from such therapy. Therefore, I allow a total cost of US$1,828.48 p.a. under this head of claim.

492. Mr Lek highlighted Dr Lei Chang Moh's testimony that some men around the ages of 50 and 60 might require hormone replacement therapy in any event.

493. Taking that into account together with the other contingencies of life (e.g. death though illness and accidents), it may be appropriate to compute on the basis of therapy up to 60 years. Capitalising the US$1828.48 p.a. expenditure running for a 24 year period (as Mr Harte is presently 36 years of age) at a discount rate of 5% p.a. (based on an estimate of future long term bank interest rates in the United States), the lump sum payment when mathematically computed comes to US$25,230, which I shall round up to **US$26,000.** If one wants to look at the calculation from the 'multiplier' basis, then in effect the multiplier is 14.2 if the lump sum is US$26,000. One cannot use a straight multiplication (based on 24 x US$1,828.48 = US$43,883) because that fails

to take account of the accelerated payment of a lump sum at present value.

(d) Likelihood of testicular salvage if treatment had been prompt

494. It is extremely difficult to estimate the probability of testicular salvage if Dr Tan had seen Mr Harte promptly on the morning following the surgery.

495. Suppose Dr Tan had asked him to come by his clinic the next day after the surgery upon receipt of Mr Harte's telephone calls. Suppose Dr Tan ordered a CDUS immediately. It would still be several hours after 9.00 am on the following morning before the CDUS results could be obtained. Counsel for Dr Tan submitted that by then, more than 24 hours would have elapsed after the fall. The testicles would still have atrophied. Any post-operative negligence would not have resulted in more damage than would be the case without any post-operative negligence. Since no damage had arisen from Dr Tan's breach of his duty of care, no damage was payable. Hence, Dr Tan would not be liable for any post-operative negligence.

496. Dr Myinte Soe relied on the following:

> (a) Dr Schlegel testified that beyond 4 to 6 hours, permanent injury would set in for torsion injury. [My comment: As this was not with reference to contusion injury, I disregarded it.]
>
> (b) Dr Clarence Lei felt that the testicle would be dead in 2 hours with no blood supply. [My comment: This is not quite the same for contusion injury, where some blood supply may still be continuing after the contusion.]
>
> (c) Dr Tay Kah Phuan felt that if there was **severe** trauma causing tissue death, nothing much could be done. The tissue takes time to die but it will die. [My comment: Dr Tay appears to me to be much too pessimistic. In any event, his is based on severe trauma which is not so in our case of moderate trauma, where some tissues left uncontused might well be salvageable.]
>
> (d) Dr Notley also did not appear hopeful. He said that not much could be done to save the testes disrupted by contusion. He said that one could operate on the testes to reduce the damaging pressure buildup and then hope for the best. [My comment: There is at least some hope.]

497. Fournier, Laing and Mc Aninch in their paper "*Scrotal Ultrasonography and the Management of Testicular Trauma*" referred to a report by Del Villar, Ireland and Cass: Early exploration following trauma to the testicle. A failure rate of 45 % was reported for non-operative management based on a review of 20 years' experience. Additionally, 45 % of those patients in whom surgical exploration was delayed required removal of the testes as opposed to only 5 % of patients who underwent exploration within 3 days of the injury. The results of this study indicate that the views expressed by the abovementioned experts might be far too pessimistic. In the absence of other evidence and data, I can only use those results in that paper as a rough guide to assess the probable success rate in our case.

498. Prof Li provided an educated guess that there could be a 50% chance of saving Mr Harte's testes had he been managed from the day after the operation and perhaps with surgical exploration. Dr Tan could have immediately drained the scrotum to relieve the pressure from the extratesticular swelling, which was further compressing the already distended and taut testes. He could perhaps have gone further by cutting the tunica albuginea to release some of the pent up pressure built up within the testes from the intratesticular haematoma. Thus had he called Mr Harte to his clinic immediately on 29 April 1997 instead of delaying by 4 days to 2 May 1997, I think there would have been a fairly good chance of saving some amount of testicular

tissue. Mr Harte would probably not suffer the degree of damage he has now suffered due to lack of proper treatment and post-operative care from Dr Tan. With such a large swelling, time must surely be of the essence.

499. Under the circumstances, I will say that with prompt intervention by Dr Tan on the morning after the surgery, perhaps 60% of Mr Harte's testes may be saved from atrophy, bearing in mind that even today, Mr Harte's testes are not completely dead. Accordingly, I award him 60% of the damages computed above.

500. Since damage arising from Dr Tan's breach of his duty of post-operative care of Mr Harte has been established, the plaintiff has therefore succeeded in proving that Dr Tan has been negligent in his post-operative care. However, the plaintiff has failed to prove that Dr Tan was negligent in his bilateral varicocele operation.

Judgment Sums, Interest and Costs

501. The judgment sums are as follows:

(a) 60 % of General Damages of S$50,000 = S$30,000.00

(b) 60% of Special Damages of S$4,992.70 = S$ 2,995.62

(c) 60% of Special Damages of US$4,982.08 =US$ 2,989.25

(d) 60% of Future Fertility Treatment cost of US$30,000 =US$18,000.00

(e) 60% of Future Hormone Therapy cost of US$26,000 =US$15,600.00

502. The total judgment sum awarded is S$32,995.62 and US$36,589.25. In Singapore dollars, the total awarded is approximately S$96,660 using the exchange rate of S$1.74 to US$1.

503. Interest is awarded on the general and special damages at 3% p.a. for the amounts in Singapore dollars and at 5% p.a. for the amounts in US dollars from the date of writ to date of judgment. No interest is allowed on the sums for future loss. The usual statutory interest on judgment sums unpaid shall apply.

504. I will hear the parties on costs on the basis that the plaintiff's claim against Dr Tan is allowed but the plaintiff's claim against the hospital is dismissed.

Chan Seng Oon

Judicial Commissioner