H v. The Royal Alexandra Hospital for Children & Ors.

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Full text of decision below.

(1990) Aust Torts Reports ¶81-000

Supreme Court of New South Wales.

Judgment delivered 04 January 1990.

Torts — Negligence — Plaintiff haemophiliac dying of AIDS after administration by hospital of blood product supplied by blood bank and manufactured by blood bank or other manufacturer — Evidence — Product administered in March 1982 and again in September 1983 — Knowledge of risk at both times — Duty of care — Foreseeability — Standard of care — Foreseeability — Failure to screen blood donors to exclude homosexuals, etc. — Failure to withdraw products manufactured prior to implementation of screening — Manufacturer's and distributor's failure to warn doctors of risk — Hospital's failure to adopt alternative treatment not involving blood products, or to withhold treatment completely — Failure to use blood selected from small group of donors, e.g. family — Failure to prefer one blood product to another — Doctor's failure to warn patient of risk — Causation at law — Causation in fact — Remoteness.

The plaintiff had been diagnosed as a haemophiliac in 1980, when he was about six. His brother had previously been similarly diagnosed. Haemophilia was a deficiency of a coagulant factor, known as Factor VIII, in the blood and in the plaintiff's case it meant that his clotting activity was well below 50% of normal. When bleeding occurred, the only available treatment was the administration of anti-haemophilia factor ("AHF") therapy.

AHF was rich in Factor VHI and was isolated or extracted from human plasma. Prior to the development of Factor VHI in the 50s and 60s, a lot of haemophiliacs died from uncontrolled bleeding. AHF came in two forms.

There was a powdered concentrate, each batch of which was derived from the pooled plasma of hundreds or even thousands of donors, and was diluted in water before use. Many hundred or thousand bottles of concentrate were produced in a batch, each of equal consistency such that the amount of Factor VHI in each bottle could be determined.

There was also cryoprecipitate, each bag of which was manufactured from the plasma of a single donor. The Factor VIII content of a bag of cryoprecipitate could not be determined before use. A greater quantity of cryoprecipitate than concentrate was required to provide the same amount of Factor VHI, and where a large amount had to be given to a small person, such as a child, the concentrate was preferred.

One danger sign for Haemophiliacs was the swelling of joints, which indicated internal bleeding. If not treated swiftly the internal bleeding would cause pain and disability, and lead to painful degenerative arthritis in later life. In March 1982 the plaintiff had a swollen elbow, and in September 1983 he had a swollen knee. On both occasions he was taken to the first defendant ("the hospital") where AHF therapy was performed.

On the first occasion he was given six bottles of concentrate manufactured by the third defendant ("the blood bank"), two bottles of concentrate manufactured by the second defendant ("CSL") and eight bags of cryoprecipitate manufactured by the blood bank. In September 1983 he was given 18 bottles of concentrate manufactured by CSL. The blood bank supplied all of these products to the hospital.

From one of these treatments the plaintiff became infected with the human immuno-deficiency virus ("HIV") which led to the killer disease acquired immune deficiency syndrome ("AIDS"), from which he would soon die. The first symptoms of the disease appeared late in 1984 and the plaintiff's condition gradually declined. By Christmas 1988 he was very sick, and he had been in hospital repeatedly since then. During this time life was pretty miserable for the lad.

It had been well known for many years prior to 1980 that syphilis, malaria and hepatitis B were transmissible in blood and blood products. The test for detecting the presence of hepatitis B was not developed until 1973. It was later discovered that another form of hepatitis, designated "hepatitis non A/non B", was transmissible in blood and blood products but to date no test for detecting it had been developed.

By December 1981 the syndrome now known as AIDS was recognised as a new and fast-growing medical disorder apparently confined to male homosexuals. On 16 July 1982 came the first report of a similar syndrome, not among homosexuals, but among persons with haemophilia. The possibility that the agent causing the illness was transmitted through blood products was first suggested.

A meeting between scientists, doctors, the blood banking industry and haemophiliacs was convened by the U.S. Public Health Service in Washington D.C. on 26 July 1982. The name "AIDS" was determined and the transmissibility through blood products was canvassed, although no recommendations were made in relation to the use of blood products.

On 10 December 1982 an infant of 20 months died following multiple transfusions, where the blood product used was derived from the blood of a male subsequently found to have AIDS. There was still no direct laboratory test to identify the AIDS virus. The U.S. Centres for Disease Control convened a meeting on 4 January 1983 at which a majority of participants were convinced that there was at least a risk that an association existed between AIDS and the use of blood or blood products.

From late 1982, because AIDS had first appeared in homosexuals, American blood collection agencies began excluding homosexual and bisexual males from donating blood. On 24 March 1983 the director of the Office of Biologics in the U.S. Food and Drug Administration circularised licensed manufacturers of plasma derivatives requesting that they immediately institute procedures to assure that their suppliers had adopted appropriate donor screening practices and procedures to exclude persons with symptoms and signs suggestive of AIDS, sexually active homosexual or bisexual men, intravenous drug abusers and the sexual partners of the foregoing.

Some time after March 1983, Dr Archer of the blood bank in Sydney made a public request through the media that male homosexuals in Sydney not donate blood. He backed down after homosexuals cried discrimination and began picketing the premises of the Blood Transfusion Services in Sydney. Instead of further public announcements, it was decided that potential donors would be given written guidelines to facilitate self-exclusion. Such a document was issued in June.

On 2 July 1983 a group of Australian researchers reported that they knew of no instances of AIDS in haemophiliacs in Australia and concluded that it had not been scientifically established that AIDS was transmitted through blood products. They recommended the continued acceptance of AHF treatment.

In late 1982 and early 1983 the first patients in Australia with clinical manifestations of HIV infection (but not full-blown AIDS) were detected and reported, together with the first case of full-blown AIDS in April 1983. The first transfusion-transmitted case of AIDS in Australia was identified in Sydney in July 1984.

The suspected connection between AIDS and the use of blood or blood products remained unproven because of the inability to identify any virus which could be responsible for the disease. Throughout 1983 and into 1984 the U.S. National Hemophilia Association urged haemophiliacs to continue using AHF.

In 1983 a doctor at the Pasteur Institute in Paris, France, had identified the AIDS virus but was unable during that year to produce sufficient evidence to convince the scientific community (who are not renowned for accepting anything very quickly) that it was in fact the AIDS virus. This confirmation did not come until May 1984.

A test for detecting the presence of the AIDS virus in blood was not developed until the latter part of 1984, and this was licensed by the U.S. Food and Drug Administration for general use in May 1985.

The plaintiff sued the hospital, CSL and the blood bank in negligence.

Against CSL and the blood bank it was alleged: that they had failed to institute an appropriate system of excluding high risk donors; that they had failed to provide warnings of the relevant risk of injury; and that they had negligently distributed blood products derived from unscreened or inadequately screened donors.

Against the hospital it was alleged: that it had failed to insist on more rigorous screening of donors by CSL and the blood bank; that it had failed to recommend and administer alternative forms of treatment which did not involve the use of blood products; that it failed to withhold treatment completely; that it failed to warn the plaintiff's parents of the relevant risk of injury; and that it failed to use only "dedicated" or "designated" blood products, being those manufactured from plasma and blood taken from a select group of donors.

The plaintiff also alleged that the hospital had erred in diagnosing haemarthrosis (bleeding into a joint) on the two occasions in question, but the Judge concluded from the evidence that the plaintiff had failed to discharge his onus of proving that the diagnosis had been incorrect.

Also in issue was whether the plaintiff had been infected by the AHF treatments administered in March 1982, or by the treatments administered in September 1983.

Two different batches of concentrate had been administered to the plaintiff in the course of the treatments in March 1982, one of which had been administered to two other persons (including the plaintiff's brother) and the other of which had been administered to three other persons (including the plaintiff's brother), all in dosages equal to, or greatly exceeding, those given to the plaintiff. There was no evidence that any of these others were HIV positive and there was evidence that the plaintiff's brother was HIV negative.

A blood donor ("D-20") gave blood on 17 March 1982, part of which was made into the cryoprecipitate which was administered to the plaintiff in March 1982. D-20 had donated blood regularly. He donated again on 27 March 1982, and the only recipient of this donation who could be traced tested negative for AIDS. Of five donations on and after 16 June 1982, at least one recipient of each had become ill; four recipients had been diagnosed HIV positive and another two had died.

Held: for the defendants.

Duty of care

1. Each defendant owed the plaintiff a duty of care in relation to both the treatments administered in March 1982 and the treatments administered in September 1983.

Well before 1982 the risk of the transmission of blood borne viruses by blood or blood products, although unquantifiable, was known and foreseeable. The specific risk of infection by HIV did not have to have been foreseeable for the duty to arise.

2. The duty of care owed by CSL and the blood bank, as manufacturers and distributors of blood products intended to be infused into the veins of human beings, to recipients of their blood products was a duty to take such reasonable precautions (if any) as were available to protect them from infection by such means.

4. The duty owed by the hospital was a duty to take reasonable care for the plaintiff as its patient, inter alia in diagnosis, prescribing treatment, giving appropriate information and warnings about any risks associated with intended treatment, and in carrying out any treatment (including the selection and administration of treatment modalities, medications, etc.).

Liability in negligence

5. Of all the allegations of breach, only two were established, and only in relation to the treatments administered in September 1983, but the plaintiff failed on the issue of causation in relation to both: CSL and the blood bank unreasonably failed to warn the treating doctors of the risk of AIDS, but the doctors were aware of this risk; and the treating doctors failed to warn the plaintiff's parents of the risk of AIDS, but even if they had been warned AHF treatment would not have been refused.

Standard of care: foreseeability

6. On the information available prior to March 1982, there was a foreseeable risk that recipients of blood products would become infected with then unidentified and unknown blood borne viruses such as HTV. It was known that certain viruses could be transmitted in blood and blood products, hence the risk of infection by other viruses was neither far-fetched nor fanciful. The risk was slight or remote, but was not a risk that reasonable manufacturers of AHF would disregard, assuming reasonable means of averting the risk were available to them.

7. It could not be said that as at March 1982 any of the defendants ought to have foreseen and guarded against the risk that the plaintiff might, through Factor VIII therapy, become infected with the specific agent causing the outbreak of immune deficiency illness in homosexuals. Prior to July 1982 there were no grounds at all for even the faintest suspicion that a blood borne virus might be involved in the disease, and there was very limited information about the disease itself.

8. Each of the defendants was, from April 1983 onwards and therefore in September 1983, under a duty, not merely in general terms to guard against the transmission of unidentified infectious agents in blood and blood products, but specifically to take reasonable care to prevent injury to the recipients of blood products by the transmission of the agent which might cause AIDS.

Reasonably informed physicians, scientists and blood transfusion services in Australia ought to have been well aware by April 1983 at the latest that there was a real risk that among the unknown and unidentified sources of infection which blood and blood products had the capacity to carry must be numbered whatever agent was responsible for the production of AIDS.

CSL's and blood bank's failure to screen donors

9. Neither CSL nor the blood bank was negligent in respect of any failure to screen donors prior to March 1982 and therefore neither was negligent in continuing, in that period, to use plasma from an unscreened donor pool or to distribute AHF made from such plasma.

10. During that period it could not reasonably have been said that there was a group sufficiently clearly identified or identifiable as a high risk group in relation to unknown viruses to permit screening. The plaintiff failed to establish that there was any reasonable prospect that the screening of donors would have been effective to eliminate from the donor pool members of high risk groups or that the risk of infection from unknown viruses was such that a reasonable blood banker would have thought it necessary to attempt such exclusion at that time.

There were obvious practical difficulties in attempting to identify with certainty members of the very wide group or groups which would have included male homosexuals, intravenous drug abusers, certain ethnic or racial groups, prostitutes, and the sexual partners or clients of such people.

Where current symptoms of donors were not discernible by blood collection personnel, the effectiveness of the screening process depended on the honesty (which could not be assumed in all cases) and the awareness (which would not always be complete) of each potential donor. In *Jones* v. *Miles Laboratories Inc.* (1988) 700 F. Supp. 1127**, a homosexual who subsequently died of AIDS had on at least 25 occasions when giving blood lied to the collection agencies.**

The system of blood collection was totally voluntary, there was a continuing shortage of blood generally, and AHF specifically, and the group that would have to have been excluded was very wide.

11. Although it appeared with hindsight that if all homosexuals had been excluded from the donor pool prior to March 1982, the probable effect would have been to exclude most if not all persons likely to be carrying the AIDS virus. This was not and could not have been known at any time prior to March 1982.

12. In all the circumstances the screening procedures implemented prior to September 1983 were reasonable and the plaintiff failed to establish that they could reasonably have done more than they did by way of screening. Notwithstanding that there was some opinion in America that all homosexuals should be excluded, there was also responsible opinion for the view that the risk increased with the number of sexual partners, and that the high risk group comprised the promiscuous homosexuals.

CSL's and blood bank's failure to withdraw products made before screening procedures implemented

13. The plaintiff failed to adduce any evidence as to the date of manufacture of the blood products administered in September 1983, and the allegation (hat CSL and the blood bank ought to have withdrawn all products manufactured from blood donated prior to the implementation of screening measures therefore failed.

14. In any event it was not self-evident that a recall was a practical and reasonable measure in the circumstances. There was scarcely an adequate supply of blood generally and there was a constant struggle to maintain a sufficient supply of AHF. The transmissibility of AIDS by AHF, though suspected, had not been proved. No Australian haemophiliacs had contracted AIDS. And there had been a wide distribution of the products throughout New South Wales.

CSL's and blood bank's failure to warn

15. In relation to the treatments of both March 1982 and September 1983, the duty of care owed by CSL and the blood bank required them to warn the persons by whom their products might be used. Warnings were in fact given; the warning prior to March 1982 was adequate to discharge the duty of care then owed, but the warning was inadequate to discharge the duty owed as at September 1983. However, the treating doctors were aware of the information that an adequate warning would have conveyed and the plaintiff failed on the question of causation.

16. The duty as at March 1982 required a warning of the risk of infection with AHF products because by this time CSL and the blood bank clearly should have been, and were, aware that there existed a risk of hepatitis non A/non B and other sources of infection neither known nor identified. An appropriate warning at this time did not extend to the risk that blood products might carry the agent responsible for the syndrome recently reported as occurring in American homosexuals.

17. An appropriate warning was given prior to March 1982 and there was no breach of duty. This warning appropriately drew the attention of the treating doctor to the risk that the product might transmit the agent responsible for hepatitis. The fact that no express reference was made to the theoretical risk of transmission of unknown and unidentified viruses was in all the circumstances not unreasonable.

18. By September 1983 the duty then existing required CSL and the blood bank to provide a warning about the risk of AIDS. Their failure to do so amounted to a breach of duty. They could not reasonably have assumed that every physican by whom their product might come to be administered would be aware of the risk.

It was clear that from soon after the beginning of 1983 CSL and the blood bank were aware of the risk then becoming known, that AIDS might be a viral disease transmissible in blood products and in particular in Factor VHI preparations. That knowledge, with their inability to do much to eliminate the risk, cast upon them a duty to give an appropriate warning of that risk.

19. An appropriate warning should have been given to the treating doctors administering the blood product rather than to the patient. The duty did not extend to warning the actual patient; in so far as the patient was owed a duty to be warned of the risk, the duty was that of the treating doctors, and not of the manufacturer and distributor.

20. The plaintiff's treating doctors were aware of everything an appropriate warning would have conveyed, hence the failure by CSL and the blood bank to warn the treating doctors of the risk of AIDS was not causally related to the plaintiff's illness.

Hospital's failure to insist on screened blood

21. It followed from the finding that it had not been reasonably practicable for the blood bank to screen the donor pool (except to the extent that it did) so as to exclude so-called high risk sections of the community, that it had also not been reasonably practicable for the hospital to insist on only AHF manufactured from plasma derived from a donor pool more completely screened.

Hospital's failure to employ alternative treatment

22. The diagnosis of haemarthrosis, or joint bleed, was a correct diagnosis. There was no realistic treatment option other than the administration of AHF.

23. The hospital was not negligent in failing to prescribe the drug DDAVP in preference to AHF therapy, as it offered no prospect at all of achieving any therapeutically significant result. DDAVP was a hormone preparation which stimulated the body's production of Factor VIII, but did not increase the body's Factor VIII levels to anywhere near those necessary to treat the joint bleeds. In any event, in September 1983 licensing authorities in America and Australia were still in the process of testing the drug.

Hospital's failure to withhold treatment

24. The exercise of reasonable care on the part of the hospital and its staff did not require that AHF therapy be withheld, either in March 1982 or in September 1983. Once the joint bleeds had been correctly diagnosed, the plaintiff's condition required the administration of AHF therapy and no reasonable physician would have regarded the no treatment option as realistic, in the absence of some powerful reason to the contrary.

25. In March 1982 the known risk of hepatitis was far from a sufficient reason to withhold treatment, and the risk of the transmission of any other kind of infection then unknown did not add significantly to the risk side of the risk/benefit balance to which a clinician had to direct himself.

26. In September 1983 the plaintiff's doctors, conscious of the growing concern about AIDS but aware of the very low incidence of AIDS among recipients of blood and blood products, the continuing strong recommendation of the American National Hemophiliac Association, and the likely dire consequences of non-treatment, acted reasonably in not regarding the risk of AIDS as sufficient to warrant the withholding of AHF therapy.

Hospital's failure to use dedicated blood

27. The plaintiff failed to establish that the provision of "dedicated" or "designated" blood was a feasible option which, if employed, would have avoided the risk to him of acquisition of the HIV infection. It was not sufficient for the plaintiff merely to raise this possibility and expect the defendants to show that it was not feasible. The plaintiff did not provide even a scintilla of evidence in this respect.

28. If blood from select groups only were to be used, then the blood bank or hospital would have had to maintain a sufficient stock for the foreseeable needs of each patient. No specific evidence was adduced as to: the quantities that would have to have been maintained; the number of donors that would have to have been relied on; and the frequency with which those would have had to donate.

29. The plaintiff failed to establish that blood drawn from select groups would have been any safer than that which was administered.

It could not be assumed that the police force and armed services were lower risk groups. Family members who were homosexuals or intravenous drug users might have given blood so as not to arouse suspicion. It was unsound to draw blood from children under the age of 16. There was no evidence that females over the age of 16 were a lower risk group. Although blood collected from females could have been reserved for the manufacture of products capable of transmitting HIV, the plaintiff led no evidence as to: what was known in 1983 as to the capacity of various blood products to transmit AIDS or the certainty that some would not; what proportion of collected blood was in fact used for one purpose rather than another; and the effect on the overall blood supply of the reservation of female blood for those purposes. There was also evidence that in America select donor groups had been found to be no safer than broader community groups.

Hospital's failure to use only cryoprecipitate

30. If the plaintiff's infection resulted from the 1983 treatments, the hospital was not negligent in failing to use cryoprecipitate instead of Factor VHI concentrate. This argument was not available in relation to the 1982 treatments, because cryoprecipitate was used on that occasion in addition to concentrate and it was uncertain, if the infection did in fact result from the 1982 treatments, whether it resulted from the concentrate or the cryoprecipitate.

31. There was no sufficient reason, at any time up to and including September 1983, for the hospital's doctors to have taken the view that cryoprecipitate afforded a significantly less risk of transmission of the AIDS virus than Factor VIII concentrate, and they were not negligent in prescribing concentrate in that period. It could not have been said that, had the plaintiff been treated in September 1983 with cryoprecipitate instead of concentrate, the risk of transmitting HIV to him would probably have been avoided.

From a clinical point of view the concentrate was superior to the cryoprecipitate because a smaller amount of the product needed to be infused, because the Factor VIII content was known before use, and because it did not contain the impurities present in cryoprecipitate. There was also a risk that cryoprecipitate would generate antibodies to Factor VIII. In Australia the blood supply was known to be generally safer than in the United States. In Amercia, a great deal of source plasma was provided by paid donors, and the incidence of hepatitis B had been shown to be higher among paid donors than among volunteers. Paid donors were likely to include poor people, who were undernourished and more susceptible to infection, and drug abusers wanting money to buy drugs. A person doing it for the money was less likely to answer questions honestly.

Hospital's failure to warn plaintiff or parents

32. On the balance of probabilities, the plaintiff's parents were informed generally by his treating doctors in 1980, when he was diagnosed as a haemophiliac, that the use of blood products in AHF treatment carried with it a minimal risk of

infection, which risk was far outweighed by the importance of having the treatment in the event of a bleed. They did not specifically mention the risk of hepatitis and no subsequent warning of risk was given.

33. This warning was completely adequate to discharge the hospital's duty of care in relation to the treatments administered in March 1982 and there was no breach. It was not reasonably necessary to warn of the remote risk of infection by an unknown virus.

The risk of hepatitis was very small, the risk of other known infections was virtually non-existent because of the serological testing, and the risk of unidentified infections was theoretical only and completely unquantifiable. The risk that existed in 1980 had not altered or increased prior to March 1982, and no additional circumstances were or ought to have been known which would have made it necessary to repeat, revise or affirm the warning earlier given.

34. It remained the obligation of the doctors to consider, on every occasion when AHF treatment was contemplated, whether the indications for such treatment were such as to outweigh the known risk, including the theoretical risk of unknown and unidentified infections.

35. In relation to the treatment of September 1983, the hospital was in breach of its duty of care in the failure of the plaintiff's doctors to warn his parents of the risk of AIDS, but the plaintiff failed on the issue of causation. The risk of AIDS had by this time arisen in relation to the use of AHF and the plaintiff and his parents had not previously been warned of it.

Although the risk was small and unquantifiable it was clearly recognisable by physicians who had kept in touch with developments, and the consequences, if the risk eventuated, were devastating. Reasonable care required that the patient, or in this case his parents, were warned in appropriate terms of this risk, and no such warning was given. The hospital was in breach of its duty, even though the experts who gave evidence considered that such a warning was not appropriate in September 1983, inter alia because it would have been unthinkable that the parents would have refused the treatment.

36. The risk was material because the potential consequence was so serious that a reasonable person in the patient's position would have been likely to attach significance to it. This did not mean that a risk was material only where a reasonable person in the patient's position, having been made aware of the operation, would decline treatment. The question was whether it was a matter which a reasonable patient would wish to take into account in deciding whether or not tor accept the treatment.

37. An appropriate warning would have canvassed: the likely consequences of failure to treat a joint bleed; the previous successful treatments of the plaintiff and his brother despite the ever-present risk of hepatitis; the small number of cases in America where recipients of AHF treatment had developed AIDS; that as at May 1983 the number of such cases was only 12 out of 15–20,000 haemophiliacs; that the suspected transmissibility of the AIDS virus by blood products had not been proved; that the U.S. National Hemophilia Association was continuing to urge haemophiliacs to accept AHF treatment; that the Australian blood supply had always been regarded as much safer than that in America because of the exclusive use of voluntary donations; that at that time there had not been in Australia a single case of AIDS related to blood products; and the strong expression of the doctor's own opinion that the risk was negligible and should not influence the refusal of AHF treatment.

38. The plaintiff failed to establish a causal link between the doctors' breach of duty in relation to the September 1983 treatments and the plaintiff's infection, if in fact those treatments were the source of his infection.

39. The test of causation was a subjective test. Evidence by the patient (or, in the case of a child, the patient's parents) as to what his or her response would have been was relevant and admissible, and may have been of considerable weight, but nevertheless it was to be viewed with caution because it was clearly likely to have been much influenced by hindsight. The court was not bound to accept the patient's assertion that, fully informed and warned, he or she would have refused treatment. The evidence of the patient was to be evaluated in the light of all the circumstances, including the terms of an appropriate warning.

40. The plaintiff failed to establish that, if an appropriate warning had been given in September 1983, his parents would have refused the AHF treatments and those treatments would not have been administered. Relevant circumstances were, among others, the confidence which quite clearly both parents had in the doctors' expertise and concern for their sons and the previous AHF treatments administered to the plaintiff and his brother with anticipated good results and the apparent absence of any harmful side-effects.

Causation in fact

41. Although not necessary to decide, the plaintiff was infected by the treatments administered in March 1982, and it was the administration of the cryoprecipitate, rather than the concentrate, which infected him.

This conclusion was based on the evidence as to the likely incubation period of the disease coupled with the evidence that the plaintiff in April 1982 exhibited symptoms consistent with sero-converting illness. Apart from statistical evidence, which was useless, there was a total absence of any evidence to suggest that the infection was acquired in September 1983.

42. Within a few weeks of infection, about 24% of AIDS sufferers developed a "sero-conversion" illness (marking the stage when the virus was translated into actual infection) with fever and vomiting. The plaintiff presented to a general practitioner with similar symptoms 15–17 days after the treatment administered in March 1982. Although the notes made

by this practitioner were by no means diagnostic, the onset of an illness with the symptoms noted at such a date after treatment with AHF was consistent with that illness being a sero-converting illness.

43. It was almost unheard of for anybody to develop symptoms of AIDS within two years of infection. The plaintiff's symptoms became apparent in about January 1985 making it highly unlikely that the plaintiff was infected in September 1983.

44. Evidence that there were more people infected with HIV in September 1983 than in March 1982, while establishing that, of a number of HIV infected persons who had received blood products, both in March 1982 and in September 1983, a larger proportion of them were probably infected in September 1983, it was not possible to draw this inference, even on a balance of probabilities, in relation to any particular person.

45. The evidence of D-20 and those he had infected enabled it to be safely assumed that as at 16 June 1982, D-20 was HIV positive, but it by no means followed that he was infected three months earlier. Again, evidence as to the statistical probability that he was infected by March 1982 established no more than that of all people infected as at June 1982, the probability was that a larger proportion of them had been infected by March 1982, but afforded no basis for saying anything as to when a particular person became infected.

46. Although not necessary to decide, of the several products administered to the plaintiff in March 1982, the cryoprecipitate was on the balance of probabilities the source of his infection.

47. The method of manufacture of Factor VHI concentrate would have tended to produce an even distribution of any virus present, and one would therefore anticipate that if the virus was present in one bottle of concentrate in sufficient quantity to be infective, it would be present in all bottles from the same batch, and that if it was diluted, any person who received an equal or larger dosage than the plaintiff would be likely to be infected. The same batch of concentrate as that used on the plaintiff was used on others without ill effect.

Remoteness of harm

48. Had negligence been established against the defendants in relation to the treatments administered in March 1982, the plaintiff would not have been denied damages on the ground of remoteness. The plaintiff's injury was of the same kind (infection by a blood borne virus) as that foreseeable by the defendants in March 1982 as a consequence of any negligence on their part. It was not to the point that the plaintiff received a retro-virus as distinct from a virus otherwise defined (such as hepatitis B), or that the AIDS virus behaved differently in blood from other viruses.

[Headnote by the CCH COMMON LAW EDITORS]

Appearances: Mr Gracie (instructed by J.M. Carvana Kay & Barry) for the plaintiff; Mr Catania, Mr Plibersek and Mr Rainbow (instructed by the Australian Government Solicitor) for the first, second and third defendants respectively.

Before: Badgery-Parker J.

Badgery-Parker J.: This is an action for damages for negligence, brought by the plaintiff, H, against three defendants: The Royal Alexandra Hospital for Children (referred to hereafter as "the hospital" or "the first defendant"); The Commonwealth Serum Laboratories Commission (referred to hereafter as "CSL" or "the second defendant"); and The Australian Red Cross (New South Wales Division) (referred to hereafter as the "blood bank" or "the third defendant").

H became infected with the human immuno-deficiency virus (hereinafter referred to as "HIV") from one or more blood products administered to him by the hospital, and thereby contracted the disease, Acquired Immune Deficiency Syndrome (referred to hereafter as "AIDS") from which he will surely and soon die.

Of the blood products administered to the plaintiff, some were manufactured by CSL, some were manufactured by the blood bank and all were supplied to the hospital by the blood bank. When and from which blood product the plaintiff became infected were matters very much in issue.

All of the defendants deny negligence.

1. The plaintiff

The plaintiff was born on 14 May 1973, the second of his parents' two children. He grew normally and was apparently healthy but for an undescended testicle. It was decided that surgery should be performed to correct this condition. In mid-1980, the plaintiff's elder brother AH was taken to the hospital for treatment to a cut on his lip which would not stop bleeding, and he was diagnosed as suffering "mild haemophilia A". That diagnosis having been made in respect of AH, it was desirable to test the blood of the plaintiff before proceeding with the proposed operation. Tests were carried out in July/August 1980 and it was established that he too had "mild haemophilia A".

2. Haemophilia A

Haemophilia A is a genetic condition of the blood, namely a deficiency of one of the coagulant factors, that which is known as Factor VIII. The Factor VIII level of the blood of one who is in this respect normal with respect to clotting activity is described as 100% and most people have a Factor VIII level of or close to that. Persons with a Factor VIII deficiency are classified as severe, moderate or mild haemophiliacs according to their individual Factor VIII level: below 5%, severe; 5%–10%, moderate; above 10% (but well below 50%), mild.

The expression “mild haemophilia” was criticised by several witnesses, all of them specialist haematologists, as dangerously misleading. It does have meaning as distinguishing one group of haemophiliacs from others, in respect of lifestyle: a mild haemophiliac can live an almost normal life, subject to very few restrictions. He must avoid heavy body contact sports such as rugby football (though he could play soccer), and he should live and work within easy reach of a major hospital where Factor VIII replacement therapy and skilled advice is available in the event that he should start to bleed. He must pay attention to any visible bleeding that occurs, and especially to any signs or symptoms that might indicate internal bleeding, especially bleeding into any joint, and seek medical advice promptly should any such occur. Swelling of a joint may have, a number of causes, but is often, at least in the case of a haemophiliac, a sign of bleeding into a joint, a condition which, if allowed to continue, may cause great pain and, later in life, degenerative arthritis of the joint with associated pain and disability. In a haemophiliac, therefore, the occurrence of joint swelling is an indication to seek urgent medical advice, for diagnosis and if appropriate, for the administration of Factor VIII replacement therapy. Should he at any time undergo elective surgery, protection against bleeding would be necessary, by the administration of such therapy. So too, in the event of other bleeding, such therapy might be called for. Factor VIII replacement therapy involves the administration of anti-haemophilia factor (AHF), a substance rich in Factor VIII, isolated or extracted from human plasma.

The problem which the haematologists have with the use of the term “mild haemophiliac” was expressed by Dr Rickard in these terms:

> “I do not use the word ‘mild’ haemophilia. I think it is dangerous to use that connotation … I think it conjures up in people’s minds the concept that they are not liable to have bleeding problems, and very often the situation is that in my experience if people who are labelled mild haemophiliacs, people do things to them thinking that they are only so-called mild haemophiliacs and they run into trouble.”

Dr Kendall seemed to have been deceived by this fallacy. He expressed the view that the fact that the plaintiff was a “mild” haemophiliac who needed only two doses of Factor VIII to stop haematuria was a predictor of the plaintiff’s likely treatment needs in the future. I prefer Dr Rickard’s evidence. I find it not possible to place any reliance on Dr Kendall’s opinion as to the appropriate treatment of haemophiliacs (a field in which he admitted very little experience).

3. Anti-haemophilic factor (AHF)

Those witnesses who were involved in the treatment of haemophiliacs were unanimous in emphasising the need for swift and adequate treatment of, in particular, joint bleeds because of the disastrous consequences which might ensue from the withholding of treatment. Not only does the continuing bleed produce pain and disability at the time, but it is likely to produce painful degenerative arthritis of the joint in later life. Many haemophiliacs in later life require surgery to relieve pain in the joints. Surgery itself for a haemophiliac involves the obvious risk of massive bleeding and can only be safely carried out with adequate Factor VIII cover. I asked Dr Dietrich:

> “Q. How did you treat the haemophiliac before the development of Factor VIII? A. Poorly.
>
> Q. What techniques did you use? A. When I first began we used freezer dried whole plasma. The volume of the bottles was a pint, 250 ml. That raised the Factor VIII level 1 per cent or 2 per cent. Virtually bleeding was almost uncontrolled. We used regular methods, we used a great deal of creative medicine, and we had a high death rate from uncontrolled bleeding.”

Clearly then the development of AHF was a major advance in the treatment of such patients and offered them relief of symptoms, protection from disability, and security which they had never enjoyed before.

Quite clearly also, physicians having been equipped with this marvellous new weapon might be expected to be very reluctant to revert to earlier methods of treatment with all their experience of known risks and disadvantages. Professor Penington summed up the importance of the development of AHF in the following passage in Exhibit 308:

> "Patients with severe haemophilia commonly died in earlier years from haemorrhage and its complications. Until the development of methods of concentrating the missing clotting factor from blood were developed in the 1950s and 1960s, major surgery could not be undertaken on such people without very great risk. Blood transfusion was required even for relatively minor surgery in patients with mild haemophilia; many patients, even with mild haemophilia suffered progressive disability due to the occurrence of bleeding in joints from time to time even where they did not fall victim to major trauma or have to be submitted to major surgery. The development of methods to concentrate the missing coagulation factor (Factor VIII) from blood plasma offered new hope to haemophiliacs. There was the expectation of being able to lead a normal life, with the exception of treatment at the first sign of any haemorrhage. The development of cryoprecipitate from individual donations of blood or Factor VIII concentrate greatly improved the quality of life of such patients …"

At all relevant times, AHF was produced in two forms. Both were manufactured from plasma extracted from human blood. One was a powdered concentrate, properly called Factor VIII concentrate but often (and sometimes confusingly) referred to simply as Factor VIII. Both the second and third defendants produced a Factor VIII concentrate but, at relevant times, their methods of manufacture were different. The other form of AHF was a cryoprecipitate of Factor VIII, usually referred to simply as cryoprecipitate or cryo without any reference to Factor VIII. Many years ago the second defendant was a manufacturer of cryoprecipitate but had ceased to be such before any time relevant to this action. The third defendant was, at relevant times, a manufacturer of cryoprecipitate and a supplier of it to the hospital.

AHF as cryoprecipitate is supplied in plastic bags, each containing the product of the plasma extracted from a single donor. A quantity of whole blood is removed from the donor into a closed system wherein the plasma or liquid portion is separated from the formed elements of the blood, the latter being returned to the donor's body. The fresh plasma is frozen rapidly and then allowed to thaw and in so doing forms a precipitate which contains most of the coagulation Factor VIII present in the starting plasma. As will be seen later, it also contains a considerable quantity of a substance called fibrinogen which, in the opinion of some experts, produces some of the disadvantages said to be associated with its use. The end product is and must be stored in a frozen state. It must be thawed before use, and having been, by thawing, converted to a liquid state, it is instilled directly into a vein through a canula.

AHF as concentrate is supplied as a freeze-dried ("lyophilized") powder which can be safely stored under ordinary refrigeration. It is dissolved in water and then administered by means of injection or intravenously in similar fashion to cryoprecipitate. It is produced from the plasma of many hundreds, even thousands, of donors. In the process used by the third defendant at all relevant times, plasma from individual donors was first made into cryoprecipitate, and a large number of those cryoprecipitates were then pooled before the Factor VIII concentrate was extracted from them. In the process used by the second defendant, the plasma of a large group of donors was pooled, and the concentrate of Factor VIII extracted directly from the plasma. The amount of Factor VIII activity present in one millilitre of normal plasma is described as one unit of Factor VIII. The amount of Factor VIII activity contained in a single bag of cryoprecipitate cannot be determined, because any attempt to assay it would destroy the sterility of the bag, and it may vary according to the Factor VIII level in the blood of the donor and the amount lost in the extraction process. A single bag may contain as little as 60 or as many as 135 units. For clinical purposes, a Factor VIII level of 90 to 100 units is assumed, so that a patient needing 1,000 units would need to be given 10 or 11 bags.

Factor VIII concentrate produced by the second defendant was made from fresh frozen plasma supplied to it by the Red Cross Blood Transfusion Services of the several States and of New Zealand. On occasions, a quantity of plasma supplied by the third defendant (which, it should be noted, is the New South Wales division of the Australian Red Cross, a legal entity separate from and not relevantly related to other State divisions and their blood transfusion services) would be manufactured by the second defendant into concentrate, the whole of which was then returned by the second defendant to the third. More often, a batch of concentrate was manufactured from fresh frozen plasma from a number of different State divisions of

the Red Cross, and sometimes from New Zealand, and in that event each plasma supplier received back from the second defendant a quantity of concentrate proportionate to the quantity of plasma which it had contributed. The processing of Factor VIII concentrate involves the production at the same time of hundreds or thousands of bottles, each containing the same proportion of Factor VIII, and accordingly sample bottles can be withdrawn and assayed and each bottle distributed can be labelled with the precise amount of Factor VIII activity present therein.

4. The plaintiff (2)

Once the diagnosis of haemophilia was made, it was decided that the testicle operation could still proceed but that it should be done at the hospital under the supervision of the skilled paediatric haematologists on the staff. The plaintiff was admitted to the hospital on 21 April 1981, for surgery the same day; and before, during and after the operation, Factor VIII replacement therapy was administered to him, namely, 8,000 units, almost certainly in the form of concentrate, not cryoprecipitate. The hospital records do not so show, but it is generally accepted that the latter substance is unsuitable when a large amount of AHF needs to be given to a person of small body weight, especially a child. Dr Kendall appeared originally to express a different view in his report, Exhibit ZZ, but in the course of his oral evidence corrected what he had written. This therapy was evidently successful, as no excessive bleeding occurred, and he was discharged home on 29 April 1981.

On 13 June 1981 in the evening, after a game of soccer earlier that day in which, so far as the plaintiff was aware, he had not suffered any particular trauma, he became aware of blood staining of his urine. On 14 June 1981, his parents took him to the hospital where he was seen by a doctor. Again, Factor VIII replacement therapy was ordered and given. The records show that two doses of Factor VIII were given, about 12 hours apart as is usual. One dose was of 450 units, the second of 550 units. Again the hospital records contain no precise statement that concentrate was used rather than cryoprecipitate, but the probability is that concentrate was used. Tie practice was widespread among the medical witnesses, of using the term Factor VIII to refer to concentrate as distinct from cryoprecipitate notwithstanding that the latter also is a preparation of Factor VIII; and the general use of the two terms in the hospital records is consistent with that usage.

On 19 March 1982 the plaintiff's right elbow became swollen. On the evening of 20 March 1982, his parents took the plaintiff to the hospital. Factor VIII replacement therapy was given that evening; and on each of the two following days.

On 25 September 1983 at 1.50 a.m., the plaintiff's parents again took him to the hospital with a swelling of his right knee. Factor VIII replacement therapy was ordered and given on that and the following day.

I shall return later to a closer examination of the evidence of the plaintiff's treatment on these two occasions.

During the years when those treatments were given, the plaintiff was living with his parents and his elder brother in a suburb of Sydney. He attended the local public school, where he made good progress in class and in sport. He was gregarious, outgoing, and had many friends. But for the minor restriction of his sporting activities and the intermittent inconvenience of trips to the hospital for AHF treatment, his genetic disorder made very little difference to his lifestyle and his enjoyment of life; nor was there any reason to anticipate that it would ever do so.

The plaintiff spent his sixth year of primary schooling at a private school in Sydney. During that year (1984) his parents opened a business in a rural city and his family moved to that city in time for the boys to start school there at the beginning of 1985.

Late in 1984, the plaintiff's mother observed that his mood was slightly down, but she attributed this to the separation from his father who had preceded the family to the country. In early 1985, the plaintiff began to appear unwell, and towards the end of March his parents took him to a local doctor for treatment of what appeared to be an upper respiratory tract infection. In April and May, it became apparent that he was losing a lot of weight; he was not eating well; he was lethargic. Investigations were carried out at the hospital during June 1985, and the diagnosis was made that he was infected with the HIV virus, that which causes the disease AIDS.

The plaintiff was not at that stage told of the diagnosis but was allowed to believe that he had a form of hepatitis which would eventually respond to treatment. Whether or not at that stage he in fact believed that, it is, I think, reasonably clear on the evidence that by September 1986, when he was told the true nature of his illness, he already had a pretty good idea what it was. He missed a lot of school in the second half of 1985 and through 1986, with the need to attend frequently for medical tests and treatment, as well as because of his generally poor state of health. His care was transferred from the hospital to Prince of Wales Children's Hospital where he came under the care of Dr Ziegler, and it was by that physician that he was in due course acquainted with the true nature of his disease.

In the first half of 1987, the plaintiff again was unable to attend school, but in the middle of that year the drug AZT became available for him, and despite initial unpleasant side effects, he had a good response to it. He began to regain some of his lost weight and energy and returned to school from about the middle of 1987, until about the middle of 1988. However, he had no spare energy for sport or homework and indeed was often unable to attend school the full five days.

In mid-1988, the family moved back to Sydney, in order to facilitate the plaintiff's medical care. They returned to the same suburb and he was enrolled at the local high school where he was able to renew old friendships from primary school days. This provided some improvement in his social life, but he remained unwell, and unable to cope with a full school program.

By Christmas 1988 he was very sick and since then he has been repeatedly in hospital. He cannot eat normally because of severe thrush infection of his mouth and gullet, and even if he were able to swallow, he has been unable to keep food down. His fear of vomiting in public has caused him to avoid going out on those few occasions when he has felt well enough to do so. Since June 1989 he has been fed through a tube which pierces the wall of his stomach, and that feeding device, mounted on a tall metal stand, must accompany him wherever he goes. Life for him now is unpleasant and boring, and not surprisingly he is often depressed and irritable. He is often in severe pain and his mood from time to time reflects the degree of that pain.

The prognosis expressed by Dr Ziegler on 25 October 1989 is that he is highly unlikely to survive for two more years and unlikely to live another 12 months.

5. Diagnosis and treatment of the plaintiff at the hospital — March 1982 and September 1983

The plaintiff's case against the hospital is primarily that, at times when it knew or should have known of the risk of HIV infection from blood products, it failed to take adequate precautions to guard the plaintiff against such risks, including the adoption of alternative methods of treatment and the furnishing of appropriate information and warnings which, if given, would, it is said, have led to the rejection by the plaintiff's parents of the proffered treatment with AHF. Those are matters appropriate for consideration at a later stage. However, it appeared as the matter proceeded that the plaintiff sought also to rely on an allegation that the hospital was negligent in another respect, namely, that on the occasion of his attendances in March 1982 and September 1983 it wrongly and negligently diagnosed his condition as haemarthrosis (bleeding into a joint) a condition ordinarily requiring treatment with blood products; and that it was therefore negligent to administer AHF. It is convenient to deal with this allegation separately and immediately.

The hospital's clinical notes relating to the plaintiff were admitted into evidence and are Exhibit OO. Also in evidence is a series of exercise books in which the hospital's blood bank maintained a record of blood and blood products issued for treatment of patients in the hospital (Exhibit PP and Exhibit 322). No other relevant records have been produced of the administration of blood products to the plaintiff in the hospital.

On the evening of 20 March 1982 at 9.25 p.m. the plaintiff's parents took him to the hospital because, on the previous day, his right elbow had become swollen. There was no history of trauma. He was seen by a medical officer whose name has not been disclosed, who was not called to give evidence, and of whose absence no explanation was offered. That person discussed the case by telephone with Dr Lammi, the senior haematologist on the hospital staff, and treatment with AHF was prescribed.

The hospital notes, Exhibit OO, are very sketchy. They contain little in the way of history, and no findings on examination are recorded other than "R elbow 21 cms; L elbow 19 cms" and "weight 27 kg". It can readily be inferred that the elbow was in fact swollen. The prescription of later doses to be given "if still

restricted movement" would support the inference that movement of the elbow was restricted at the time when the plaintiff was seen. Further, the fact that AHF was prescribed at all would support an inference that a diagnosis of a joint bleed was in fact made. Mr Murray argued that no such inferences should be drawn in view of the unexplained absence of the relevant resident medical officer from the witness box; and that on the basis of swelling alone, a diagnosis of joint bleed should not have been made. It would have been appropriate, he argued, to wait and see — to treat the elbow with ice packs which would reduce the swelling, if it were indeed not due to a bleed, and may stop the bleeding if such was the cause of the swelling.

When the plaintiff was first seen, 900 units of Factor VIII was ordered for immediate administration and that was given. At 10.15 a.m. on 21 March 1982, a further 600 units of AHF were administered to the plaintiff. Later on 21 March, further AHF was administered — 255 units of concentrate and three bags of cryoprecipitate. At 9.15 a.m. on 22 March 1982, 580 units of Factor VIII were administered. The hospital notes relating to the administration of the last dose record "rang Dr Bryant" (a reference to a haematologist junior to Dr Lammi) and it is a reasonable inference that he was consulted before that dose was prescribed.

I accept that swelling and/or restriction of movement continued throughout 21 March and that the symptoms had not totally resolved next morning: that is, that the condition which caused the elbow to swell was somewhat persistent, and responded but slowly (if at all) to treatment with AHF. Competing inferences are perhaps available: that it was not a joint bleed at all, for which reason the successive administrations of AHF had no beneficial effect upon it; or that it was a bleed of some severity, necessitating repeat treatments.

Professor Dwyer was of opinion that the material disclosed in the notes was insufficient to make a diagnosis of joint bleeding and I accept that view. It cannot, however, in my view be presumed that nothing was known to the resident medical officer and to Dr Lammi other than what is recorded in the notes.

Dr Lammi gave the following evidence which I accept:

> "Q. Dr Lammi, how do you make a diagnosis of bleeding into the joint? A. It's based on—
>
> Q. Based on swelling or are there other factors? A. It's based on the history and then examination and — several things. It — the main thing is swelling.
>
> Q. Does swelling of the joint necessarily indicate bleeding? A. In a haemophiliac, if you have swelling of a joint you would be very — one should make a diagnosis of bleeding until proved otherwise.
>
> Q. Because of the risk that it may be bleeding? A. It virtually always is."

It appears to me that Dr Lammi is a very careful and competent medical practitioner; and it is clear that he was consulted by the resident medical officer on duty before the decision was made to order AHF. I accept that he would not have ordered AHF unless satisfied that the diagnosis was correct, and that he would have elicited from the resident medical officer such information as was necessary in his view to confirm the diagnosis.

Mr Murray criticised the first defendant's failure to call the treating doctors, and submitted that in the absence of that evidence, I should not infer that the diagnosis of joint bleed was correct. That, however, would not take the plaintiff far enough. The onus is on him to show that the diagnosis was wrong; and although, in the absence of more satisfactory evidence from the first defendant who is in the best position to establish the facts, the plaintiff might discharge that onus with but a scintilla of evidence, it seems to me that there is not even a scintilla of evidence of any other diagnosis that ought to have been preferred. It does not appear to me that the following evidence of Professor Dwyer goes far enough. He said:

> "Q. What other than a joint bleed could it have been to produce those findings if not a joint bleed? A. Certainly the top diagnosis in this child obviously would be it was bleeding but there could be other causes. It says in the notes there is no known injury here but people can have swellings associated with joints where they don't have known injuries if they are mild enough at the time. There could have been a bleed not into the joint but into the tissue* around the joint. There could have been fluid escape from blood vessels which is simply because of shock to those blood vessels causing the sort of inflammation of swelling that any of us can get with trauma. Infection is a possibility but unlikely. It is unlikely that some other signs of heat, etc., would not have been mentioned in the notes. I would like to make it clear I am not suggesting that any diagnosis other than bleeding would be high on my differential diagnosis list."

(*The uncorrected transcript says “section” but my own note says “tissue”, and that I believe to be correct.)

I am satisfied that the diagnosis has not been shown to be incorrect; indeed, were it necessary for me so to find, I would have no difficulty in concluding on a substantial balance of probability that the diagnosis of a bleed into the joint was the correct one.

The plaintiff’s next visit to the hospital, at 1.50 a.m. on 25 September 1983 is more fully recorded in Exhibit OO. The problem which led to his attendance was “sore R knee”. The history given by the plaintiff or his parents was “knocked knee one week ago. No symptoms until tonight. Complaining of sore right knee. Pain has slightly increased. No swelling. Well otherwise”. The resident medical officer on duty examined the knee and notwithstanding the absence of any complaint of swelling by the plaintiff or his parents, she recorded “R knee — obviously more swollen compare L. Warmer compare L. Full range movement — no obvious bony-cartilage-ligamentous damage”. She also recorded — “R knee circum. 32 cms compare 29.5 L”. She recorded a provisional diagnosis “haemarthrosis in patient with mild haemophilia — for Factor VIII”. It is then recorded that she discussed the patient with Dr Moore, a registrar, and with Dr Lammi. AHF was prescribed for immediate administration and at 3.00 a.m. he was given 1,080 units of Factor VIII concentrate. He was allowed home but returned for a further administration of AHF at 5.00 p.m., 675 units. On 26 September 1983 the plaintiff returned to the outpatient department where he was seen by Dr Lammi and a resident medical officer, Dr Kakakics. He was prescribed further AHF therapy, 675 units of concentrate which was given. It was contemplated that he should return for a later dose at 7.00 p.m. the same day — “repeat (and probably last) dose Factor VIII tonight 7 p.m. * could Factor VIII please be obtained from haematology and kept in normal fridge this afternoon in readiness”. There is no sufficient evidence that any later treatment was in fact given; I am not satisfied that it was; Exhibit OO makes no further reference to it, and the record in Exhibit PP is incomplete.

Dr Dwyer accepted and counsel for the plaintiff did not seek to challenge the accuracy of the diagnosis of haemarthrosis in September 1983. I am satisfied that the diagnosis of haemarthrosis on that occasion also was correct.

It follows from the findings that I have made that there is no basis on which the first defendant could be held to have been negligent in diagnosis on either of the relevant occasions. In those circumstances, the administration of AHF was clearly the appropriate treatment unless the view is open that, notwithstanding that it was prima facie the appropriate treatment, treatment of that kind should have been withheld because of the risk of transmission of infection. To that aspect I will return at the appropriate time.

6. Acquired immune deficiency syndrome

The disease known as AIDS attacks the immune system of the body, thus destroying or severely reducing the body’s ability to defend itself against a wide range of infections and other illnesses. Deficiencies of the immune system may be congenital; they may have other external causes, including the side-effects of chemotherapy for cancer. Two particular diseases, Kaposi’s sarcoma and Pneumocystis carinii pneumonia were known to be complications of profound disturbances of the immune system. They were not, before 1981, known as diseases associated with homosexuality.

Professor Paul Vincent Holland, now of Sacramento, California where he is the medical director of the Sacramento Medical Foundation Blood Centre and professor of haematology and oncology at the University of California, was in 1980 located in Washington at the National Institutes of Health, Bethesda. In that year at that institution he said he began to see patients, young men, as it turned out homosexuals, with “rather peculiar multiple infections in young men who should not have had these infections”.

In June 1981, in the *Morbidity and Mortality Weekly Report* (*MMWR*) (No. 30, p. 250), the Centres for Disease Control at Atlanta, Georgia, reported five cases of Pneumocystis pneumonia in homosexual men from Los Angeles. On 3 July 1981, *MMWR* carried a further report (Exhibit EEE) that in the past 30 months (which I take to mean from about January 1979) Kaposi’s sarcoma had been diagnosed in 26 homosexual men, 20 in New York City and six in California. The same article stated that since the earlier report, 10 additional cases of Pneumocystis pneumonia had been identified in homosexual men in California.

In retrospect it appears that the cases seen by Professor Holland and the cases reported in *MMWR* were the first AIDS sufferers in the United States of America.

The *New England Journal of Medicine* of 10 December 1981 published a paper entitled "Pneumocystis Carinii Pneumonia and Mucosal Candidiasis in Previously Healthy Homosexual Men" (Gottlieb et al. — Exhibit CCC). The report dealt with four cases, in all of which investigations revealed the presence of an acquired cellular immune deficiency. The illness was thought to be possibly associated with cytomegalovirus (CMV), a sexually transmitted infection of which all four patients had serologic evidence and which was known to be highly prevalent in the male homosexual community. In the view of the authors, the "distinct and unusual clinical syndrome" presented in these four patients represented "a potential transmissible immune deficiency".

A second paper in the same issue (also part of Exhibit CCC) by Masur et al. reported 11 cases of Pneumocystis carinii pneumonia in young New York men. Seven were drug abusers, six were homosexuals, two were both. The disease appeared to be the consequence of immune suppression. The authors were not aware of any previous data suggesting that immunosuppression had been frequent among homosexuals. They suggested that since now homosexuals were suddenly contracting a variety of opportunistic infections, it seemed unlikely that the outbreak of Pneumocystis carinii pneumonia was due exclusively to a new virulent or resistant strain of Pneumocystis. In their opinion, the outbreak was "more probably related to the immunologic consequence of some unknown process".

A third paper in the same issue reported other evidence of severe acquired immuno-deficiency in male homosexuals.

The growing evidence of opportunistic infection and Kaposi's sarcoma in homosexual men attracted editorial comment in the same journal (Exhibit DDD):

> "In the summer of 1981, the Centres for Disease Control (CDC) alerted the medical world to an unexpected outbreak of Pneumocystis carinii pneumonia and Kaposi's sarcoma in young homosexual men who had no known reason to contract these uncommon diseases. New and surprising though this association may be, enough cases have already been collected to establish its reality. More than 160 examples are now being analysed at the CDC, and five or six new cases are being reported each week. Recently, eight cases of Kaposi's sarcoma in young homosexual men in New York City were reported in *Lancet*. In this issue the journal presents for our evaluation further detailed reports of the syndrome of opportunistic infections and Kaposi's sarcoma homosexual men (three papers, 19 patients, and 31 authors).
>
> These salient features have emerged: The patients are typically young homosexual men, most of whom live in large cities and many of whom use drugs; the infectious agents are low-grade pathogens that often cause opportunistic infections in compromised hosts; and the death rate is fearfully high. Two thirds of the patients described in this issue of the Journal have died, as have more than one third of the patients on record at the CDC."

The writer speculated as to the significance of the link with homosexuality; the possibility that the susceptibility of patients to a wide range of opportunistic infections was a manifestation of a primary immunosuppressive viral disease; the evidence of previous or current CMV infection as a common thread and a suspect cause of the syndrome; and the possibility of a link to the use of recreational drugs.

Thus it can fairly be said that by December 1981, the syndrome now known as AIDS was recognised as a new medical disorder, fast growing, but apparently to be found almost exclusively among homosexual men: according to the writer of that editorial (Exhibit DDD p. 1466), "At present, only one woman is included among the patients known to the CDC".

On 16 July 1982 came the first report of a similar syndrome not among homosexuals but among persons with haemophilia. The vehicle was again the *MMWR* (Vol. 31 No. 27, p. 365 — Exhibit L):

> "CDC recently received reports of three cases of Pneumocystis carinii pneumonia among patients with haemophilia A and without other underlying disease. Two have died; one remains critically ill. All three were heterosexual males; none had a history of intravenous drug abuse. All had lymphopenia, and

> the two patients who were specifically tested had had in vitro laboratory evidence of cellular immune deficiency."

Each of the three had received frequent injections of Factor VIII concentrate over many years, by reason of severe haemophilia.

An editorial note was appended to that report:

> "Pneumocystis carinii pneumonia has not been previously reported among haemophilia patients who have had no other underlying diseases and have not had therapy associated with immunosuppression
>
> …
>
> The clinical and immunologic features these three patients share are strikingly similar to those recently observed among certain individuals among the following groups: homosexual males, heterosexuals who abuse IV drugs, and Haitians who recently entered the United States. Although the cause of the severe immune dysfunction is unknown, the occurrence among the three haemophiliac cases suggests the possible transmission of an agent through blood products.
>
> CDC has notified directors of haemophilia centres about these cases and, with the National Haemophilia Foundation, has initiated collaborative surveillance. A Public Health Service advisory committee is being formed to consider the implication of these findings. Physicians diagnosing opportunistic infections in haemophilia patients who have not received antecedent immunosuppressive therapy are encouraged to report them to the CDC through local and state health departments."

Notwithstanding the comment in the first paragraph of that note, there was before me unchallenged evidence of Dr Mozen that haemophiliacs are known to suffer some immune dysfunctions as a result of the frequent infusion of AHF in concentrate form.

Following the publication of that paper, the United States Public Health Service convened a meeting in Washington D.C. on 26 July 1982. That meeting was attended by scientists from the National Institute of Health, physicians and scientists from the Centres for Disease Control, scientists and physicians from the Federal Food and Drug Administration, representatives of the blood banking community and plasma manufacturers, prominent haematologists, and members of the haemophilia community. One who was present was Professor Holland. At that meeting the name AIDS was settled upon as the appropriate name for this new disease. It had previously had many different tags, including GRIDS ("gay related immune deficiency syndrome") reflecting the perception that it was peculiarly a disease of homosexuals. The second topic discussed at the meeting, apart from the selection of a name, was whether or not the disease could have been caused in these three individuals by the infusion of blood components or whether, notwithstanding the description of them in the paper, they may have acquired it in some other way. The meeting made no recommendations affecting the treatment of haemophiliacs or the administration or manufacture of blood products because, according to Professor Holland, there was at that stage insufficient information.

The search for additional information continued and the National Haemophilia Foundation, the national organisation of haemophilia groups in the United States, sent a questionnaire to centres where haemophilia was treated, evidently seeking information as to the incidence of AIDS-related symptoms among haemophiliacs.

On 10 December 1982 *MMWR* reported the case of a 20-month-old infant who had developed unexplained cellular immunodeficiency and opportunistic infection following multiple transfusions, including a transfusion of platelets derived from the blood of a male subsequently found to have AIDS. The following editorial note was appended to the report:

> "The etiology of AIDS remains unknown, but its reported occurrence among homosexual men, intravenous drug abusers, and persons with hemophilia A suggests it may be caused by an infectious agent transmitted sexually or through exposure to blood or blood products. If the infant's illness described in this report is AIDS, its occurrence following receipt of blood products from a known AIDS case adds support to the infectious agent hypothesis … None the less since there is no definitive laboratory test for AIDS, any interpretation of this infant's illness must be made with caution … This report and continuing reports of AIDS among persons with hemophilia A raise serious questions about

> the possible transmission of AIDS through blood and blood products. The Assistant Secretary for Health is convening an advisory committee to address these questions."

On 4 January 1983 the Centres for Disease Control convened a meeting at Atlanta at which was discussed the possible relationship between AIDS and the use of AHF in the treatment of haemophiliacs. Dr Dietrich who gave evidence before me was one of those present at the meeting, and a report of the meeting was published in the *Journal of the American Medical Association*, Vol. 249, No. 5 of 4 February 1983 (Exhibit G). There was a diversity of view expressed and no consensus was arrived at. Some of those present were not convinced of the existence of any link between AIDS and the use of blood or blood products although apparently the majority were convinced at least that there was a risk that such an association existed.

Three papers published in the *New England Journal of Medicine* of 13 January 1983 (Exhibits B, C and D) reported further studies which lent support to the proposition that AIDS was caused by a blood borne pathogen and might be transmitted by infusion of AHF. *The Lancet*, 29 January 1983, published a report (Exhibit J) of depressed immune function in two haemophiliacs, and the authors expressed the view that "These findings may represent a prodromal phase … of AIDS. Transmission of an infectious agent in blood products seems likely".

The theory that AIDS was blood borne was but one of a number of theories which were under discussion and investigation. Three at least were reputable theories commanding substantial support from knowledgeable and reputable scientists and medical practitioners. Professor Holland stated those three theories in these terms:

> "One was that it occurred in individuals who were infused one way or another, that is, by their veins or by their rectum or other means, with many different antigenic materials from other humans which may also have contained other materials and their immune system eventually became tired or worn out trying to respond to these insults. The second theory was that individuals with AIDS were assaulted with a variety of infectious agents, infectious viruses which in sum total were overwhelming and wearing out and tiring the immune system. The third theory was that there was a specific agent which could in some way, a new undescribed agent, which could specifically attack the immune system and render it unable to fight off simply ordinary everyday infections."

Dr Mozen, director of Biochemical Research and Development of Cutter laboratories, a major plasma processor, Dr Dietrich, director the World Hemophilia Aids Centre and of the Hemophilia Centre of Huntingdon Hospital at Pasadena, California and Dr Gatenby, a distinguished Australian immunologist endorsed that description of the principal theories current as at January 1983. Not only were these reputable theories, "they were all equally applicable to the different kinds of individuals who were developing AIDS, whether they were very sexually active gay men, or IV drug users or hemophiliacs."

The connection, suspected by many, between AIDS and the use of blood and blood products remained unproven, because of the inability to identify any virus which could be responsible for the disease. Thus on 11 May 1983 the newsletter of the National Hemophilia Foundation (USA) urged haemophilia patients to continue to make use of AHF as prescribed by their doctors notwithstanding recent publicity given to the recall by a commercial manufacturer of a batch of Factor VIII concentrate after it was learnt that one donor whose plasma had been used had later developed AIDS. The newsletter pointed out that in a national haemophilia population of about 20,000, there were only 12 reported cases of AIDS. On 2 July 1983 a group of researchers at Royal Prince Alfred Hospital headed by Dr Kevin Rickard reported in *The Lancet* (Exhibit 101) that they knew of no instances of AIDS in haemophiliacs in Australia although they had detected "T-cell subset changes", an indicator of immune suppression and a common finding in AIDS sufferers. Again on 1 September 1983 the National Hemophilia Foundation Newsletter (Exhibit 205) emphasised that "it has not been scientifically established that AIDS is transmitted through blood products" and recommended that patients continue to accept treatment with AHF.

The literature continued to refer frequently to the possibility of some unrecognised viral agent as the cause of AIDS. In fact, during 1983 Dr Luc Montagnier at the Pasteur Institute in Paris identified a virus which he believed to be the agent responsible for AIDS. It seems that in fact he had done so, but was not able during 1983 to produce sufficient evidence to convince the scientific community that he had done so. In May 1984, Dr Gallo in the United States using a different research technique identified the same virus and

was able to replicate it and to demonstrate that it was indeed the AIDS virus. It was tagged at that time HLTV-III but has since been renamed HIV-I. The virus having been identified, the next development was the devising of means for testing for its presence in blood and such tests were developed by a number of pharmaceutical companies in the latter part of 1984 and were tested by the Centres for Disease Control and also, in Australia, by a team of researchers led by Dr Ian Gust of the Fairfield Infectious Diseases Hospital. The efficacy of two such tests was established and in May 1985 they were licensed by the United States government Food and Drug Administration for general use.

In the meantime, use of the tests in research laboratories had made it possible to trace approximately the course of AIDS in the haemophiliac population in Australia. I have referred already to Dr Rickard's letter to *The Lancet*, published on 2 July 1983. Examination of stored sera from the blood of a large number of Australian haemophiliacs showed that in 1984, 31% had circulating HIV antibodies; in 1983, 11.9%; in 1982, 9.8%; but in 1981, nil. The finding of circulating antibodies in a patient's blood meant that at some stage in his life he had come in contact with the AIDS virus and had developed an immune response to it, and it is a reasonable conclusion that no Australian haemophiliacs and few if any Australian residents were carrying the infection as early as 1980.

In late 1982 and early 1983 the first patients in Australia with clinical manifestations of HIV infection (but not full-blown AIDS) were detected and they were reported, together with the first case of full-blown AIDS in April 1983 (one witness referred to_ the publication as being later, about June 1983; no actual publication was placed in evidence). The first transfusion-transmitted case of AIDS in Australia was identified in Sydney in July 1984.

The developing knowledge of AIDS can then fairly be summarised as follows — mid-1981, recognition of the existence of a new syndrome; mid-1982, the first reports of cases of AIDS among haemophiliacs and the first consideration given to the question whether AIDS might be due to a transmissible blood borne agent; January 1983, the risk of blood borne transmission, though still unproven, thought sufficient to warrant the implementation of precautionary protective measures; May 1984, positive identification of the virus responsible for AIDS, and at last, irrefutable evidence that the disease was transmissible by blood; late 1984, availability of a test to confirm the presence or absence of the virus and enabling retrospective confirmation of its presence in the blood of AHF recipients.

There was some difference of opinion among the expert witnesses as to the significance of the July 1982 report of immune deficiency symptoms in haemophiliacs similar to those reported the previous year in homosexuals: whether it was a report which aroused (or should have aroused) "serious suspicions" (Professor Dwyer) or did no more than raise a possibility requiring observation but no other response (Professor Holland, Dr Dietrich and others); and as to what if any response was called for in changing treatment programs; and as to whether or not the publications in the *New England Journal of Medicine* on 13 January 1983 (Exhibits B, C and D) were as Professor Dwyer maintained "pivotal", "convincing … most people … that it was likely we had a major problem with the transmission of the agent that might cause AIDS among haemophiliacs", or whether, as Professor Holland and other American experts maintained, that was somewhat overstating the significance of that information.

So too there was debate as to whether the publication in *The Lancet* in April 1983 of the case of a child who acquired AIDS apparently after and as a result of a blood transfusion (the same case as had been reported in the United States in *MMWR* in December 1982) had, as Professor Dwyer suggested, put to rest much of the previous doubt as to whether blood products could transmit the infectious agent whatever it was. Other witnesses were not prepared to put the matter as firmly as that.

Such questions are really of academic or semantic interest only and of no practical relevance in this case. The fact is that before July 1982 (and hence as at March 1982, the first date relevant to this case), reasonable physicians and blood scientists had no reason to apprehend that the new disease, later tagged AIDS, was likely to be blood borne and called for any specific response on their part. Further, the fact is that however the risk be quantified in the light of knowledge available in January 1983, and whether they were convinced or not that AIDS was blood borne, physicians treating haemophilia and blood scientists and persons and organisations involved in the collection of plasma and the manufacture of blood products in the United States began to perceive the need to take such precautions as could be devised in case it should turn out that AIDS was a blood borne disease, and within a comparatively short time thereafter, it

was reasonable that similar persons in Australia, and hence the defendants, should have adopted similar attitudes and precautions. Certainly, by September 1983, the second date relevant to this case, persons in the relevant fields ought to have been well aware of the possibility that AIDS was blood borne and giving active consideration to the adoption of appropriate precautions.

7. Alternative formulations of the plaintiff's case

It was common ground that the plaintiff did not become infected by any of the treatments given in 1981. Apart from the issue as to whether a fourth treatment with AHF was given on 26 September 1983 (and I have found that it was not given), it was common ground that the products administered to the plaintiff on the two later occasions comprised:

> *(a) in March 1982*, 6 bottles of concentrate manufactured by the third defendant (batch No. 201); 2 bottles of concentrate manufactured by the second defendant (batch No. 287-1); and 8 bags of cryoprecipitate manufactured by the third defendant;
> *(b) in September 1983*, 18 bottles of concentrate manufactured by the second defendant (batch Nos. 481-1 and 487-1).

All such products, whoever was the manufacturer, were supplied to the hospital by the third defendant.

That being so, the plaintiff's case was put in a number of alternative ways. It was first submitted that the evidence should lead to a finding that the plaintiff became infected in September 1983. On that basis it is asserted that all three defendants are exposed to liability: the hospital, having administered all treatments; CSL as manufacturer of all those products used; and the blood bank as distributor of all. However, liability could not fall on any defendant not involved with every one of the products used in September 1983. As to each defendant which was so involved, it must be shown to have been negligent in respect of each and every product given: but it is clear enough that any of the defendants negligent in respect of one product or treatment in September 1983 was negligent in respect of all.

Alternatively, it was submitted that the evidence shows that the plaintiff was infected with HIV by one or more of the blood treatments administered to him in March 1982 or September 1983, but that the evidence does not enable any finding to be made as to precisely which treatment or treatments conveyed the infection to him. Putting the case that way perhaps avoids some difficulties which might otherwise confront the plaintiff, but it emphasises others. If no single treatment can be identified as more probably than not the source of infection, and all are equally likely to have been the source, the plaintiff cannot recover against any of the defendants which was not associated with every one of the treatments given. The hospital, of course, is still potentially liable, for all of the treatments were administered by it. It is argued on behalf of the plaintiff that the third defendant is on this basis potentially liable as manufacturer of some but as distributor of all of the products used to treat the plaintiff, including those manufactured by the second defendant, whether or not also the plasma used by the second defendant was supplied by the third defendant.

On this basis, however, the plaintiff conceded that the second defendant could not be held liable, because it was in no way connected with certain of the products used to treat the plaintiff in March 1982.

As to the third defendant, on the assumed basis, it could not be held liable unless it is proved to have been negligent in respect of every one of the products administered to the plaintiff. The negligence proved need not be the same in every case: the plaintiff might rely on failure to screen donors or test blood, in respect of such of the products as were manufactured by the third defendant or manufactured by the second defendant from plasma supplied by the third, and on inadequacy of warning in respect of all products involved, but one way or another, some causative negligence must be shown in respect of every product used.

So too, on the assumed basis, the first defendant may only be held liable if proved to have been negligent in respect of each and every treatment administered to the plaintiff in 1982 and 1983.

A third way in which the plaintiff's case was put accepts the contention of the defendants that the evidence compels a finding that the plaintiff became infected in March 1982, either as the result of the administration to him on 20 March of a bag of cryoprecipitate manufactured from the blood of a donor identified as D-20 who later was tested HIV positive and has developed AIDS; or from one (but an unidentified one) of the AHF treatments given at that time. The plaintiff accepts that on this basis, the second defendant cannot be held

liable, because cryoprecipitate used on that occasion was manufactured not by it but by the third defendant, but presents arguments that both the first defendant and the third defendant were negligent.

8. The duty of care

Against all three defendants the plaintiff's case is brought in tort, for negligence. There was no contractual relationship between him and either the second defendant or the third defendant. They are sued as the manufacturers/distributor of products, the use of which by the plaintiff caused him harm. There is no principle of strict liability upon which the plaintiff may rely — in this respect, the law of New South Wales differs significantly from the law of most United States jurisdictions.

No doubt there was a contractual relationship between the plaintiff and the first defendant, whereby the defendant undertook his treatment, but the duty of care thereby imposed upon the defendant is no different from the duty in tort. It is not suggested on behalf of the hospital that the contract contained any provision limiting or excluding its liability. The contract itself is therefore of no relevance.

It was argued by Mr Stitt on behalf of the second defendant that the defendants did not owe the plaintiff a duty of care in respect of HIV or AIDS because at relevant times it was not foreseeable that the plaintiff might suffer damage by being infected with HIV. He argued, indeed, that it was irrelevant to consider at all blood borne viruses or infections at large, since it is known in fact that the plaintiff became infected not with any other virus but with HIV.

No doubt, the submission had its genesis in the words of the Privy Council in *The Wagon Mound (No. 1)* (1961) A.C. 388 at p. 425:

> "It is no doubt proper when considering tortious liability for negligence to analyse its elements and to say that the plaintiff must prove a duty owed to him by the defendant, a breach of that duty by the defendant, and consequent damage. But there can be no liability until the damage has been done. It is not the act but the consequences on which tortious liability is founded. Just as (as it has been said) there is no such thing as negligence in the air, so there is no such thing as liability in the air. Suppose an action brought by A for damage caused by the carelessness (a neutral word) of B, for example, a fire caused by the careless spillage of oil. It may, of course, become relevant to know what duty B owed to A, but the only liability that is in question is the liability for damage by fire. It is vain to isolate the liability from its context and to say that B is or is not liable, and then to ask for what damage he is liable. For his liability is in respect of that damage and no other. If, as admittedly it is, B's liability (culpability) depends on the reasonable foreseeability of the consequent damage, how is that to be determined except by the foreseeability of the damage which in fact happened — the damage in suit? And, if that damage is unforeseeable so as to displace liability at large, how can the liability be restored so as to make compensation payable?"

That passage has been much discussed. It has, as *Windeyer* J. suggested in *Mount Isa Mines Ltd.* v. *Pusey* (1970) 125 C.L.R. 383 at p. 397 given us "the blessed, and sometimes overworked, word 'foreseeability' as a single test for both the existence of liability in negligence and the extent of recoverable damage".

But, although "foreseeability" is the single test, it is a word which does not have only a single significance. As *Glass* J.A. pointed out in *The Minister* v. *San Sebastian Pty. Ltd.* (1983) 2 N.S.W.L.R. 268 (at pp. 295–296):

> "… a recognition has emerged that the foreseeability inquiry at the duty, breach and remoteness stages raises different issues which progressively decline from the general to the particular. The proximity upon which a *Donoghue* type duty rests depends upon proof that the defendant and plaintiff are so placed in relation to each other that it is reasonably foreseeable as a possibility that careless conduct of *any kind* on the part of the former may result in damage *of some kind* to the person or property of the latter: *Chapman* v. *Hearse* (1961) 106 C.L.R. 112, at 120, 121. The breach question requires proof that it was reasonably foreseeable as a possibility that *the kind* of carelessness charged against the defendant might cause damage *of some kind* to the plaintiff's person or property: *Overseas Tankship (UK) Ltd.* v. *Miller Steamship Co. Pty. Ltd.; The Wagon Mound (No. 2)* [1967] A.C. 617, at 642, 643; *Wyong Shire Council* v. *Shirt* (1980) 54 A.L.J.R. 283, at 285, 286; 29 A.L.R. 217, at 219–222. Of course, it must additionally be proved that a means of obviating that possibility was available and would have been adopted by a reasonable defendant, ibid. The remoteness test is only passed

> if the plaintiff proves that *the kind* of damage suffered by him was foreseeable as a possible outcome of *the kind* of carelessness charged against the defendant: *Mount Isa Mines Ltd.* v. *Pusey* (1970) 125 C.L.R. 383, at 390. The many faces of foreseeability is a theme which has been developed with illustrations in an article *Duty to Avoid Economic Loss*, Glass, (1977) 51 A.L.J. 372 at 373, 374."

It seems to me that Mr Stitt's "no duty" submission overlooks the breadth of the foreseeability enquiry at the duty stage. In my opinion, there can be no doubt that each of the defendants owed to the plaintiff a duty of care. The second defendant and the third defendant, as manufacturers and distributors of blood products intended to be infused into the veins of human beings, were under a duty to take reasonable care to make such products safe for their intended use. As has been seen, it was well known long before 1982 that syphilis, malaria and viral hepatitis (at least) were transmissible in blood and blood products and it cannot be doubted that the manufacturers of such products had a duty to take all reasonable and available steps to protect patients from infection from any such known source of infection.

It was also recognised that blood and blood products were capable of transmitting to the recipients *any* blood borne virus and that there was an ever present risk that such could occur with viruses unknown and unidentified. The risk of transmission of such viruses was foreseeable, if not quantifiable, and it follows that the second defendant and third defendant were under a duty to recipients of their blood products to take such reasonable precautions as were available (if any) to protect patients from infections by such means. The issue in this case is not as to the existence of a duty, but as to breach. The plaintiff must prove that there were available to the second and third defendants precautions which if taken, would have obviated or reduced the risk of transmission of HIV and that those defendants' failure to take such precautions was in all the circumstances unreasonable.

Mr Garling, for the first defendant, did not seek to argue against the existence of a duty of care.

The first defendant was under a different duty: the duty to take reasonable care for the plaintiff as its patient, a duty which might be analysed as embracing a duty to use reasonable care in diagnosis, in prescribing treatment for the diagnosed condition, in giving to the patient (or in the case of a child, his parent) appropriate information about and warnings of any risks associated with intended treatment, and in carrying out any treatment (including reasonable care in the selection and administration of treatment modalities, medications, etc.). The duty arises because it is clearly foreseeable that if the hospital fails to use reasonable care in each of those respects, harm may result to its patient. Again, the issue is not duty, but breach.

9. Allegations of negligence

The plaintiff particularised his case against the first defendant in respect of both March 1982 and September 1983 as follows:

> "Having within its means of knowledge the knowledge that the infusion of blood products could result in reception by a patient under its care of a blood borne virus, the first defendant
>
> > (a) failed to warn the plaintiff or his parents as to the risk of infection with a blood borne virus;
> > (b) failed to inform the plaintiff or his parents that alternative treatment, namely, palliative care and/or administration of Factor VIII therapy (i.e. clotting therapy) by infusion of dedicated blood products was available;
> > (c) failed to inform the plaintiff or his parents that alternative treatment, namely, administration of Factor VIII therapy (i.e. clotting therapy) by infusion of cryoprecipitate was available;
> > (d) failed to enter into arrangements or agreements with blood collection agencies which would facilitate or positively implement screening of donors at point of collection."

Notwithstanding that the particulars were expressed in that restricted form, it was always clear that para, (b) and (c) were intended to raise allegations also of failure to prescribe the alternative forms of treatment therein referred to. It was also made clear as the proceedings progressed that by the term "palliative care" in para, (b), the plaintiff intended to refer to any form of treatment of the plaintiff's conditions which did not involve the use of blood products, and reference was made in particular to the withholding of any form of treatment (a mere wait and see approach), the use of ice packs, and the use of a substance referred to as DDAVP to raise the level of clotting factor in the plaintiff's blood. It also became apparent that in support

of the allegation that treatment without blood products was possible, the plaintiff sought to rely in the first instance on an assertion that such treatment was not called for because no condition was diagnosed appropriate to be treated with blood products. This last assertion can henceforth be ignored in view of the finding as to diagnosis which I have already made.

It was also made clear and the case was conducted on the basis that the phrase “dedicated blood” used in the particulars was intended to be understood as referring both to blood drawn only from specially selected groups of donors, which might include donors having some particular relationship with the plaintiff, whether by family, extended family, friendship or community, and to blood drawn from donors who belonged to particular groups of which it could be postulated that they were low risk groups for infection.

The allegation (d) “failed to enter into arrangements or agreements with blood collection agencies which would facilitate or positively implement the screening of donors at point of collection” parallels the allegations of negligence made against the second and third defendants to which I will now refer.

The negligence alleged against the second defendant was particularised as follows:

> “Having within its means of knowledge the knowledge that the infusion of blood products manufactured and supplied by it could result in reception by the ultimate donee of a blood borne virus the second defendant
>
> > (a) failed to enter into arrangements or agreements with blood collection agencies which would facilitate or positively implement screening of donors at point of collection;
> > (b) failed to disseminate information to ultimate and intermediate users of blood products warning of the risk of infection with blood borne viruses.”

The negligence particularised against the third defendant is as follows:

> “Having within its means of knowledge the knowledge that the infusion of blood products manufactured and supplied by it could result in reception by the ultimate donee of a blood borne virus the third defendant
>
> > (a) failed to implement a system whereby it screened donors at point of collection to remove from its manufacturing process blood which was likely to contain blood borne virus;
> > (b) failed, having collected such blood to prevent its use in the manufacture of products for the treatment of haemophilia (Factor VIII deficiency) namely, lyophilized Factor VIII and cryoprecipitate;
> > (c) failed to disseminate information to ultimate and intermediate users of blood products warning of the risk of infection with blood borne virus.”

It was throughout the proceedings clear that the reference in the particulars to a system of screening of donors was intended to embrace two distinct measures — namely, excluding from the donor pool persons who were members of groups identified as high risk groups whose blood was likely to carry infectious viruses, and also, testing of collected blood by appropriate means to identify infected blood.

It was at all times made clear and the case was conducted upon the basis that the plaintiff alleged against the second and third defendants that each was negligent in distributing blood which it had obtained from unscreened donor sources.

It is to be noted that there is no allegation against either the second nor third defendant of negligence in the method of manufacture of the blood products manufactured by them. In particular, it is not suggested that there was any precaution which they ought to have adopted at any relevant time but failed to adopt which, if adopted, could have been effective to eliminate from AHF products manufactured by them any virus, including HIV which was present in the plasma source. This obviates the need to examine at all evidence as to the process of manufacture and as to the steps which became available to kill viruses present in plasma. Dr Mozen in particular gave evidence of experiments directed towards and the eventual development of means of heat treating Factor VIII concentrate so as to kill viruses present therein. His evidence was that blood products thus treated in the hope of destroying HIV were first distributed in March 1984 although the virucidal effectiveness of the process could not at that date be demonstrated. There is no suggestion on the

part of the plaintiff that the defendants were negligent in failing to adopt heat treatment of their product at any time relevant to this action.

The history previously set out of the development of the AIDS epidemic and the development of knowledge about the disease, its incidence and aetiology makes it clear that these allegations of negligence have to be separately examined in respect of March 1982 and in respect of September 1983. The growth of knowledge between those two dates may have the consequence that a risk which could reasonably be ignored as at March 1982 could no longer be ignored as at September 1983. The starting point must be to identify and quantify what was, at each of those relevant dates, the foreseeable risk against which it is said that the defendants had a duty to take precautions.

10. March 1982 — the risk

Notwithstanding some careless statements by Dr Kendall suggesting the existence of relevant knowledge at an earlier date, it is abundantly clear that there was no evidence at all that AIDS was transmissible in blood before the *MMWR* report of 16 July 1982. Before March 1982, information about the disease was very limited. It was not even certain that there was a new disease, that is to say, that the observations made of the existence of Pneumocystis pneumonia and Kaposi's sarcoma in groups of homosexual men had any common origin. Obviously there were strong grounds for suspicion that they did, but little or no information as to what the nature of that common origin might be. It was not until July 1982 that any ground arose at all, so far as the evidence before me shows, for even the faintest suspicion that a blood borne infection might be involved.

It is therefore quite impossible for the plaintiff to succeed on the basis that in and before March 1982 any of the defendants ought to have foreseen and guarded against the risk that the plaintiff might, through Factor VIII therapy, become infected with, specifically, the agent causing the outbreak of immune deficiency illnesses in homosexuals.

The most that the evidence establishes is that there was in March 1982 and in the period before that during which blood products then administered were being manufactured, a foreseeable risk that recipients of blood products would become infected with blood borne viruses as yet unknown and unidentified.

The risk thus described cannot be said to be far-fetched or fanciful. Until the identification of the AUS antigen the major known risk associated with infusions of AHF, given that syphilis and malaria could be screened out by serologic tests, was liver disease — viral hepatitis. Following that discovery it became possible from about 1973 to test for and screen out hepatitis B, as viral hepatitis was then known; and this undoubtedly represented a major advance in the safety of the use of AHF. It was, however, not long before it became apparent that blood products were in many instances transmitting a virus different from hepatitis B — patients were revealing upon laboratory tests liver disease notwithstanding the exclusion of hepatitis B. Blood products were transmitting a different form of viral hepatitis, a virus which became known as hepatitis non A/non B. That disease or category of disease has been further investigated and it is clear now that hepatitis non A/non B (which was always, as Dr Gatenby pointed out, "a diagnosis of exclusion") in fact comprises several different diseases; one is now tagged hepatitis C; Professor Gust described also hepatitis D and hepatitis E. When in due course it was established that AIDS was the result of a virus capable of transmission in blood and blood products, physicians and blood scientists may have been shocked or horrified but they could hardly have been surprised. They might, before 1982 have hoped that such would never occur: but no one could have said that the risk of its occurrence was far-fetched. It has to be said that it was and was known to be a real risk. It was not such a risk as reasonable manufacturers of AHF would disregard — if there were available to them any reasonable means of averting it.

Nevertheless it could not be said on the basis of any information available to physicians or blood scientists or blood transfusion services that the risk was other than slight. In 1982, the science of blood transfusion was about 50 years old. AHF and other plasma-derived products had been developed many years before and were in constant use in hundreds of thousands of patients worldwide. Since manufacturers had learned to exclude hepatitis B, hepatitis non A/non B appeared to be the only new virus to come to notice. While the emergence of another virus could always be seen as a possibility, there was no particular reason to anticipate that if it had not come under notice already it would do so in the foreseeable future.

11. September 1983 — the risk

By September 1983, the state of knowledge of AIDS was somewhat more advanced than 18 months earlier. As has been seen, cases of AIDS in haemophiliacs had been reported in July 1982 and the case of the baby who developed AIDS after a blood transfusion had been reported in America in December 1982 and in the U.K. in April 1983. In January 1983, although by no means persuaded that AIDS was the result of a blood borne agent, the blood banking industry in America in conjunction with the National Hemophilia Foundation and government health agencies had come to the conclusion that some precautions against the transmission of AIDS by blood and blood products were appropriate. The disease was under discussion in medical and scientific circles in this country, and much of the relevant literature was available here. Several witnesses, notably Professor Gust, drew attention to significant differences between the United States and Australia in relation to the blood donor populations and the incidence of high-risk sexual and drug use practices, which gave good reason to believe that the American AIDS experience would not be duplicated in this country. On the other hand, a study by Rickard and others, published in *The Lancet*, 11 July 1982 (Exhibit RRR) clearly indicated, as Dr Gatenby pointed out (in Exhibit NNN) that the Australian blood supply was contaminated with hepatitis B and presumably non A/non B hepatitis. Dr Gatenby suggested that "this paper should have warned the authorities responsible for blood collection and for the manufacture of Factor VIII that once any other infection appeared that was associated with the receipt of blood products, then it was highly likely to occur in Australia".

The first Australian case of AIDS was published in April 1983. I have no difficulty in concluding that reasonably informed physicians, scientists and blood transfusion services in this country ought to have been well aware by at latest April 1983 that there was a real risk that among the unknown and unidentified sources of infection which blood and blood products had the capacity to carry must be numbered whatever agent was responsible for the production of AIDS. Indeed, I did not understand any of the defendants to contend to the contrary.

It follows that each of the defendants was, from that time onwards under a duty not merely, in general terms to guard against the transmission of unidentified infectious agents in blood and blood products, but specifically to take reasonable care to prevent injury to the recipients of blood products by the transmission in the same of the possible infectious agent which might cause AIDS.

The risk in March 1982 could fairly be described as slight or remote though by no means far-fetched or fanciful. The risk which was foreseeable in September 1983 was rather more substantial. At the start of that year the tiny incidence of the disease among a substantial population of haemophiliacs in the United States afforded a substantial reason to believe that the disease was not due to a blood borne agent. Professor Holland pointed out that the three July 1982 cases were the only three in a population of about 20,000 haemophiliacs in the country. Dr Sayers pointed out that in January 1983 in a country with three million blood transfusions per annum and 11 million blood donations, there were fewer than 10 cases of AIDS post-transfusion (apart from haemophiliacs) and even as late as November 1984, there were but 46 haemophiliac cases and 81 post-transfusion cases of AIDS recorded in the United States of America. In oral evidence Dr Sayers said that by January 1983 there were but seven haemophiliacs reported with evidence of disease attributable to immune suppression and the one child post-transfusion case: "Against a background of probably in excess of 20,000 haemophiliacs in the United States and something like fifteen million transfusions per year." There was still a great deal to learn about this new syndrome. Nevertheless, by September, what in January had been mere faint suspicions would necessarily have been elevated in the minds of responsible Australian physicians and blood scientists into a strong apprehension because of their knowledge of the demonstrated long incubation period, the undoubted fact that the incidence among haemophiliacs was greater than in the general population (apart from the known risk groups of homosexuals and intravenous drug users) and that in May of that year Dr Montagnier had produced evidence which, if it did not demonstrate the fact, strongly suggested that AIDS was due to a virus.

12. The reasonable response to the foreseeable risk—

(a) screening of donors?

Where a specific viral infection is known to be transmissible in blood or plasma or products manufactured therefrom, the first line of defence against such transmission of that virus is to exclude from the donor pool persons manifesting the symptoms of the particular illness or persons of whom it could be predicated that they belonged to a group within the community likely to have a higher than usual incidence of the particular disease. The second line of defence is the performance of tests upon collected blood to determine the presence of infection: a topic to which I shall return.

In respect of viruses not known to exist or viruses known to exist but in respect of which no test is available to determine their presence in collected blood, the collector of blood can only rely on what I have referred to as the first line of defence and the manufacturer of products from collected blood or plasma likewise can rely only on the blood collector's first line of defence. In March 1982, the existence of the virus HIV was unknown. Clearly there was no laboratory test available. In September 1983, the existence of the virus was strongly suspected but its identification was far from clear. No direct test for it was available. Surrogate testing was (as will be seen) investigated but found not to be practicable. The plaintiff's case is that in those circumstances the third defendant was negligent in failing to exclude from the blood donor pool certain categories of individuals of whom it could be postulated that they were likely to be carriers of viral or other infections unknown.

Whether the screening of donors was a reasonably practicable course which the third defendant should have adopted depends on a number of considerations, the first and not the least of which is whether during that period it could reasonably be said that there was a group sufficiently clearly identified as a risk group and sufficiently identifiable or definable to permit screening.

At its highest, the plaintiff's case that there existed before March 1982 identifiable community groups known to be at high risk of carrying blood transmissible infections rests on the evidence of Professor Dwyer that male homosexuals had a higher incidence of hepatitis; Dr Kendall to the like effect; and Exhibit AAA, an article in 1980 drawing attention to the higher incidence of hepatitis B and a number of enteric infections among male homosexuals in the San Francisco and Los Angeles areas. None of this evidence touched on their propensity to carry or transmit other viral infections.

(Dr Gatenby's remark that by March 1982 male homosexuals were known to suffer infections not commonly seen in the general community seems to have been a reference to the 1981 reports of Pneumocystis carinii pneumonia among homosexuals, and so was not relevant to the present issue.)

Dr Kendall's evidence appears to me to be in this respect unreliable, being tainted by hindsight.

What is left unclear is whether the group which it is said ought to have been screened out in March 1982 is a group defined as comprising all male homosexuals or only those with multiple partners. It does not appear that Dr Dwyer, who was a very careful impressive and undoubtedly expert witness was ever asked really to direct his attention to the difference between the homosexual community as a whole, and those members thereof with a promiscuous lifestyle, the "fast lane" homosexuals as Professor Holland referred to them. Professor Dwyer did say in the course of his evidence in chief:

> "at that time, (i.e. 1981) in the United States there was a lot of careless, indeed, dangerous sexual activity occurring in many gay communities as a result of a sociological revolution that was occurring and at that time, and in the years beforehand, we had seen, as a result of this, a very marked increase in the spread of the hepatitis B virus throughout gay communities. They were very much at risk of contracting this infection and the risk was related to the number of sexual partners which they experienced".

This portion of his evidence was not pursued by counsel and it does not appear to me that at any stage he clearly distinguished between (as risk groups for the transmission of viral infection) homosexuals in general and homosexuals with multiple partners. For him as for every expert witness, the opportunity to express his opinions clearly and precisely had to depend to some extent on the manner in which questions directed to him were framed.

It was put to Professor Penington that from 1 January 1980, "it was known that, for example, male homosexuals were a higher risk group of the transmission of blood transmittable infections". Professor Penington's answer was:

> "I don't think I would accept that as a straight statement. It was not male homosexuality which in particular was identified with disease, it was the promiscuous lifestyle within the male homosexual community which was associated with a high incidence of sexually transmissible disease, and I would say by the start of that period, by 1980, it would be known that they would have a higher incidence of hepatitis B as well as other sexually transmissible diseases, but that is the promiscuous male homosexuals, not a commentary on male homosexuality as such. The same would apply, as I have said before, to female prostitutes and their male clients."

Professor Penington went on to point out that persons at high risk of transmitting blood borne infections included also, and were known as early as 1980 to include, intravenous drug abusers, their sexual partners, the sexual partners of promiscuous homosexual males, and the clients of female prostitutes. He pointed out as well that there were certain ethnic or racial groups with a much higher incidence of endemic liver disease — Aborigines, certain persons of Mediterranean origin, certain races from south-east Asia. So, he said, "it becomes a very wide group within the community".

It seems to me that the evidence does not establish the existence as at March 1982 of an identifiable group who could be said to be at high risk for the transmission of unknown viruses, as distinct from the transmission of viral hepatitis. In any event there would seem to be some obvious practical difficulties in attempting to identify with any certainty, so as to exclude them prior to their donating blood, members of the very wide group or groups to which the evidence points. Exclusion of donors on the basis of current symptoms would no doubt be reasonably effective so long as the symptoms were discernible by the blood collection personnel. But if the symptoms were not directly discernible, the effectiveness of the screening process must depend on the honesty and no doubt the awareness of each potential donor. No doubt if it was desired to exclude persons who were users of intravenous drugs, inspection of their arms for needle marks would allow the exclusion of many of them; but whether needle marks on other parts of the body would be readily detectable may be another question. In respect of such a group also the honesty of the donor's responses would be important. Exclusion of donors on the ground that they were members of a particular high risk group would also ordinarily depend upon their awareness (which might often be incomplete) and honesty (which could not be assumed in respect of all of them). It is I think realistic to assume that not every intending donor asked to disclose whether or not he is a member of a presumed high risk group will answer honestly. This has always been a matter of concern when screening of donors has been discussed — see for example, Exhibit G, reporting on the Atlanta meeting in January 1983:

> "Whether members of risk groups will identify themselves remains to be seen."

It is well known that various community or employment groups have been accustomed to go as a group to donate blood. Whether in such circumstances every practising male homosexual would be prepared to face the possible curiosity of his colleagues as to his reasons for declining to donate blood is a matter of speculation; so too, whether he would be prepared to disclose his reasons and face whatever embarrassment might ensue. That such concerns are not far-fetched is illustrated by the facts in *Jones* v. *Miles Laboratories Inc.* (1988) 700 F. Supp. 1127. A particular donor disclosed to his treating physician, just before he died of AIDS, that he was a homosexual. It appeared that he made a large number of plasma donations during 1982 and 1983. During 1982 and on the first two occasions in 1983 he was not asked whether he was a homosexual. However, he was asked that question on every other occasion (at least 25 occasions) in 1983 when he donated. He responded in the negative every time. On two occasions he was asked to sign a form verifying that he was not a homosexual, and on both occasions did so. Those forms stated specifically that the reason the verification was being sought was because of concern that a homosexual would be more likely to be an AIDS carrier.

The plaintiff has failed to satisfy me that there was any reasonable prospect that screening of donors by questionnaire or interview in and before March 1982 would have been effective to eliminate from the donor pool members of so-called high risk groups.

There is no evidence, so far as I can see, that exclusion of persons known to be at high risk for hepatitis B could have been seen prior to March 1982 to be an effective way of eliminating from the donor pool persons likely to carry viruses of a different type then unknown and unidentified. The survey undertaken by Professor Gust (Exhibit 320) showed no correlation between the presence of HIV and the presence of antibodies to hepatitis B core antigen; also Professor Penington was of opinion that although homosexuals may be at high

risk of both hepatitis and HIV, it cannot be assumed that a person who carries hepatitis B is likely also to carry the AIDS virus.

There were, in any event, other reasons explained by Professor Penington and which I accept, for not taking steps to exclude members of such groups from the donor pool. The background which must be borne in mind is that serological tests existed for the determination in the blood of infectious malaria, venereal diseases, and hepatitis B. It was the practice to exclude persons who were detected as intravenous drug users or who admitted to being such, but no other steps were taken apart from that. Asked why not, Professor Penington said:

> "It was not considered appropriate, given that screening procedures were in place for hepatitis B. Screening procedures were in place for the detection of hepatitis B by testing for the surface antigen of hepatitis B which is the principal agent transmitted by blood in blood transfusions and that was a laboratory screening procedure which was well established, carefully monitored in terms of quality control, and that was also used, re-used in monitoring all of the blood products which were produced."

The reference was to a process of testing the blood after it had been taken from the donor. Professor Penington went on:

> "The further reason why further steps were not taken to exclude any group in the community that might have a higher than normal incidence of certain diseases was because we had a totally volunteer system and we wished to encourage people to come forward from the community to voluntarily donate blood and it was seen as inappropriate to put barriers to donation unless there was strong reason to do so."

Because of the width of the group which would have to be excluded, "the dependence was one of relying upon the laboratory screening process rather than narrowing down to a very considerable extent those in the community who would be regarded as eligible to donate".

Professor Penington emphasised that the situation which had existed in Australia is that "we were always short of blood supply to meet the demand, where we were always seeking additional donors and never had a sufficient panel of donors to meet comfortably all the requirements, all the demands being made on the transfusion service". Elsewhere in his evidence he spoke of the difficulty, to which other witnesses also testified, in getting sufficient antihaemophilic factor to enable elective surgery to be carried out as and when required. In those circumstances, he said, "it was considered that the screening tests of the blood would identify the serious diseases which could be transmitted, hepatitis B and syphilis being the two most important, and that given that those could be excluded by laboratory screening, there was no reason to seek to restrict the panel of people from the community coming forward voluntarily to donate blood".

He agreed that despite the screening test which could be applied, there was a significant incidence, about 2%, of post-transfusion hepatitis, which was always a matter of concern and to the elimination of which attention was always directed. The problem was, not so much hepatitis B which ordinarily was screened out by the surface antigen test to which reference has been made, but a further form of post-transfusion hepatitis which, when first discerned about 1977, was named non A/non B hepatitis. The risk of the transmission of such infections was known and research continued in order to identify the viruses involved, to develop tests by which their presence could be detected, and also to develop means (e.g. by heat treating of which Dr Mozen gave evidence) to destroy viruses in collected blood and manufactured blood products.

It is the fact, as the evidence shows, that the incidence of hepatitis B is higher among promiscuous male homosexuals than in the general community. It is also the fact, now known and becoming known in 1981, that the incidence of AIDS is higher in the homosexual community generally and *a fortiori* among promiscuous homosexuals. It may now be seen with hindsight that if homosexuals had been excluded from the blood donor pool prior to March 1982, the probable effect would have been to exclude from the donor pool most if not all persons at that time likely to be carrying the AIDS virus. That that was so was not and could not have been known at any time prior to March 1982.

The plaintiff has failed to persuade me that the exclusion of particular groups from the blood donor pool was a reasonably practicable means prior to March 1982 of protecting blood product recipients including the plaintiff from infection by blood borne disease; and has failed to persuade me that the risk of infection

from unknown viruses was such that a reasonable blood banker would have thought it necessary to attempt such exclusion at that time. The plaintiff has failed to persuade me that the third defendant was negligent in failing to take steps to exclude homosexuals or any other identifiable group from the blood donor pool prior to March 1982 or that the second defendant was negligent in accepting for the purposes of AHF manufacture plasma drawn from a donor pool from which such groups had not been excluded.

As has been seen, by September 1983, the risk of HIV transmission by blood products was more clearly recognisable. By September 1983 it was well known that there was a significant risk that blood products might transmit AIDS, for although the nature of the causative agent was still unclear, there was evidence that it could be a blood borne virus. Furthermore, it was well known that AIDS was a disease which had first manifested itself in the homosexual community and that the incidence of the disease in the homosexual community was much higher than in the community at large. It was submitted therefore that by mid-1983 it was a reasonable precaution for the third defendant to have taken, that all male homosexuals be excluded from the donor pool.

The evidence shows that screening of donors commenced in some parts of the United States at the very end of 1982 and more widely as 1983 progressed. There does not appear to have been a uniformity of practice nor a consensus as to what group should be excluded. From late 1982 onwards, the National Hemophilia Foundation was urging Factor VIII suppliers to exclude from the production of Factor VIII concentrate any plasma donations from, among others, homosexuals. By 30 December 1982 the Mount Sinai Medical Centre in New York was “making a major push to eliminate from the donor pool potential AIDS candidates such as homosexuals …”, and it was disclosed at the Atlanta meeting in January 1983 that some commercial plasma collection centres were excluding plasma from homosexuals. There does not appear to have been at that stage any emphasis placed on the distinction between homosexuals in general and homosexuals with multiple partners. Two days after the Atlanta meeting, a meeting was held in Washington of the members of the American Association of Blood Banks Transfusion Transmitted Diseases Committee. The upshot was the publication of a joint statement on 13 January 1983 (Exhibit 301) (Exhibit 217 (15)). The statement emphasised that the possibility of blood borne transmission of AIDS was still unproven but said:

> “The finding of cases in haemophiliacs especially those who use AHF concentrate coupled with the long incubation period and the continuing increase in reported cases is of sufficient concern to warrant the following suggestions for action on the part of blood banks …”

It went on to make recommendations for increased caution in the use of blood, that is to say, restricting its use to situations where no other treatment was available, and for the avoidance of donor recruitment from groups which might have a high incidence of AIDS. No recommendation was made as to the screening of donors to eliminate from the voluntary donor pool persons who were members of those groups in the community identified as being high risk groups for AIDS, although it was contemplated that such recommendation might be made in the future “should evidence of a clearly implicated donor population become apparent”.

The evidence of Professor Holland and of Dr Sayers was that in early 1983 in the United States the practice being followed was the exclusion not of homosexuals as such, but of those with multiple partners. On the other hand, when Dr Dietrich gave evidence on the same topic, she indicated that by spring of 1983 members of high risk groups were being asked to refrain from making donations, and she identified homosexual and bisexual men as such without reference to multiplicity of partners. Professor Holland, however, limited the group to “very sexually active gay men” and his evidence at p. 338 contained this passage:

> “Q. You heard Professor Dwyer talking today about a group which he called homosexual males. Do you agree that that group should be classified as homogeneous? A. No. That was one of the problems early on as to what was the definition of a homosexual male. It was clear that for this disease AIDS what we were talking about were those gay men who were extremely sexually active, had many many partners, hundreds and hundreds who appeared to be at high risk of this disease because other gay men who either were monogamous or who just did not like women and felt that they were gay who had no sexual activities clearly they were not developing this disease.

> Q. So between 1982 and say 1984 how did the description of homosexual males so far as AIDS was concerned change if at all? A. It changed very little until 1985 and 1986. That is, from '83 into '85 basically early '85 we were looking for and trying to rule out those gay men who were quite sexually active and who had more than one sexual partner."

This discrepancy in the evidence reflects the different views and practices in the United States revealed by some of the documentary material. Exhibit 224 is the *MMWR* of 4 March 1983 which lists as "persons who may be considered at increased risk of AIDS, 'sexually active homosexual or bisexual men with multiple partners'". On 24 March 1983, the director of the Office of Biologics in the U.S. National Centre for Drugs and Biologics (Food and Drug Administration) circularised licensed manufacturers of plasma derivatives:

> "Plasma collected from donors suspected of being at increased risk of transmitting AIDS (as presently defined: persons with symptoms and signs suggestive of AIDS, sexually active homosexual or bisexual men with multiple partners, Haitian entrants to the United States, present or past abusers of intravenous drugs and sexual partners of persons at increased risk of AIDS) should not be fractionated into derivatives already known to have a risk of transmitting infectious diseases … We request that you immediately institute procedures with your plasma suppliers to assure that they have adopted appropriate donor screening practices and procedures."

The U.S. Department of Health and Human Services issued a statement on 25 March 1983 recommending educational programs to inform persons with increased risk of AIDS that they should refrain from donating plasma or blood. The statement said:

> "Persons at increased risk of AIDS are defined as those with symptoms suggestive of AIDS, sexually active homosexual or bisexual men with multiple partners, recent Haitian immigrants, present or past abusers of intravenous drugs and sexual partners of individuals at increased risk of AIDS."

Elsewhere the publication asserted:

> "The disease has primarily affected homosexual males with multiple sexual partners …"

In February 1983, both Dr Schiff of CSL and Dr Archer of the blood bank became aware of the publications in the *New England Medical Journal* of 13 January 1983 ar.d both took steps to bring them to the attention of the National Blood Transfusion Committee with a view to the question of AIDS transmission in blood and blood products being brought up for discussion at the forthcoming meeting of the Blood Transfusion Service Executive Subcommittee. That subcommittee met on 3 and 4 March 1983 and resolved that a survey of the immune status of haemophiliacs be carried out at Royal Prince Alfred Hospital, Sydney and Royal Children's Hospital, Melbourne, to ascertain the state of affairs in Australia. The matter was referred to the next meeting of the working party on Factors VIII and IX concentrates scheduled for 27 May. However, without waiting for that working party to meet, Dr Archer made a public request through the media that male homosexuals in Sydney not donate blood. Thereafter the matter was the subject of considerable publicity and public debate. Some elements of the homosexual community complained of unfair discrimination and the Blood Transfusion Services premises in Sydney were picketed. During May, Dr Archer and members of his staff met with a deputation from the homosexual community and in the course of that meeting it was agreed that potential blood donors should be given written guidelines which would facilitate self-exclusion of high risk persons. At some time in the month of May such a document was prepared and a revised version of it was issued in June, those being Exhibit CC in the proceedings. These documents drew to the attention of donors that, there being no laboratory test for AIDS, it was necessary to rely on the donor's health history to exclude individuals whose blood might transmit AIDS. Persons who thought they might fall within one of the categories described in the document as "persons at particular risk" were asked to speak to a medical officer or, in the later version of the document, to refrain from donating unless positively advised that it was all right to do so. The documents identified persons at particular risk as being those with symptoms and signs suggestive of AIDS, "sexually active homosexual or bisexual men with multiple partners. The risk increases as the number of partners increases", intravenous drug users and sexual partners of any of those groups.

The Commonwealth Government's *Communicable Diseases Intelligence Bulletin* of 3 June 1983 (Exhibit Y) contained an article by a representative of the Commonwealth Department of Health which recorded that "current epidemiological evidence has identified several groups at increased risk of developing AIDS; homosexual or bisexual men with multiple sexual partners …". On 30 May 1983, the Commonwealth

Department of Health wrote to Dr Archer of the blood bank a letter which is Exhibit EE, which included this statement:

> "People at greatest risk are basically homosexual men and bisexual men. The risk increases as the number of partners increases. Only a very small percentage of people at high risk develop the disease. Intravenous drug users are also at risk."

On 27 May 1983 there was a meeting of a working party of the Australian Red Cross Society on Factor VIII and IX concentrates at which were present among others Dr Schiff of CSL and Professor Penington, the chairman of the National Blood Transfusion Committee. It was noted that, "it was clear that certain groups at least should be discouraged from donating their blood", and it was proposed to issue guidelines for the Blood Transfusion Service and also a press statement aimed at public reassurance. The press statement was issued on 2 June (Exhibit 308, pp. 38–39) and informed the public that recommendations aimed at minimising the risk of transmission of AIDS through blood and blood products had been issued to blood transfusion services. Those recommendations (Exhibit 308, p. 40) were as follows:

> "The working party on Factor VIII and IX concentrates has considered the question of AIDS and its implications for blood transfusion in Australia. It recommends to the National Blood Transfusion Committee that, for the time being, transfusion services not collect whole blood or any of its components from the following categories of potential donors:
>
> 1. Persons with symptoms and signs suggestive of AIDS.
> 2. Sexually active homosexual or bisexual men with multiple partners.
> 3. Present or past abusers of intravenous drugs.
> 4. Sexual partners of persons at increased risk of AIDS, i.e. of persons in the above categories.
>
> Divisional BTS directors are requested to take steps to implement these recommendations within the general ethical principle which apply to all Red Cross activities. How best to do this must be the responsibility of the divisions and their blood transfusion services. Methods suggested include information leaflets for donors, lectures to common interest groups, articles in selected papers and magazines etc.
>
> The working party noted that no case of AIDS in Australia had yet been associated with the administration of blood or blood products. Nevertheless it considered it prudent to err on the side of caution, and to adopt the above recommendations which are based on those of the U.S. Department of Health and Human Services in a form applicable to this country. These recommendations are intended as an interim measure to protect recipients of blood and blood products until specific laboratory tests for AIDS become available."

In all the circumstances the approach adopted by that working party appears to me to have been a reasonable one. Notwithstanding that some opinion in the United States was to the effect that all homosexuals should be excluded from the donor population, there was responsible opinion in support of the view that the risk increased with the number of sexual partners, and that the high risk group comprised the promiscuous homosexuals. I have already referred to the evidence of Dr Penington and Professor Dwyer to that effect. It was the official view of the United States health authorities as at March 1983 and of the Commonwealth Department of Health in June of that year.

Much of course that was there recommended was already in train in New South Wales because of the information sheet for blood donors which Dr Archer had implemented. The third defendant continued to avail itself of newspaper publicity and enlisted the assistance of various homosexual community groups. As Professor Penington put it, media articles at the time were monitored to ensure that the community was being given a correct understanding of the request made by the Red Cross that male homosexuals with multiple partners desist from donating blood. On 15 August 1983 a public forum was arranged at Paddington Town Hall and Professor Penington was one of those who addressed some 300 people, principally of the homosexual community. He said:

> "I spoke about the nature of AIDS, what information was available about AIDS, the spread of AIDS in the United States and at that time outside of the United States; the need for precautions to protect the

community against the spread of AIDS, including the gay community and, in particular, the need to safeguard the blood transfusion service from donations from people who might be at risk of carrying what we believe to be an infection likely to be the cause of the disease.

Q. And was that particular statement made in relation to a particular group of the homosexual community? A. Well, it was a statement to all of those present, seeking their co-operation given that we had a volunteer donor system, that any person who belonged to the category which was clearly identified as at risk of having a tendency to AIDS that they refrain from donating blood.

Q. And was that the group referred to in your statement of June 1983, the active and multiple partner group? A. That's correct. That was explained to the meeting, that any member who had more than one partner, that is multiple partners, should refrain from donating blood, or if their partner had more than one partner."

It does not appear to me that the plaintiff has established that there was anything more in the way of donor screening which reasonably could and should have been done by the third defendant during the period January to September 1983 to reduce the risk of transmission of AIDS through blood products. What was in fact done appears to me on the evidence to have been a reasonable response to the risk. It was also, in the circumstances, reasonable that the second defendant should continue to accept and process plasma supplied by the third defendant and other blood transfusion services, without demanding any more stringent screening of donors.

13. The reasonable response to the risk—

(b) withdrawal of product

It follows from my finding that neither the second defendant nor the third defendant was negligent in respect of any failure to screen donors prior to 1982 that neither has been shown to have been negligent in continuing, in that period, to use plasma from an unscreened donor pool and to distribute AHF made from such plasma. By September 1983, however, there was a risk of transmission of AIDS in blood sufficient to demand at least such processes of donor screening as in fact were implemented, and so the plaintiff contended that the second and third defendants were negligent in continuing to distribute AHF made before donor screening was introduced or in failing to recall AHF which had been already distributed. The contention was not supported by any evidence in the plaintiff's case that such measures were feasible, and no cross-examination was directed to Dr Schiff or Professor Penington on this topic. Given that there was a scarcely adequate supply of blood and a continuous struggle to maintain a sufficiency of supply of AHF; that AIDS transmission by blood and blood products was still unproven although suspected; that there had been no case of AIDS occurring among haemophiliacs in Australia; and given the wide distribution of blood and blood products from the blood bank throughout N.S.W., it seems to me that the Court would be indulging in speculation were it to conclude that recall was a practical and in the circumstances a reasonable measure, in the absence of some evidence to that effect. It is not self evident that recall could or should have been undertaken.

In any event, the plaintiff has not adduced any evidence of the date of manufacture of the AHF administered to the plaintiff in September 1983 so as to make relevant any allegation of negligence in this regard.

14. The reasonable response to the risk—

(c) surrogate testing?

Neither in March 1982 nor in September 1983 was it technically possible to test donated blood for the presence of HIV. In circumstances where a direct test for the presence of a particular disease or its antibodies does not exist, resort may sometimes be had to "surrogate testing". The concept depends on the ability to identify particular pathological abnormalities, the presence of which is shown to have high correlation with the presence of the disease sought to be detected. If it could be postulated that all or even most persons carrying the HIV infection would display in their blood increased levels of a particular type of cell, enzyme, or hormone or antibody, or decreased levels, the presence of such an increased or decreased level of such cell, etc., could be regarded as a marker for the presence of HIV — and the exclusion of HIV

infected donors could be achieved by the exclusion of those persons carrying that particular marker. No form of surrogate testing was a possibility in March 1982 or earlier, not only because it was as yet unknown that AIDS was a viral infection, but also because there had been so little research yet done to identify markers for the illness. The possibility of excluding infected donors by means of a surrogate test was discussed at the Atlanta meeting in January 1983. In particular, it was suggested by some that testing for the presence of hepatitis B core antibody might be a suitable surrogate test. The problem with any surrogate test is that if it is not sufficiently specific it will give false positive results, and if not sufficiently sensitive, false negatives. It was feared, at Atlanta in January 1983, that none of the surrogate tests suggested would be sufficiently sensitive and specific: and so it turned out. According to Professor Holland, some 22 surrogate tests were propounded and studied by researchers during 1983, but no workable test was found. One such, a test for alpha interferon levels, was mentioned by Professor Dwyer as one that could have been used: but his own evidence was that it was not until September 1983 that research into that test was published. As to the test most favoured at Atlanta, the presence of hepatitis B core antibody, its validity as a surrogate test for AIDS was eventually evaluated by Professor Gust (p. 786, Exhibit 320). Some 601 blood samples known to carry hepatitis B core antibody were tested for HIV once the laboratory test for that virus became available. Not a single one was found to be positive. That is to say, the use of this test as a surrogate test for HIV in respect of those samples would have caused the discarding of 601 perfectly safe blood donations for no actual benefit whatsoever.

The plaintiff has failed to show that the second and third defendants were in any way negligent in failing to utilise surrogate tests to exclude HIV infected blood from the pool of blood from which they manufactured AHF.

15. The reasonable response to the risk—

(d) warnings

> "If a product is dangerous and the danger is one of which the defendant manufacturer was or should have been aware but not one which justifies removal of the product from the market, the product must be accompanied by appropriate warnings" — *Product Liability in Australia* Cavanagh and Phegan, 1982 p. 167.

Clearly, by March 1982, the second and third defendants were aware (they clearly should have been and neither claimed that it was not) of the well-known risk that despite the screening measures which were in place for hepatitis B and other known diseases, there existed a risk that AHF produced by them might carry and transmit to the recipient hepatitis B, hepatitis non A/non B, and other sources of infection neither known nor identified. In principle, therefore, they had a duty to give an appropriate warning of that risk to those by whom the products might be used. It is clearly not the case that prior to March 1982 there was any obligation on the second or third defendants to give warning of the risk that blood products distributed by them might carry, specifically, the agent responsible for the syndrome recently reported as occurring in American homosexuals. It is equally clear that from soon after the beginning of 1983 the second and third defendants were aware of the risk then becoming known that AIDS might be a viral disease transmissible in blood products and in particular, in Factor VIII preparations. That knowledge, with their inability to do much by way of screening of donors or otherwise to eliminate the risk cast upon them a duty to give an appropriate warning of that risk also.

It is to be borne in mind that these products were in general administered to patients by doctors, usually in hospital. In a comparatively small proportion of cases, Factor VHI concentrate was made available to patients to be kept by them in their own homes and injected in the event of symptoms of bleeding developing. It would, however, come to such patients for that purpose only after they had been diagnosed as haemophiliac and advised by their treating physician. Accordingly, the warning that was necessary was a warning not to the patients but to the clinicians. The latter might reasonably be assumed to be a group well aware because of their own training and expertise of the infection risks attendant upon the use of blood and blood products. Indeed, it is clear on the evidence in this case that the doctors primarily responsible for the plaintiff's care (Dr Lammi and Dr Bryant and their staff) were well aware in and before March 1982 of the

existence of the hepatitis risk and of the general theoretical risk of transmission of infection in blood products and in and before September 1983 of the risk also of transmission of AIDS.

There is no evidence as to whether the products manufactured by the third defendant did or did not carry a warning; there is evidence of the warnings printed on the labels used by the second defendant for its product and those warnings in and before March 1982 appropriately drew the attention of the treating doctor to the risk that the product might transmit the agent responsible for hepatitis. The fact that no express reference was made to the theoretical risk of transmission of other unknown and unidentified viruses was in my opinion in all the circumstances not unreasonable. The labels which are in evidence show no change of language during the year 1983 and no reference at any time during that year to the risk that the product might carry the AIDS virus. Notwithstanding that the labels were designed to be read by clinicians and not by their patients, it seems to me that the manufacturer and distributor of AHF could not reasonably assume that every physician by whom it might come to be administered would be aware of the risks associated with it, and that, given the state of knowledge which the second and third defendants must be taken to have possessed during the year 1983, they ought, acting reasonably, to have included on the label a specific warning of the risk, at least to the extent that it was then known, that the product might transmit AIDS. The plaintiff has failed to establish that the second and third defendants were negligent in and before March 1982 in failing to warn of the infection risk involved in the use of their AHF products; but I am satisfied that in 1983 and before September of that year, failure to warn of the AIDS risk would amount to a lack of reasonable care.

It is, however, in my view quite clear on the evidence that the inclusion in the label as at March 1982 of a warning that the use of the product carried a risk of infection from bacteria or viruses not yet known or identified, and as at September 1983 of a warning of the risk that the product might transmit the agent responsible for AIDS, would have had no effect at all upon the attitude of Dr Lammi, Dr Bryant and their staff to the use of the products for treatment of a haemophiliac with a joint bleed. The risk, such as it was perceived by them to be, was already taken into account in the decision to treat or not to treat with AHF. No warning which may have been given by the second or third defendant could have afforded any significant information additional to that which the treating doctors already possessed.

It well may be the case that the form of warning on the products was such as would not bring to the notice of the patient the existence of any infection risks; but it does not appear to me that the obligation of the second and third defendants went so far. There is no evidence which would suggest that in a hospital the patients themselves would examine the product. The question of warning or no warning in the hospital situation must surely rest not with the manufacturer or distributor, but with the treating physician.

16. Whether the second and third defendants were negligent

If it be the case that the plaintiff received his infection from a product administered to him in March 1982, whether in the form of cryoprecipitate or concentrate, I find that neither the second defendant nor the third defendant so far as relevantly involved was in any way negligent in respect of the manufacture or distribution of the product.

If it be the case that the plaintiff received his infection from a product administered to him in September 1983,1 find that any conduct on the part of those defendants which could be found to have been negligent was not a cause of the plaintiff's injury (i.e. infection).

17. The reasonable response to the risk — the hospital

If as I have held, it was not reasonably practicable for the third defendant as the collector of plasma so to screen the donor pool (except to the extent that it did so in fact) as to exclude members of particular sections of the community said to carry a particularly high risk of the transmission of blood borne viruses, then clearly it was not reasonably practicable for the hospital to insist that it be supplied only with AHF manufactured from plasma derived from a donor pool more completely screened. That disposes of particular (d) in the plaintiff's particulars of negligence against the first defendant.

The plaintiff's case against the first defendant raises the following issues:

> (a) whether at the time of each of the treatments given, other forms of treatment were available, practicable and appropriate;

(b) whether at the times when treatment was given the risk of transmission of blood borne infection was such that in the exercise of reasonable care for the safety of the plaintiff the defendant should not have prescribed treatment with AHF;
(c) whether at the times when treatment was given the risk of transmission of blood borne infection was such that in the exercise of reasonable care for the safety of the plaintiff the defendant should have warned the plaintiff and his parents of the risk and given them such information as would have enabled them to make an informed choice between alternative forms of treatment;
(d) whether the defendant failed to give such warnings and information as were appropriate;
(e) whether any such failure to warn and give information was causative of the plaintiff's injury.

The test which determines whether in a particular case a medical practitioner is to be regarded as negligent in matters of diagnosis and treatment was laid down by *McNair* J. in *Bolam* v. *Friern Hospital Management Committee* (1957) 1 W.L.R. 582 in terms which have repeatedly been approved by the House of Lords and by superior courts in this country. In *Sidaway* v. *Board of Governors of Bethlem Royal Hospital* (1985) 1 A.C. 871, Lord *Scarman* said:

> "The *Bolam* principle may be formulated as a rule that 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 the duty of care: but the standard of care is a matter of medical judgment."

In *Sidaway* the majority of the House of Lords held that the question whether an omission to warn a patient of inherent risks of proposed treatment constituted a breach of the doctor's care towards his patient was in general to be determined by application of the *Bolam* test. Lord *Diplock* said at p. 895:

> "No doubt if the patient in fact manifested this attitude" (viz. that he wanted the fullest information so that he could form his own judgment as to whether to refuse the advised treatment or not) "by means of questioning, the doctor would tell him whatever it was the patient wanted to know; but we are concerned here with volunteering unsought information about risks of the proposed treatment failing to achieve the result sought or making the patient's physical or mental condition worse rather than better. The only effect that mention of risks can have on the patient's mind, if it has any at all, can be in the direction of deterring the patient from undergoing the treatment which in the expert opinion of the doctor it is in the patient's interest to undergo. To decide what risks the existence of which a patient should be voluntarily warned and the terms in which such warning, if any, should be given, having regard to the effect that the warning may have, is as much an exercise of professional skill and judgment as any other part of the doctor's comprehensive duty of care to the individual patient, and expert medical evidence on this matter should be treated in just the same way. The *Bolam* test should be applied."

Lord *Keith of Kinkel*, Lord *Bridge of Harwich* and Lord *Templeman* added, however, a qualification, namely that there might be circumstances where the proposed treatment involved a substantial risk of grave consequences in which a judge could conclude that, notwithstanding any practice to the contrary accepted as proper by a responsible body of medical opinion, a patient's right to decide whether to consent to the treatment was so obvious that no prudent medical man could fail to warn of the risk save in emergency or where there was some other sound clinical reason for non-disclosure. Lord *Scarman* took a different view, namely, that:

> "English law must recognise a duty of the doctor to warn his patient of risk inherent in the treatment which he is proposing: and especially so, if the treatment be surgery. The critical limitation is that the duty is confined to material risk. The test of materiality is whether in the circumstances of the particular case the court is satisfied that a reasonable person in the patient's position would be likely to attach significance to the risk. Even if the risk be material, the doctor will not be liable if upon a reasonable assessment of his patient's condition he takes the view that a warning would be detrimental to his patient's health."

In *F* v. *R* (1983) 33 S.A.S.R. 189, the question was whether the failure of a medical practitioner to inform a woman of the remote possibility of a tubal ligation operation being unsuccessful was a negligent breach of his duty to her. *King* C.J. said at p. 193:

> "Finally the question must be: 'Has the doctor in the disclosure or lack of disclosure which has occurred, acted reasonably in the exercise of his professional skill and judgment, or, as *Bristow* J. put it in *Chatterton* v. *Gerson* [1981] 1 All E.R. 257, in the way a careful and responsible doctor in similar circumstances would have done?' In answering that question much assistance will be derived from evidence as to the practice obtaining in the medical profession. I am unable to accept, however, that such evidence can be decisive in all circumstances."

His Honour continued at p. 194:

> "In many cases an approved professional practice as to disclosure will be decisive. But professions may adopt unreasonable practices. Practices may develop in professions, particularly as to disclosure, not because they serve the interests of the clients, but because they protect the interests or convenience of members of the profession. The court has an obligation to scrutinize professional practices to ensure that they accord with the standard of reasonableness imposed by the law. A practice as to disclosure approved and adopted by a profession or a section of it may be in many cases the determining consideration as to what is reasonable. On the facts of a particular case the answer to the question whether the defendant's conduct conformed to approved professional practice may decide the issue of negligence, and the test has been posed in such terms in a number of cases. The ultimate question, however, is not whether the defendant's conduct accords with the practices of his profession or some part of it, but whether it conforms to the standard of reasonable care demanded by the law. That is a question for the court and the duty of deciding it cannot be delegated to any profession or group in the community."

In *Ellis* v. *Wallsend District Hospital* (1989) Aust. Torts Reports ¶80-259 the Court found it unnecessary to consider whether or not the trial Judge, *Cole* J., was correct in basing himself upon Lord *Scarman's* speech in *Sidaway* and the decision of the South Australian Full Court, especially the judgment of *King* C.J., in coming to his conclusion that the surgeon involved in that case was in breach of his duty to warn. Were it necessary for me to come to a specific conclusion, I would, as did *Cole* J., prefer the views of *King* C.J. and Lord *Scarman*. As will appear hereafter, it does not seem to me in the present case to make any difference.

(a) Prescription of other forms of treatment — the "no treatment" option

I have already found as a fact that the diagnosis of haemarthrosis both in March 1982 and in September 1983 was a correct diagnosis. That being so, there is a powerful body of medical opinion before me that there was no realistic treatment option other than the administration of antihaemophilic factor. At its highest, the plaintiff's case to the contrary was expressed by Professor Dwyer and he put it no higher than this:

> "March of 1982 I would not have expected the clinician caring for H to have been worrying about the possibility of AIDS transmission but in terms of general management, they would obviously have been concerned and in this exercise of decision making they would be weighing up the possible risks in terms of, say, hepatitis transmission, of needing blood products versus the clinical needs of the child at that stage and how important it was that they give this material which would stop any further bleeding and perhaps damage to the joint. In fairness I must say that I was not there and did not see the clinical situation. These notes are sparse and I am not experienced in the treatment of children with haemophilia. Others therefore could comment better than I as an experienced physician. This certainly looks like a situation where it may be that conservative treatment, withholding of this blood may have been reasonable, but I can say no more."

He went on to say that there were other investigations available which might have clarified the diagnosis but he did not suggest that, if the diagnosis of bleeding was correct (as I have found that it was) AHF should not have been used. Dr Kendall speculated as to whether alternative treatment could and should have been instituted. Dr Kendall has virtually no experience in the treatment of haemophiliac patients with joint bleeds. His views about that topic are in my view unreliable, being postulated on the proposition that the plaintiff was only a "mild" haemophiliac. As indicated earlier, I accept the evidence that once bleeding has commenced, the classification of the plaintiff as mild or otherwise is of little significance.

All of the specialist haematologists who gave evidence, persons involved regularly in the treating of haemophilia, conscious of the dire consequences of failure to treat a joint bleed, were firm in their view that once the diagnosis of joint bleeding had been made, AHF therapy must be given. I am satisfied that both in March 1982 and in September 1983, the condition of the plaintiff was such that reasonable medical care required the administration of AHF and no such physician would have regarded the no treatment option as realistic, unless there were some powerful reason to the contrary. I have earlier characterised the risk of the transmission of an unknown and unidentified blood borne virus in March 1982 as slight and as remote though not by any means fanciful or far-fetched. The risk of transmission of some form of hepatitis was significant and well known. Nevertheless, the common opinion of the specialist haematologists was that the known risk of transmission of hepatitis was far from a sufficient reason to abandon that form of treatment. As I understood their evidence, not one of them thought that the risk in March 1982 of the transmission of any other kind of infection then unknown added significantly to the risk side of the risk/benefit balance to which a clinician must direct himself.

The same group of physicians, conscious in 1983 and prior to September of that year of the growing concern that AIDS might be transmitted in Factor VIII preparations nevertheless did not regard the risk as sufficient to warrant the withholding of Factor VIII therapy for a haemophiliac with a joint bleed. They were and were entitled to be conscious of the very low incidence of AIDS among recipients of blood and blood products and to weigh that against their awareness of the likely dire consequences of non-treatment. They were entitled also to note the strong views expressed by the National Hemophilia Foundation in the U.S.A. which continued in 1983 and into 1984 to urge its haemophiliac members to continue to accept treatment with AHF despite the AIDS risk. Applying the *Bolam*~~Bolam~~ test, I am satisfied that the exercise of reasonable care on the part of the hospital and its staff did not require that AHF therapy be withheld either in March 1982 or in September 1983.

(b) Prescription of other forms of treatment — DDAVP

DDAVP is the short-hand reference to a drug known as desmopressin. It is a hormone preparation which has the characteristic that it stimulates the body's production of Factor VIII. It was originally used in the United States as a means of stimulating the production of Factor VIII in blood donors so that their plasma when extracted would be Factor VIII rich. From that, it was a simple step to extrapolate that DDAVP might be used to increase the Factor VIII level in haemophiliacs so as to avoid the need for AHF therapy. Experiments along that line were carried out in the United States during the early 1980s but at no time prior to September 1983 had the substance been licensed in the United States for use for that purpose. Its licensed use related to the treatment of diabetes insipidus and indeed for that purpose it was in use in New South Wales in the first years of the decade. The suggestion that it might have been appropriate for the hospital to use DDAVP in treatment of the plaintiff rather than to use blood products emanated from Dr Kendall but was not supported by any reference to relevant research reports nor any relevant clinical experience. In fact, during the year 1983 Dr Rickard, haematologist at Royal Prince Alfred Hospital, carried out at the request of the Commonwealth Department of Health an evaluation of DDAVP as a potential replacement in appropriate cases for AHF. His report was delivered late in 1983. DDAVP was not, in that year, in use for the treatment of haemophiliacs at the Royal Prince Alfred Hospital, nor, to Dr Rickard's knowledge, in other major treatment centres in Australia.

The substance was in fact used on the plaintiff on two occasions after the diagnosis of AIDS was made, during periods when he was an inpatient at Prince of Wales Hospital. On one occasion it achieved no more than an increase from 9% to 12% in his Factor VIII assay and on the subsequent occasion from 10% to 12%. In Dr Rickard's view, those are not clinically significant increases. Factor VIII assays are not accurate within plus or minus 3 to 4%. In effect, these results show no effective haemostatic response in the plaintiff and demonstrate that the substance was incapable (in the plaintiff) of achieving Factor VIII levels which would have been required to treat the condition for which he was at the hospital in March 1982 and September 1983. The objective in the case of joint bleeds is to raise the Factor VIII level in the patient's blood by something in the order of 30% (i.e. from 9% to 39%). I accept this evidence.

It is obvious from the experience of the use of DDAVP on the plaintiff more recently that it offered no prospect at all of achieving any therapeutically significant result. The plaintiff has failed to establish that the hospital was negligent in not using DDAVP at any relevant time.

(c) Prescription of other forms of treatment — dedicated blood

The plaintiff sought to establish, not by any evidence in his own case but by cross-examination of experts called by the defendants, that the defendant hospital was negligent in failing to arrange supplies of and treat the plaintiff with what was referred to sometimes as “dedicated blood”, sometimes as “designated blood”.

The concept as raised by Mr Murray on behalf of the plaintiff has a variable content. In some references to it, what was propounded was the use for all child haemophiliacs of AHF manufactured from blood drawn only from specific groups postulated as low risk: i.e. lower risk than the general donor population. Groups that came to be mentioned from time to time thereafter in the case were the police force, the armed services, school children, schoolgirls, and female donors of any age. As to those groups there is no specific evidence that they are indeed lower risk groups. Certainly one could not without specific evidence assume that the police and the armed forces were a lower risk group. As to school children, the evidence shows that consideration could only be given to those of and above the age of 16 because it is not regarded as sound practice to draw blood from younger children. Schoolgirls over the age of 16 may be a group at lower risk than the female population generally: one would prefer to believe so but there is no evidence of it. A female donor population *ex hypothesi* excludes male homosexuals but not the female partners of bisexual men. The group may indeed, should indeed, be lower risk, but could not be assumed to be risk free, so that the effectiveness of proceeding in this way is not self evident. Further, uninstructed by evidence, one would have thought it likely that there might be difficulties encountered in the maintenance of an adequate supply of blood and blood products. If it be the case, as one might assume, that females comprise about one-half of the donor population, and if there has always been, as the evidence shows there has, difficulty in securing an adequate blood supply, it might be difficult to increase the number or proportion of female donors. No doubt it is the case that female blood could be reserved for the manufacture only of those products capable of transmitting HIV, with male blood being used for all other purposes: but in order that I could make a finding that the defendants should have proceeded in that fashion, I would at least need to have some evidence of what was known in 1983 as to the capacity of various blood products to transmit AIDS or the certainty that some would not; as to what proportion of collected blood was in fact used for one purpose rather than another; and, in general, what would be the effect on the overall blood supply of the reservation of female blood for those purposes. In fact, according to Dr Schiff, the use of female-only blood for certain purposes was suggested in mid-1984, at which time females were already approximately 60% of the donor population — evidence which tends to confirm the uninstructed prima facie view that there might be supply difficulties.

I do not attach any significance to the suggestion of Dr Rickard at p. 856 of the transcript that the blood supply would be put at risk by the announcement that male blood was not accepted, causing even healthy non-infected males to be dissuaded from giving blood. No such announcement would need to be made. Obviously, means could readily be adopted to keep male and female blood separate at the point of collection (at p. 824, the transcript omits the word “no” from Dr Schiff’s answer on this point), and there would be no necessity to desist from collecting male blood. Mr Murray argued that, once the possibility of using designated blood in this sense of the term had been raised, it being obviously a theoretical possibility, it was for the defendants to show that it was not feasible. This is a misunderstanding, in my view, of the proposition that when the circumstances are best known to the defendant, the plaintiff may succeed in discharging the onus of proof with but a scintilla of evidence. Suggestions from the bar table do not equate to even a scintilla of evidence. In the words of *Barwick* C.J. in *Maloney* v. *Commissioner for Railways* (1978) 52 A.L.J.R. 292 at pp. 292–293:

> “The likelihood of the incapacitating occurrence, the likely extent of the injuries which the occurrence may cause, the nature and extent of the burden of providing a safeguard against the occurrence and the practicability of the specific safeguard which would do so are all indispensable considerations in determining what ought reasonably to be done. Of all these elements, evidence is essential except to the extent that they or some of them are within the common knowledge of the ordinary man. The fertile but unqualified imagination of counsel or judge can never be a substitute for such evidence.”

There is before me no evidence that it would in and before September 1983 have been reasonably practicable to avoid the risk of transmission of HIV by drawing blood from selected donor groups only.

The second form in which the concept of designated blood was adumbrated represents a much more appropriate use of that term. What is contemplated is blood dedicated to or designated for a particular patient only, being blood provided by his family, his relatives, family friends, or members of some restricted community of which he is a member — his schoolmates for example. Uninstructed by evidence I would see grave logistical difficulties as likely to arise in the treatment of haemophiliacs in this way. It would seem to be necessary that a blood bank or the hospital to which a particular haemophiliac patient would resort for treatment should maintain there a stock of blood sufficient for his foreseeable needs. What that would mean in terms of quantities to be held on hand and in terms of the number of donors and the frequency with which they should be required to donate is not something that the court could determine without specific evidence of experts. One could not conclude that that was feasible without evidence as to how a hospital might be able to maintain individual supplies in that fashion. Evidence would be necessary as to whether a supply of Factor VIII concentrate could be manufactured for the use of a particular patient only or whether it would be necessary in all circumstances to treat him with cryoprecipitate; and if the latter, how that could be managed in the event that he was required to undergo surgery. Perhaps it would be sufficient for the plaintifFs purposes here to show that it was a suitable precaution to maintain a sufficient supply for anticipated periodical treatments other than surgery because it was in the circumstances of such treatments in March 1982 and September 1983 that the plaintiff acquired his infection. Still the question would arise whether it was reasonable to proceed in that fashion given that protection provided in that fashion might be set at nought if protection of the same kind could not be arranged for any surgical procedure which might become necessary or any procedure which might involve massive doses of AHF. One can speculate as to all sorts of answers which might be available to problems of that kind. The only evidence before me on the subject is that of Dr Dietrich and Dr Rickard, both of whom expressed the view that there would be significant logistical difficulties. There is no evidence to the contrary.

There is another aspect of the use of designated blood which is dealt with in the evidence and which provides a further stumbling block for the plaintiff. The rationale of the use of designated blood is the presumed greater safety of the blood supply from which the product is manufactured. There is evidence before me to suggest that in fact groups which might have been thought to be safer and suitable for the use of their blood as designated blood may not in fact be so. Dr Sayers referred to statistical surveys in the United States which have shown that the incidence of blood borne disease in groups selected for dedicated blood transfusion has been at least as high as in the general donor population. Dr Merlin Sayers of the Puget Sound Blood Centre and the University of Washington, Seattle, gave evidence (Exhibit 218) of a study made by the Irwin Memorial Blood Bank in San Francisco:

> “After experience with 11,000 designated donors they felt that directed donors were ‘as safe but not demonstrably more safe than volunteer homologous donors’.”

Dr Sayers pointed out that that conclusion was not supported by statistical analysis but another study with statistical support reached a different conclusion. This was a report in March 1987 of a review conducted by the Washington State Department of Social and Health Science Services:

> “The national survey compared markers for HIV antibodies and hepatitis B surface antigen among 11,547 directed donors and 260,040 homologous donors. It was found that in programs encouraging directed donations, there was no difference in the prevalence of antibodies to HIV. However, 0.242 per cent of the directed donors were hepatitis B surface antigen positive, in comparison with 0.090 per cent of the homologous donors. The difference was statistically highly significant …”

It is also suggested that if the group from which the blood is to be drawn is limited to the close family and relatives of the patient, there still remains a risk that infection will be transmitted because a member of such a group who has successfully over many years concealed his homosexual activity or other dangerous sexual practices may, believing that he is not infected be unwilling to disclose his habits but at the same time unwilling to refrain from donating because if he were to refrain, a suspicion would be aroused as to his reasons. The social and family consequences of such an eventuality might be disastrous. On 22 June 1983 the American Red Cross, the American Association of Blood Banks and the Council of Community Blood Centres published a “Joint Statement on Directed Donations and AIDS” (Exhibit 220). It noted first that the

risk of possible transfusion-associated AIDS was in the order of one case per million patients transfused. It made this comment in respect of directed donations:

> "One consequence of the understandable but excessive concern for transfusion associated AIDS has been requests by patients and their physicians to have blood donors selected from family members, friends, co-workers and even newly formed private donor clubs. There is no evidence to support this notion that these 'directed donations' are safer than those available through the community blood bank.
>
> The concept that family members, friends, co-workers, church members or other selected groups are sure to provide safer blood is unrealistic. These same individuals are and have been the nation's volunteer blood donors who have in the past given freely for all patients rather than for a particular individual. There is no reason to think that segregating these individuals into selected donor panels provides safety over and above the level provided by current arrangements. In addition a system of directed donation may create intense pressures on family and friends who may therefore be untruthful about their ability to meet donor requirements. It is possible that the administrative and operational complexity that will be part of any widespread application of directed donations may lead to a significant increase in clerical errors and, in this way, reduce the safety of transfusion.
>
> Finally there is the risk that widespread attempts to direct donations, while not increasing the safety of transfusions, will seriously disrupt the nation's blood donor system. Voluntary donation is essential for meeting our nation's needs for blood and blood products. There is a real concern that donors may refrain from routine blood donations while awaiting requests to provide directed donations and, thereby, could disrupt the blood supply to the point that routine and even some emergency needs for transfusions may go unmet.
>
> Given these considerations we strongly recommend that directed donation programs not be conducted."

That appears to me to be a realistic assessment and it is one which I accept.

The plaintiff has failed to satisfy me that the provision of designated blood was a feasible option the use of which would have avoided the risk to him of acquisition of the HIV infection as in fact occurred.

(d) Prescription of other forms of treatment — the choice between Factor VHI concentrate and cryoprecipitate

It is asserted on behalf of the plaintiff that the defendant hospital was negligent in choosing to treat him with Factor VIII concentrate instead of making use of cryoprecipitate. This is not an available complaint in respect of March 1982 because on that occasion the plaintiff was treated with both products and, quite apart from the question to which I will later come, whether the evidence shows that in fact it was cryoprecipitate from which the plaintiff received his infection, there is certainly no evidence capable of proving that in March 1982 he received his infection from Factor VIII so that the risk would have been averted by the use of cryoprecipitate on that occasion. In September 1983 he was treated with concentrate only, and so the question whether cryoprecipitate should have been preferred does have relevance.

As has been noted previously, cryoprecipitate is the product of the plasma drawn from a single donor. Factor VIII concentrate is the product of the pooled plasma of a very large number of donors. If both products are made from the same donor population, then quite clearly there are more chances that a batch of Factor VIII will be infected than that a single bag of cryoprecipitate will be infected. It cannot however be postulated, as Dr Kendall was originally inclined to say, that if concentrate is made from the plasma of 2,000 donors, the risk to a patient treated with concentrate is 2,000 times the risk to a patient treated with cryoprecipitate. There are other factors to be taken into account. One is that a patient would never be treated with only a single bag of cryoprecipitate. The Factor VIII level varies from one bag of cryoprecipitate to another but for treatment purposes is usually assumed to be 90 or 100 units. Treatments given to the plaintiff in September 1983 involved respectively 1,080, 675 and 675 units, so that had cryoprecipitate been used, it would have been necessary to use on each occasion at least seven bags. There is also a dilution factor to be taken into account. It is not yet known what dose of HIV must be transmitted to a person to cause him

to become infected with AIDS. If a batch of concentrate contains infected material from one donor only, it is quite possible that given the dilution which occurs, no recipient of concentrate would become infected. Obviously the risk of infection increases accordingly as the number of infected donations increases. In the United States in July 1983 one manufacturer of a Factor VIII concentrate withdrew a batch of concentrate from hospitals because after it was made it was found that some of the donors, apparently at least three as I understand Dr Dietrich's evidence, were infected with AIDS. The product was recalled but before the recall took effect some of it had been used. Somewhere between 70 and 80% of those persons who received concentrate from that batch later developed AIDS (although it cannot be postulated, according to Dr Dietrich, that that batch was necessarily the source of their infection because, as haemophiliacs, they were frequently receiving treatment with AHF from many sources). On the other hand, it is clear that if a single bag of cryoprecipitate is infected, it is virtually certain that the recipient will become infected.

Nevertheless, although the comparative risk cannot be quantified, there was reason, in the United States at any rate, to assume that the risk of infection from concentrate was greater than the risk from cryoprecipitate. A study reported on 13 January 1983 (Exhibit D) compared the immunologic state of two groups of haemophiliacs, one treated with concentrate and one with only cryoprecipitate, and, as I read it (and notwithstanding Dr Dietrich's comment to the contrary, which I reject), showed immunological changes in the former group which were not present in the latter. Exhibit C, published in the same journal, reported similar results. However, although the earliest reports of AIDS in haemophiliacs involved patients who had been treated only with concentrate (Exhibit L), the report in December 1982 (Exhibit M) of AIDS symptoms in a child transfused with platelets showed (if the symptoms were accepted as transfusion related) that a blood product of a single donor could carry the infection.

These considerations led Professor Dwyer to the opinion that by March 1983 at any rate, physicians should have been preferring cryoprecipitate to concentrate if it were possible given clinical requirements, and that in September 1983 it would have been more prudent to treat the plaintiff with cryoprecipitate than with concentrate. Nevertheless the perception of haematologists in Sydney using both products was that from a safety point of view in Australia there was nothing to choose between them, and that view reflected the general perception that the Australian blood supply, relying totally on volunteer donors was significantly safer than the United States blood supply where a great deal of the source plasma used to manufacture AHF was provided by paid donors. There is statistical evidence in the United States to show that the incidence of hepatitis B is higher among paid donors than in a volunteer donor population and various witnesses expressed opinions as to the reason why that should be so. Shortly it comes down to this: that a paid donor population will include persons who by reason of poverty are likely to be less well nourished and therefore more susceptible to infection, and will include drug abusers who wish to sell their blood or plasma to finance addiction. Further, it was postulated the person who needs the few dollars that he can derive by selling his plasma is less likely to answer frankly questions designed to exclude high risk donors.

Even in the United States treating haematologists did not see the AIDS risk in 1983 as a reason for changing established treatment practices and abandoning concentrate in favour of cryoprecipitate. Furthermore, as noted above, the National Hemophilia Foundation in the United States continued in repeated newsletters through 1983 and into 1984 at least to urge its haemophiliac members to continue to accept treatment with concentrate notwithstanding the AIDS risk.

From a clinical point of view, concentrate was recognised as the superior product. A much smaller volume needed to be infused to produce the same haemostatic effect. The method of manufacture of concentrate permitted the Factor VIII content to be assayed and marked on the package whereas the system of manufacture of cryoprecipitate precluded assay and the Factor VIII content might vary from as low as 60 units to something over 100 units. It was necessary for treatment purposes to make an arbitrary assumption of an average Factor VIII level of 90 units but not possible to know until after the treatment had been given what effect it would actually have. Cryoprecipitate contained impurities which were known from time to time to produce adverse allergic reactions whereas in the process of manufacture of concentrate most of those impurities were removed. Both preparations contained fibrinogen but because of the need to administer much greater quantities of cryoprecipitate to produce a given haemostatic effect, the amount of fibrinogen introduced to the patient's body would be very much larger if cryoprecipitate were used. There was concern that the infusion of significant amounts of fibrinogen might generate within the patient antibodies to Factor VIII itself. It was known already in the 1980s that 10 to 15% of haemophiliacs had developed antibodies

to Factor VIII, apparently by reason of the presence in AHF preparations of fibrinogen and other proteins. Hence there were very powerful clinical reasons for preferring concentrate.

It appears to me that Professor Dwyer summed up the situation in which a treating doctor might have found himself in September 1983 when he said that it would have taken a considerable extrapolation on the part of such a physician to enable him to conclude that a product made from pooled blood was likely to be more dangerous than a product from an individual donor.

Dr Lammi and Dr Bryant, treating at the hospital, adopted the stance that there was no relevant difference between the two products and that each might be used according to availability with, however, a preference in favour of concentrate because of its greater reliability and predictability. The same view was espoused by Dr Rickard.

I accept the view that there was no sufficient reason at any time up to and including September 1983 for the hospital treating doctors to have taken the view that Factor VIII concentrate afforded a significantly less risk than cryoprecipitate of transmission of the AIDS virus and that they were not negligent in prescribing concentrate in that period. Furthermore, research since 1983 and in particular since the test for HIV became available has demonstrated the presence of the virus in patients treated with cryoprecipitate, as well as in those treated with concentrate, and although there are suggestions that the incidence is less among the former group, it could certainly not be said in the light of present knowledge that had the hospital treated the plaintiff in September 1983 with cryoprecipitate only instead of concentrate, the risk of transmitting HIV to him would probably have been avoided.

(e) Warning of the risk

The specialist haematologists who gave evidence would, as I understand it, in March 1982 have thought it appropriate that their patients be informed of the slight risk of hepatitis infection, though none would have thought it necessary to give a specific warning about that or to provide specific information about that on every occasion when treatment was administered. In my view the exercise of reasonable care at that time demanded no more. The situation might be different if they were specifically asked about it. In that event one would expect them to give specific information as to the known risk. The ordinary expectation, however, would be that over a long experience of his condition a haemophiliac patient and the parents of a haemophiliac child patient would have become aware of the risk and repeated warnings would not be necessary. It was obviously the expectation that a patient would be fully instructed at the time when the diagnosis was first made. Clearly in formulating the warnings or information to be given at that initial stage, it would be proper for the doctor to emphasise the smallness of the risk, to point out that the known incidence of post-transfusion hepatitis in Australia was about 2% and that clinical hepatitis as opposed to an inflammation of the liver detectable only by laboratory tests was virtually unknown. It would have been appropriate to emphasise the known safety record of the Australian blood supply, stemming from the use of voluntary donations only and the implementation of rigorous serological screening procedures, and on the other hand to draw to the attention of the patient the benefits of treatment with AHF and the highly unpleasant and even dangerous consequences likely to ensue if treatment were not carried out. It was not, in my view, reasonably necessary nor in accordance with usual and sound medical practice to warn of the remote risk of infection by an unknown virus.

When the plaintiff's condition of haemophilia A was originally diagnosed, in August 1980, his parents had a consultation with Dr Lammi and Dr Bryant. It is quite clear that neither of the parents retained any clear recollection of what took place. Indeed, although Mrs H recalled that what took place was a discussion with both Dr Lammi and Dr Bryant, her husband did not recall Dr Bryant having been present. I am satisfied that he was, not only because of the recollection of three of the parties to that effect, but also because on 23 August 1980 Dr Bryant wrote to the family's general practitioner a letter including the sentence, "Dr Lammi and I have spoken to parents about what they should do". Notwithstanding their poor recollection of the detail of the conversation, both parents were adamant that they were never told that the administration of clotting factor to their sons carried with it any risk — whether of an adverse reaction to the foreign substance or of infection with hepatitis, liver disease or anything else. Dr Lammi and Dr Bryant gave evidence that the task of explaining to parents of newly-diagnosed haemophiliacs the nature of the disease and treatments available for it was a task which they had regularly to perform at the relevant time and that although there was not

a set check-list according to which they proceeded on every occasion, there was a standard set of topics which were discussed. Those included, according to Dr Lammi, the diagnosis, the prognosis, the treatments available, some detail about the type of treatment, the indications for home treatment and for referral to a hospital, the nature of the clotting factor which would be used including the fact that it was a blood product, and the danger of infection associated with the use of blood products. As to the last matter, Dr Lammi's evidence in chief was:

> "As regards specifically, they would have been told that the dangers of these products were the same as any other blood product. I am not sure whether you are asking, but I would not have given them detailed — I did not in those days or even now give detailed descriptions of all the potential problems blood may give.
>
> Q. Why did you not do that in 1980? A. I thought the risk of getting infections was low in the products that were used."

He said it was not his practice to discuss specifically the topic of hepatitis because the risk thereof was low and did not in his opinion warrant a specific warning. What he said was in general terms that you may get infections from blood products. Dr Bryant gave evidence to much the same effect but could not confirm that warnings about the risk of infection were given. He said:

> "I can't answer that specifically. I would believe that risk of infection would have been mentioned as a consequence that the child would be receiving a blood product. Haematologists, as a rule, are people who are concerned about giving blood. We seem to give blood and blood products with more consideration than perhaps other members of the medical fraternity since we are dealing with it all the time."

In cross-examination he said that the usual practice they follow was that both he and Dr Lammi would be present so that if any topic were overlooked by one the other would see that it was adequately explained to the parents of the patient.

It seems to me that the probability is that the parents of the plaintiff were informed, at least in the general way that Dr Lammi described, of the slight risk of acquiring an infection from the use of blood products. I have already indicated my impression of Dr Lammi as a very careful and concerned physician and it seems to me he would certainly have taken care to give what he regarded as adequate information. That, after all, was the purpose of the meeting. It is of course clear that both he and Dr Bryant regarded the risk of infection from AHF in 1980 when this conversation took place as being minimal and as being of almost no significance as compared with the importance that the child should have appropriate AHF treatment when necessary. The probability is that, for that reason, the risk although referred to was played down, but I would not accept that no appropriate warning or information about it was given. I reach that conclusion the more easily taking account of the evidence of the plaintiff's mother that "we wanted to find out everything we could about it" and of his father, that we were "very interested in the health risks that might follow from treatment", and I would have no doubt in those circumstances that both of them probably, but the mother certainly, would have asked what risks there were, if she had not been told of any and if, as I find to have been the fact, she was told that there was a risk, albeit a very slight one, that she would have enquired further and ascertained precisely what risks, in Dr Lammi's view, existed at that time.

The plaintiff has failed to satisfy me on the balance of probabilities that his parents were not given information about those minimal infection risks which in Dr Lammi's view were associated with the use of AHF.

The real question is whether such information and warning as were given were adequate to discharge the hospital's duty of care. In answering that, it must be acknowledged that the risk was slight. As to known infections other than hepatitis, the risk was virtually non-existent by reason of the serological tests available. In relation to hepatitis, it was a very small risk and reasonably believed by Dr Lammi to be very small, and it was a risk of a serologically detectable hepatitis which in the vast majority of cases was asymptomatic and not detectable clinically. The risk of the presence of other infections was theoretical only and completely unquantifiable. The form which any warning about risk should take was reasonably to be determined by the doctor with regard to the importance to the patient that he should not, by reason of undue concern about remote risks, be dissuaded from having treatment at all.

It appears to me that the giving of such warnings as Dr Lammi in fact gave accorded with practice accepted as proper by a responsible body of medical practitioners skilled in the art of treating haemophilia. There is but slight opinion to the contrary.

I am satisfied that in the circumstances a warning such as was in fact given, limited to a statement that there was a very small risk of infection in the use of any blood product, which risk was far outweighed by the importance of having treatment in the event of a bleed was a completely adequate discharge of the physician's responsibility at that time.

The probability is that there was no further discussion of such risks on any occasion thereafter up to and including the time of the plaintiff's treatment with AHF in March 1982. There was no reason why there should have been. The risk had not during that period altered or increased; no additional circumstances were or ought to have been known to the hospital doctors which would have made it necessary to repeat or revise or affirm the warning earlier given. They were, as it seems to me, entitled to proceed on the assumption that the plaintiff's parents were aware of the risk and accepted it in any situation where they were advised by the doctors that AHF treatment was requisite. It remained, of course, the obligation of the doctors to consider, on every occasion when AHF treatment was contemplated, whether the indications for such treatment were such as to outweigh that risk which was known to exist, including the theoretical risk of unknown and unidentified infections. There is no evidence that consideration of that was not given, but in any event the reality is that the risk was so slight that whatever consideration was given to it, the existence of the risk would not have dissuaded Dr Lammi, Dr Bryant or their subordinates from proceeding with AHF therapy once a diagnosis of joint bleed was made. There is no evidence to suggest that the failure to give renewed warnings on the occasions of subsequent treatments up to and including March 1982 was not in accordance with ordinary responsible medical practice. Nor, in my view, did the physicians' duty to take reasonable care for the plaintiff require that such repeated warnings be given.

By September 1983, a new and different risk attached to the use of AHF. It was a risk of which the plaintiff and his parents had not been previously warned. It was a risk which, although it could not be quantified, was clearly recognisable by physicians who had kept in touch with developments, both in the United States and in this country and although it could reasonably have been said that the chance that the risk would eventuate was small, it had also to be recognised that if the risk did eventuate, the consequences to the plaintiff would be disastrous. Notwithstanding that, it appears to have been the view of the American clinicians who gave evidence, of Professor Penington and Dr Rickard, and of the two treating doctors, Dr Lammi and Dr Bryant, that it was not appropriate in September 1983 that the plaintiff's parents should have that risk brought to their attention. The view which they held and expressed was, I have no doubt, a view arrived at in good faith upon very careful consideration and a view which reflected their perceptions that the risk was minuscule, that the provision of appropriate treatment with AHF was extremely important for the plaintiff's well-being, not only at the time of the treatment but throughout his life, and that it was unthinkable that the plaintiff's parents, being fully acquainted with the circumstances, would have refused treatment for their son. Although Professor Dwyer and perhaps Dr Gatenby would have thought it appropriate to discuss the risk with the parents, this is clearly a situation where the application of the *Bolam* test without the qualification expressed by Lord *Bridge*, Lord *Templeman* and Lord *Keith*, would compel a conclusion that Dr Lammi was not at fault in failing to warn of the emergence of the new risk. However, the devastating consequence should the risk mature into reality brings this case within Lord *Bridge's* qualification, as a case where "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". Lord *Bridge* instanced what he had in mind by referring to "an operation involving a substantial risk of grave adverse consequences, as, for example, the 10% risk of a stroke from the operation which was the subject of the Canadian case of *Riebl v. Hughes*"; but I would not understand him as intending to define exclusively the circumstances where disclosure of a particular risk was obviously necessary. The greater the chance that the risk will eventuate, the more obviously will disclosure be necessary, even though the consequences of the happening of the risk may not be enormous. Conversely, where the possible consequence is disastrous, disclosure may be "obviously necessary" even though the risk be quantified as tiny. In my opinion this was such a case.

If one approaches the case on the basis of Lord *Scarman's* test, then in my opinion the risk here was a material risk, for the potential consequence was so serious that a reasonable person in the patient's position would be likely to attach significance to the risk. That does not, as I understand it, mean that a risk is material

only where the reasonable person in the patient's position, being made aware of the risk, would decline treatment. The question is whether it is a matter which the reasonable patient would wish to take into account in deciding whether or not to accept treatment. I am of opinion that reasonable care on the part of the hospital required that before treatment with AHF was administered in September 1983, the patient, and in the case of a child the parents of the patient, should have been warned in appropriate terms of the AIDS risk. It is clear in the present case that no such warning was given.

It is then necessary to consider what would have been the response of the plaintiff's parents had that warning been given which the exercise of reasonable care required. Each of them was asked a question apparently designed to elicit evidence relevant to that question. Mrs H:

> "Q. Had you been warned that there was a treatment available which presented less risk, what would have your response been? A. I would have requested the treatment that held less risk."

Her husband gave this evidence:

> "Q. What would you have done if you were told that there was more than one treatment and one or other of the spectrum available was less liable to risk than another, what selection would you have made? A. Well, the less risky option."

The question at issue is a question of causation. Assuming negligence in failing to give appropriate warnings or information, the question is did that negligence cause the plaintiff's injury? It may only be held to have done so if it is established by the plaintiff that if the appropriate warning and information had been given, he would have refused to have the treatment alleged to have caused his damage. The test of whether, properly informed and warned, a plaintiff patient would or would not have accepted the proposed treatment is a subjective test, not an objective test — that is to say, the question is not what would a reasonable person in the position of the plaintiff had done, but what would the particular plaintiff have done — *Ellis* v. *Wallsend District Hospital* (1989) Aust. Torts Reports ¶80-289 — has authoritatively settled that question. In the present case, of course, the decision whether or not to proceed with AHF treatment would have been made not by the plaintiff personally, but by his parents, and it is clear, having regard to the evidence which each of them gave and particularly that of the father, that the decision would effectively have been that of Mrs H who primarily took the responsibility for matter concerning the health of the children. Because the test is subjective, evidence by the person involved as to what his or her response would have been in the assumed circumstances is relevant and admissible and may be of considerable weight but nevertheless it must be viewed with caution because it is clearly likely to be much influenced by hindsight. The court is not bound to accept the assertion of the patient that, fully informed and warned, he or she would have refused treatment. The evidence of the patient to that effect must be evaluated in the light of all of the circumstances, and upon some assumption founded in the evidence as to what would have been the terms of an appropriate warning at the time.

Appropriate discussion of the question by the treating doctor would properly have emphasised the likely consequences of failure to treat a joint bleed — consequences in the nature of present pain and future disability. It would have been appropriate for the doctor to remind Mr and Mrs H that there had always been and remained a risk of infection with hepatitis in consequence of AHF therapy but that in fact the plaintiff and his brother had had such therapy on many occasions since original diagnosis with no adverse effect but with excellent therapeutic results. It would have been appropriate then to tell them that in a very small number of cases in the United States, AIDS had developed in patients who had been treated with AHF; that as at May 1983 the number of such cases was only 12 out of a haemophiliac population of between 15,000 and 20,000; that it was not proved that the disease had been transmitted by AHF but that that was regarded as a possible source of infection in that small number of cases; that despite this appearance of the disease in the United States, the National Hemophilia Foundation was strongly recommending to all haemophiliacs in the United States that they should continue to accept treatment with AHF, whether by way of concentrate or cryoprecipitate (Exhibit 204, 11 May 1983; Exhibit 205, 1 September 1983); that the Australian blood supply had always been regarded as much safer than the American blood supply, particularly because of the exclusive use here of voluntary donations; and that there had not been in Australia a single case of AIDS related to blood transfusion or treatment with AHF. It would have been proper for the treating doctor to express strongly his own opinion that the risk was negligible and should not influence Mr or Mrs H to refuse AHF treatment for their son.

The question of the patient's (parents') probable response to such a warning must be evaluated in the light of the experience which up to that time, the patient had had and of treatment and the confidence which the patient had at the relevant time in the medical practitioner. The circumstances relevant to the patient's response would have included, among other things the confidence which quite clearly both parents had in Dr Lammi's expertise and concern for their sons, and the fact that on three previous occasions the plaintiff, and on a number of previous occasions his brother AH had, on the advice of Dr Lammi or his staff, received AHF therapy with the anticipated good result and no evidence of any harmful side-effects.

The answers given by the parents which I have quoted above carry little weight in my view especially because the questions were not so framed as to include any assumption of a particular form of warning which would have been appropriate.

The plaintiff has failed to satisfy me that if an appropriate warning of the risk had been given in September 1983 the consequence would have been that AHF was not administered to him on that occasion. It follows that even if the treatments in September 1983 were the source of the plaintiff's infection, it has not been established that that was a consequence of any negligence on the part of the hospital or its staff.

18. The time at which the plaintiff became infected

In view of the conclusions reached on other issues, it is not necessary for me to determine on which occasion and from which product the plaintiff acquired the HIV infection. Nevertheless, the issue is one which received a great deal of attention at the trial and it is appropriate that I should deal with it.

There were four separate strands of evidence bearing on the issue.

The plaintiff relied on evidence of a statistical sort, from Professor Dwyer, Dr Kendall and Dr Ziegler. Their proposition was that as there were in Australia more persons infected with HIV in September 1983 than there were in March 1982 (a fact Which is common ground), it was more probable that the plaintiff became infected at the later than at the earlier date. This form of reasoning does not appear to me to be valid. The fact that there were more individuals in the community carrying the infection as at September 1983 than there were as at March 1982 may be accepted. It is no doubt permissible to extrapolate from that that there were more infected donors of blood in September 1983 than in March 1982, because it may reasonably be assumed that the distribution of infected persons in the general community is approximately the same as among blood donors. Those pieces of information would enable a choice to be made between two lots of AHF, one made in March 1982 and one made in September 1983 on the basis that the former carried a lower risk of HIV infection. It is information which would enable one to say of a population of persons found after September 1983 to be infected with AIDS, all of whom had received treatment with AHF both in March 1982 and in September 1983, there being no other indication of the source of infection, that a larger proportion of them were probably infected by treatment in September 1983 than were infected by treatment in March 1982. It does not, however, seem to me possible to draw from this information any conclusion, even on a balance of probabilities, as to when any particular person was infected.

The second strand of evidence bearing on the date of the plaintiff's infection was evidence that a significant proportion of AIDS sufferers whose date of infection can be ascertained (patients who had received a single blood transfusion, health workers suffering needle injuries, etc.) develop within a period of a few weeks what is referred to as a sero-conversion illness which marks the stage in the development of the disease when the virus lodged in the body is translated into actual infection. According to Professor Holland, whose evidence was not challenged or contradicted on this, studies show the development of this type of illness in about 24% of cases. It was common ground among the experts that where this sero-converting illness occurs, the symptoms are similar to a viral mononucleosis type illness, marked by fever and vomiting without signs of respiratory illness but sometimes accompanied by sore throat, aches and pains and sometimes a skin rash, diarrhoea and tender or enlarged lymph glands. According to Professor Penington, whose evidence was on this point not challenged or contradicted, the onset of such symptoms may be as soon as six days after infection or as late as several weeks after. Professor Holland referred to studies showing the development of this type of illness "two to three or four weeks after the infection"; Dr Dietrich used the expression "some few weeks after".

Exhibit NN is a copy of the clinical notes relating to the plaintiff from the practice of a suburban general practitioner who was the H family doctor in 1982. These reveal that on 6 April 1982 the plaintiff attended at that practice and the notes of that attendance are as follows:

> "6/4/82 Fever, vomiting. Chest ✓ ears ✓."

He returned on 21 April 1982 for treatment for a strained ankle, and there is on that occasion no reference to any of the symptoms noted at the earlier visit.

Dr Kendall did not deal with this aspect of the evidence. The other physicians who gave evidence were unanimous that the findings recorded in Exhibit NN were consistent with the presence at that date of the sero-converting illness although they all agreed that what was recorded was by no means diagnostic of such an illness; Professor Dwyer and Dr Ziegler went further and suggested that had it been a sero-converting illness one would have expected more symptoms to be noted. Clearly in the absence of serological tests done at the time, one could not conclude from this evidence alone that the plaintiff had received his infection at the hospital in March 1982, with the administrations of AHF 15 to 17 days before his consultation with his general practitioner. It is to be noted also that Professor Dwyer thought it unusual that there should have been a sero-converting illness only two and a half weeks after infection. He was not asked to enlarge on that comment nor did he at any time explain it, and notwithstanding that remark I am satisfied that the onset of an illness with the symptoms noted in Exhibit NN at such a date after treatment with AHF is consistent with that illness being a sero-converting illness.

The third strand of evidence bearing on this issue is evidence relating to the incubation period of AIDS — that is to say, the period of time which elapses between the date of infection and the manifestation of the symptoms of AIDS itself. The plaintiff developed symptoms, which with hindsight can be seen to have been symptoms of AIDS, at about the beginning of 1985 when, as noted earlier, he began to appear unwell. By April 1985 he had begun to lose weight. It is possible that the depressed mood noted by his mother late in the previous year was connected with the onset of his illness. If January 1985 be assumed as the date of onset of the illness (certainly it was about that time) that is about 16 months after the September 1983 treatments and 34 months after the March 1982 treatments.

Professor Dwyer thought that the evidence of the plaintiff's clinical course was not helpful, because, in his view, the literature was not clear as to the length of time which may elapse between infection and development of AIDS in cases where the source of infection was an administration of Factor VIII.

Dr Kendall was prepared to say in Exbibit ZZ that:

> "the lead time between acquisition of the infection and clinical presentation is variable, but on probability it is more likely that the later administration of Factor VIII was responsible rather than the earlier one".

In his oral evidence in chief Dr Kendall did not explain the basis of that view and it became apparent in the course of cross-examination of him upon it that he had no knowledge at all of any of the relevant research work directed to that sort of question. I reject his evidence.

Professor Holland gave convincing and cogent evidence on the same issue, based particularly upon his familiarity with and participation in research projects, and his attendance at a conference in Montreal in June 1989 where papers directed to that issue were delivered. I accept his evidence that it is almost unheard of for anybody to develop symptoms of AIDS within two years of a known date of infection. That does not mean that the plaintiff could not have developed AIDS in early 1985 as a result of an infection received in September 1983, but it makes it highly unlikely. Indeed, according to Professor Holland the incubation period of just under three years is also relatively short. Professor Holland's evidence appears to be supported by the research papers to which he referred including Exhibit 212 and Exhibit 214, and I accept his evidence.

This brings me to the fourth strand of evidence which may be referred to as the "Look Back" evidence. Following the development of an awareness in New South Wales that blood products may have carried the AIDS virus prior to the time when a test became available for it, the Red Cross instituted a program called "Look Back" to identify, so far as possible, infected blood donors and recipients of infected blood. The details do not matter for present purposes. In the course of the ordinary conduct of that program, prior to the commencement of this litigation, the third defendant had identified an infected donor who became identified

in its records as D-20. D-20 was a regular donor of blood and/or plasma over a number of years. Exhibit 327 which is the material relating to the Look Back program records him as having made 32 donations of blood since 1980. So far as appears by that material, 13 of those donations have been investigated. On 28 March 1986, D-20 reported by telephone to Miss Learmont at the blood bank that he had been tested HIV positive and had had the disease since 1984. A medical certificate is in evidence which confirms that the gentleman known as D-20 is indeed an AIDS sufferer.

Investigation of four attendances at the blood bank by D-20 from December 1980 to January 1982 are recorded. On 10 November 1981 and 26 January 1982 he was not bled but the reason for that course being followed does not appear. One reason why intending donors are rejected is of course that they are manifesting symptoms of some viral illness, but there is no evidence that such was the case on those occasions and it would be sheer speculation to proceed on any such view. Of the blood taken on two previous occasions, that taken on 10 December 1980 was ultimately not used. That taken on 27 March 1981 was divided into two portions. Platelets sent to Prince Alfred Hospital on 27 March 1981 have not been traced. Red cells sent to a private hospital were used on 23 April 1981. The recipient has since been tested for AIDS and tested negative.

On 17 March 1982 the donor gave blood again. Part of the donation was made into cryoprecipitate bearing No. 30870, and it is apparent from Exhibit PP and Exhibit OO that that cryoprecipitate made from the plasma of D-20 was part of the cryoprecipitate administered to the plaintiff at the hospital on 20 March 1982. The balance of the donation has not been able to be traced.

Blood taken from D-20 on 16 June 1982 was separated into a “quad-pack”, that is, four separate blood components or products. Part, comprising red cells, was on 1 July 1982 transfused to a woman who was referred to during the trial as case 106. The balance of the donation has been unable to be traced. Case 106 was a married woman, 32 years of age, living in an apparently happy marital relationship who received blood, including that which came from D-20 for a post-partum haemorrhage on 1 July 1982. She in fact received blood from three other donors but each of them has been tested negative to HIV. So too has been her husband and her children. She had not had any previous blood transfusion. It is highly probable that she acquired the HIV infection from the blood of D-20.

The next two recipients of blood from D-20 who have been able to be traced had both died by the time of the investigation. They received blood donated by him respectively on 26 October 1982 and 10 February 1983. The recipient of blood donated by him on 2 August 1983 has not been traced. Recipients of blood donated by him on respectively, 11 October 1983 (case 40), 11 October 1983 (case 92) and 19 April 1984 (case 42), and one of two recipients on 26 June 1984 have all been tested HIV positive. Another recipient on 26 June 1984 has not been tested because of her great age. Other recipients of blood donated on 26 October 1982, 10 February 1983, 2 August 1983, 7 February 1984 and 19 April 1984 have not been traced.

It is submitted on behalf of the defendants, and some of the expert witnesses have expressed the opinion, that this evidence makes it highly likely, indeed (it is submitted) almost certain, that the plaintiff acquired the HIV infection by reason of the administration to him of cryoprecipitate made from plasma donated by D-20, an AIDS-infected person. I do not accept that submission. It may, I think reasonably be assumed that D-20 was HIV positive as at 17 June 1982, when he donated blood which was subsequently administered to case 106. It by no means follows that he was infected three months earlier. There is evidence which I accept that a person may transmit the virus as early as two weeks after himself acquiring it, so that the only inference that can be drawn from the fact that case 106 became infected is that D-20 was infected by, at latest, about the end of May 1982. He may or may not have been infected in March of that year — there is simply no way of knowing.

Professor Penington had calculated the statistical probability that D-20 was infected in March as being in the order of 98%. The line of reasoning which he expressed is in my view not capable of establishing anything more than that of all those people who were infected as at June 1982, the probability is that 98% of them had been infected by March 1982. It does not in my view afford any basis for saying anything as to when a particular person became infected. In respect of D-20, the absence of evidence of any person having become infected apparently as the result of receiving his blood before March 1982 is from the defendants’ point of view at best equivocal, at worst indicative that he was not infected earlier. It is appropriate to note in passing that Professor Holland expressed an opinion based in part on information provided to him that

a patient transfused in January 1982 with blood from D-20 had recently developed a brain tumour which Professor Holland considered to be possibly an AIDS-related condition. However, there is no evidence of any such event; on the contrary, the evidence is that in January 1982 D-20 was not bled.

The fact that a recipient of blood donated by D-20 in April 1981 did not become infected is very strong evidence that D-20 was not infected at that time although the statistical probability, calculated as Professor Penington would have calculated it, as I understand his reasoning, would exceed 80%.

In my view, the Look Back evidence relating to donor D-20 does not assist in determining the date at which the plaintiff became infected. Nevertheless, it does appear to me on the balance of probabilities that the plaintiff received the infection in March 1982. I base that finding on the evidence as to the likely incubation period of the disease, coupled with the evidence that the plaintiff in April 1982 exhibited symptoms consistent with a sero-converting illness; together with the total absence of any evidence (except of a statistical sort) to suggest that the infection was acquired in September 1983.

Since I have found that none of the defendants involved in the treatment of the plaintiff or the manufacture of the product by which he was treated in March 1982 was negligent, it is not necessary for me to reach a conclusion as to which of the several products administered to the plaintiff in March 1982 was the source of his infection. The parties may perhaps prefer, however, that I make a finding on that issue. For reasons that I have given I do not regard the Look Back/D-20 evidence as of assistance in establishing the date of infection of the plaintiff. Nor do I regard that evidence as persuasive on the question whether the plaintiff received his infection as a result of an administration of cryoprecipitate or of concentrate.

There is, however, other evidence which in my view should lead to a finding that it is more probable than not that he was infected from cryoprecipitate rather than from concentrate. Exhibit PP shows that the plaintiff was not the only child to be treated with concentrate from batches 201 and 287-1. During the period 13 March to 23 March, three children were treated with Factor VIII concentrate from batch 287-1. One was the plaintiff, a second was the plaintiff's brother AH. The plaintiff received two doses from that batch, AH received nine. A third child, of whose HIV status no evidence has been adduced, received 30. During the same period, four children were treated with concentrate from batch 201. The plaintiff received five doses. His brother AH received 12. Another child received 30 and a fourth child five. There is no evidence of the HIV status of either of those two children. There is evidence that the plaintiff's brother AH is HIV negative — that is to say, treatment in March 1982 with concentrate from the same batch as the plaintiff received, and treatment indeed with a larger quantity from each batch than the plaintiff received did not cause AH to become infected. The method of manufacture of Factor VIII concentrate, involving the pooling of plasma from a large number of donors and the treatment of the whole in the same fashion, would tend to produce an even distribution of any virus present. One would anticipate therefore, that if the virus as thus spread and diluted was present in one bottle of concentrate in sufficient numbers to be infective it would be present in all; or that if it was diluted so that two doses were necessary to transmit the virus to a recipient, any person who received two or more doses would be likely to be infected. The plaintiff received two doses of batch 287-1, his brother received nine. The plaintiff received five doses of batch 201, his brother received 12. I am satisfied on the balance of probabilities that neither batch of concentrate from which the plaintiff was treated in March 1982 was infected with HIV and the probability therefore is that he was infected by a treatment with cryoprecipitate.

19. The question of remoteness of damage

Had I reached a conclusion that the plaintiff's infection was in fact the result of negligence on the part of one or more of the defendants, I would need to give careful consideration to the question whether the damage suffered by the plaintiff was too remote to be the subject of a claim for damages at common law. It was argued on behalf of the defendants that damage of the kind suffered by the plaintiff was not foreseeable as a possible outcome of the negligence alleged against the defendants. In the circumstances I do not propose to give any exhaustive attention to this question but simply to express, in deference to the arguments that were put, in a very summary way my opinion about it.

So far as September 1983 is concerned, it appears to me to follow from the findings that I have already made that it was clearly foreseeable that the AIDS virus might be transmitted in AHF made from plasma and that the acquisition of such an infection was the very kind of damage foreseeable as a consequence of any failure by the defendants to exercise reasonable care. It is true that the nature of the agent which caused

AIDS was not known, nor was it by any means certain that it was transmissible in blood. In my view neither circumstance prevents the conclusion which I have expressed.

In respect of treatments given and precautions which ought to have been taken in March 1982, the argument on behalf of the defendants was that infection with the AIDS virus was an infection of an entirely different kind from anything which could have been foreseen as at that date. Some attention was paid during the course of the evidence to differences between a virus and the group known as retro-viruses of which HIV is one. The particular features which distinguish them in the eyes of virologists were enumerated. Some attention was paid also to the difference in the nature of the HIV antibody and the hepatitis B antibody. The presence in blood of the latter is not associated with infectivity, whereas the presence of the former may be. Obviously much depends on the level of generality at which one defines the kind of damage foreseeable. The plaintiff's case is that it was in March 1982 foreseeable that a blood borne virus might be transmitted to the recipient of AHF who might thereby become infected. The fact is, as I have found, that the plaintiff did become infected by the transmission to him of a blood borne virus. In my view it is not to the point that what he received was a retro-virus as distinct from a virus otherwise defined, nor is it to the point that the virus in blood behaves differently from others. Were it necessary for me so to find, I would find that the damage incurred by the plaintiff was damage of the same kind and was a foreseeable consequence of any negligence on the part of the defendants in March 1982.

20. Conclusion

The plaintiff has failed to establish that he became infected with HIV as the result of any negligence on the part of any of the defendants. I find a verdict for each defendant and direct the entry of judgment accordingly.