

THE HIGH COURT

2001 No. 639 P

BETWEEN

BRENDAN WARD

PLAINTIFF

AND

SOUTH WESTERN HEALTH BOARD, MIDLANDS HEALTH BOARD, PAUL O'REGAN AND HUMPREY J. O'CONNOR

DEFENDANTS

Judgment of the Honourable Mr Justice John Quirke delivered the 24th day of June, 2008

1. The plaintiff, Brendan Ward, was born on 11th April, 1968 and is now forty years old. He is married and lives with his wife and three year old daughter in Cahir, County Tipperary.
2. He enjoyed good health before 1998 and was an active footballer during his youth and early twenties. He has been in constant full-time employment since the age of eighteen years working manually; (a) in a "Cash and Carry" premises in Clonmel, (b) as a lorry driver's assistant and, (c) on a production line with a firm called Seagate.
3. On 23rd February, 1998, he commenced employment as a manual labourer with Messrs. Cahir Meatpackers Limited. He was then engaged to be married and had planned his wedding for September of that year.
4. On 25th February, 1998, (the third day of his employment with Messrs. Cahir Meatpackers Limited), he suffered a sudden bout of illness while working.
5. He continued at his job but consulted his General Practitioner on the same evening. He was referred to Clonmel Hospital where he gave a history of right lumbar pain and nausea. He was passing dark coloured urine.
6. A number of tests were undertaken within the Hospital including blood and urine tests and liver function tests.
7. At 11.35p.m he was admitted to Cashel Hospital under the care of a Consultant Physician, Mr. Murchan, who diagnosed renal stones.
8. Additional and further tests (including an abdominal ultrasound scan and an intravenous pyelogram) were undertaken in Cashel Hospital but no abnormality was disclosed and he was discharged on 1st March feeling better with a letter of referral to Dr. Kingston who is a Consultant Surgeon in St. Joseph's Hospital, Clonmel, County Tipperary, (hereafter "Clonmel Hospital").
9. The plaintiff attended Clonmel Hospital on 20th March, 1998. Further tests were undertaken on the instructions of Dr. Kingston who formed the opinion that the plaintiff had contracted hepatitis and should remain out of work for some weeks.
10. On 14th April, 1998, the plaintiff was again examined in Clonmel Hospital by Dr. O'Regan, who is a Consultant Physician and is the third named defendant in these proceedings. On the advice of Dr. O'Regan he returned to work on 16th April, 1998.
11. Immediately on his return to work he sustained another serious bout of illness and returned to Clonmel Hospital where he was admitted under the care of Dr. O'Regan complaining of passing dark urine and feeling tired with headaches and nausea.
12. A further series of tests were undertaken on the instructions of Dr. O'Regan and on 20th April, 1998, Dr. O'Regan reviewed the plaintiff and referred him for investigation to Dr. Humphrey O'Connor (who is the fifth named defendant in these proceedings and is a Consultant Physician and Gastroenterologist then employed by the second named defendant in Tullamore Hospital).
13. Dr. O'Regan suspected that the plaintiff was suffering from a condition known as ascending cholangitis which is a bacterial infection of the bile ducts caused by an obstruction within those ducts. He wanted Dr. O'Connor to carry out a procedure known as an endoscopic retrograde cholangiopancreatography (hereafter called an "ERCP") upon the plaintiff which is a diagnostic procedure performed using a long flexible viewing instrument within the stomach and intestine.
14. An ERCP is a procedure which is not undertaken lightly because it carries with it a number of known complications including haemorrhage, perforation, hypoxia and, in particular, acute pancreatitis which occurs in 3 to 5% of patients who undergo an ERCP.
15. The plaintiff was discharged from Clonmel Hospital on 20th April, 1998 and instructed to return on 5th May. He did so. When he arrived in Clonmel Hospital, he felt well and had no cause for concern. Liver function and other tests which were performed in Clonmel Hospital on that day disclosed no abnormality.
16. He was told that he was to be taken by ambulance to Tullamore Hospital to undergo an ERCP. Upon arrival in Tullamore Hospital, he was interviewed by a Senior House Officer within Tullamore Hospital (Dr. Jayanthi) who advised him that she was a member of "Dr. O'Connor's team".
17. The plaintiff stated in evidence that Dr. Jayanthi told him that he would be sedated for procedure which was about to commence and that a tube would be put down his throat which would inject dye into his abdomen for investigative purposes. He said she told him nothing else, asked him to sign a form and that he did so without reading it.
18. He said he was taken to a treatment room and remained there for "an hour or two" before the procedure commenced. He said he never met Dr. O'Connor and has no recollection of the procedure.
19. He stated that he was placed in an ambulance to be returned to Clonmel Hospital and after approximately thirty minutes he began to suffer unbearable abdominal pain which caused him to scream in agony and to vomit.
20. He said that those acute symptoms continued for the remainder of the journey which took more than an hour and the ambulance attendants could do nothing for him.
21. When he arrived in Clonmel Hospital he was still screaming in agony. It was some hours before the unbearable pain and other distressing symptoms could be reduced to some extent.

22. Over the succeeding weeks and months he was extremely ill and in constant pain. His condition worsened before it was finally brought under control.

23. He had contracted what Dr. O'Regan in a report dated 19th October, 1999, described as ". . . *the recognised complication of post-ERCP pancreatitis which was complicated by the development of a pancreatic abscess and pseudocyst. Both of these complications are recognised and unavoidable occasional results of ERCP in every centre worldwide.* . . .".

24. The plaintiff claims that he was caused to develop this pancreatitis by reason of negligence and breach of duty on the part of the first and second named defendants who, as (then) statutory bodies responsible for the provision of hospital care and treatment at Cashel Hospital, Clonmel Hospital and Tullamore Hospital, were vicariously liable for the various investigations and treatments undertaken upon the plaintiff in respect of his illness and by the staff and medical practitioners within those hospitals (including Dr. O'Regan and Dr. O'Connor).

25. He also claims damages for injury loss and damage resulting directly, he claims, from negligence and breach of duty by Dr. O'Regan and Dr. O'Connor.

26. It is claimed that, by subjecting the plaintiff to an ERCP, the defendants acted negligently and in breach of their various duties to the plaintiff because; (a) ERCP is an investigative procedure which has well known and grave risks of complication, (including the risk of acute pancreatitis), and should not be prescribed unless there is a clearly demonstrated need for such a serious diagnostic procedure and, (b) none of the many tests performed upon the plaintiff disclosed evidence which could have warranted a diagnosis of either acute ascending cholangitis or primary sclerosing cholangitis which were the only conditions which could have justified the performance of an ERCP upon the plaintiff on 5th May, 1998.

27. Additionally, it is claimed on behalf of the plaintiff that, at no time prior to the performance of an ERCP upon him, was he informed of the risks associated with that procedure including the risk that he might develop and suffer from acute pancreatitis as a consequence of the procedure. It is claimed that, had the risks been explained to him and their full implications understood by him, he would not have agreed to undergo the procedure.

28. The plaintiff claims that as a result of the defendants' negligence and breach of duty he suffered extreme pain, distress, inconvenience and serious illness arising directly from the post-ERCP pancreatitis which resulted from an unnecessary and inadvisable procedure and that in addition he has developed ataxia and dysarthria, both neurological conditions which are permanent and have developed as a direct result of the post-ERCP pancreatitis from which he has suffered.

29. The defendants initially denied any negligence or breach of duty in the investigation and treatment of the plaintiff's illness or in the provision of adequate information as to the risks associated with the procedure. On the sixth day of the trial of these proceedings they admitted negligence and breach of duty and acknowledged that the pancreatitis contracted by the plaintiff had been caused directly by reason of the defendants' negligence and breach of duty.

30. However, the defendants contend that the ataxia and dysarthria from which the plaintiff suffers have not been caused or contributed to by reason of the defendants' negligence. They contend that they result from a mitochondrial disorder, (a genetic disturbance), within the plaintiff which has been present since his birth.

Special Damages

31. It has been agreed between the parties that the plaintiff has incurred losses in the amount of €22,368.75 as a result of the defendants' admitted negligence. He is therefore entitled to recover that sum by way of damages from the defendants jointly and severally.

32. Although he has not been able to return to the type of manual work of which he was capable prior to February, 1998, he, nonetheless, sought and obtained permanent and remunerative employment in September, 1999 and he has now been promoted to the rank of Quality Control Supervisor with a successful corporation. In consequence, he makes no further claim for loss of earnings.

Issues

33. Three issues remain to be determined by this court, they are as follows:

1. How much is the plaintiff entitled to recover from the defendants by way of general damages for the pain, suffering, distress, inconvenience, upset and disruption of his life and lifestyle resulting from the post-ERCP pancreatitis which he developed as a result of the defendants' admitted negligence and breach of duty?
2. Were the neurological symptoms from which the plaintiff now suffers, (and in particular the ataxia and dysarthria), caused by and consequential upon the pancreatitis which the plaintiff developed as a result of the defendants' admitted negligence and breach of duty; and if so
3. How much by way of damages is the plaintiff entitled to recover from the defendants to compensate him for the pain, suffering, inconvenience, distress and disruption of life and lifestyle which the plaintiff has suffered and will suffer in the future as a result of those neurological deficits.

1. General Damages for Pancreatitis

34. The plaintiff suffered pain which he described as "*unbearable*" for more than an hour during the ambulance journey between Tullamore Hospital and Clonmel Hospital. He was vomiting during that time and his pain was so severe that he found himself "*screaming in agony*" during the journey and for several hours in the hospital thereafter.

35. He was admitted to Clonmel Hospital where he was found to be extremely ill and in constant pain. He was diagnosed with pancreatitis and treated with intravenous fluids and painkillers.

36. He remained very ill in hospital until 14th May when he was discharged. He suffered continuous abdominal pain at home, was regularly vomiting and could not eat. His condition deteriorated further and his General Practitioner who had been treating him with analgesics found it necessary to refer him back to Clonmel Hospital where he was admitted in distress with an elevated pulse rate and raised blood pressure.

37. He had a number of distressing symptoms which required intravenous fluids and a variety of different types of medication to control the pain, stomach acid- production, nausea and other symptoms.

38. Dr. O'Regan diagnosed a pancreatic pseudocyst and on 25th May, 1998. He referred him to Mr. Murchan who is a Consultant Surgeon.

39. On 26th May, 1998, Mr. Murchan diagnosed a post-instrumentation pancreatic phlegmon/necrotic pancreas and felt that he would require a dynamic CT scan of the abdomen. He recommended naso-ental feeding.

40. The plaintiff then required surgical drainage and debridement of the cystic mass but this was not possible until the pseudo-capsule thickened and became fibrous. It was estimated that this would take a further five or six weeks.

41. It was acknowledged at this time that the plaintiff was gravely ill and might require urgent surgery at any time. He required continuous intravenous fluids and continued feeding by naso-gastric tube. He continued in constant pain.

42. It was discovered that the pseudocyst was enlarging and the plaintiff's condition deteriorated further. An attempt was made to drain the pseudocyst but this was unsuccessful because of the extreme viscous content of the cyst.

43. Because major surgical intervention was necessary, the plaintiff was transferred to the Mercy Hospital, Cork, where he came under the care of Mr. O'Sullivan.

44. The plaintiff was treated in the Mercy Hospital on a continuous basis between 29th May, 1998 and 7th August, 1998. He was extremely ill during most of that time and suffered from acute pain and extremely distressing symptoms on a constant basis. I do not intend to describe, in detail, his very distressing symptoms and the various treatments which he required. It is sufficient to say that he suffered severely to the point where he would shake uncontrollably and undergo what he termed as "rigours" on a number of occasions. He required blood transfusions several times and was subjected to continual attempts to drain the cyst. The symptoms and treatments were most distressing and uncomfortable.

45. He suffered very substantial weight loss and received food and nutrition by naso-gastric tubing and intravenous fluids.

46. When he was returned to solid foods, he was placed on a low fat and high nutritional diet but the return to solid foods caused him severe abdominal pain and he found it difficult to keep food down.

47. After discharge from hospital, the plaintiff's weight was recorded at fifty-seven kilograms and he was prescribed a number of different medications.

48. He was readmitted to hospital on a number of occasions and was an in-patient from 2nd September, 1998, for six nights.

49. He was married two weeks after his discharge on that occasion but during the church ceremony and the subsequent reception he had to remain seated in a chair because of his debilitated condition and circumstances. He had to cancel his honeymoon in Spain and he felt that it took some twelve months before he had regained his weight and much of his former strength.

50. On 6th January, 1999, he was admitted to hospital with further symptoms and on 23rd May, 1999, he was again admitted to hospital for two nights with severe abdominal pain and other symptoms.

51. During May 1999, he required intravenous injection of Pethidine for pain on five separate occasions. In August, 1999 he again required medication for abdominal pain.

52. In September, 2000, he suffered a bout of abdominal pain and nausea and vomiting which required medication.

53. In October, 2001, he again required medication (oral Morphine) for abdominal pain and vomiting.

54. He now believes he has recovered from the symptoms consequent upon his pancreatitis but he stated also, (and I accept), that as his strength returned some months after his discharge from hospital and his wedding, he became increasingly conscious that he often had difficulty with his balance and coordination.

55. He said that when he swayed he lost balance and he noticed that he was unable to carry a cup of tea from one room to another without spilling it.

56. He found that, on occasions, others seem to think that he was intoxicated when he spoke.

57. He found that he was unable to return to playing indoor football which he had enjoyed very much prior to his illness.

58. In September, 1999, he secured employment with Abbott Laboratories on a production line. The nature of that job required him to remain seated and that suited his condition. After two years he was promoted and he subsequently became Quality Control Supervisor with that company. His job now requires and enables him to work at a desk 80% of the time and that accommodates his current capacity and circumstances.

59. It is admitted by the defendants that the plaintiff is entitled to recover damages from the defendants to compensate him for the pancreatitis which he suffered as a result of the defendants' admitted negligence for the life threatening and the disabling consequences which he was required to endure in the immediate aftermath of that illness.

60. It is also acknowledged that the plaintiff is entitled to recover damages to compensate him for the ongoing effects of the pancreatitis (other than the ataxia, dysarthria and other neurological symptoms of which he complains).

61. I am satisfied on the evidence and on the balance of probabilities that; (a) the pain which the plaintiff was required to endure in the immediate aftermath of the ERCP and during the subsequent three months was often excruciating and, in his own words, "unbearable" and that, (b) his condition was life threatening and that he was aware of that fact.

62. I am satisfied also that his symptoms were disabling, extremely distressing and uncomfortable and that his overall experience during that time was quite dreadful.

63. Thereafter his wedding, which was an event of great importance to his wife and to himself and to his family, was marred and his honeymoon had to be cancelled because of his condition.

64. His loss and the other symptoms which he suffered have been described in detail earlier and his recovery was slow notwithstanding immense and courageous efforts made by him to assist that recovery.

65. I am satisfied that he is entitled to recover the sum of €75,000 to compensate him for the injuries (other than neurological injuries and symptoms) which he suffered as a result of the pancreatitis which he developed as a result of the defendants' admitted negligence in May, 1998.

Neurological Symptoms

66. In evidence, the plaintiff stated that after his discharge from the Mercy Hospital in Cork on 7th August, 1998, he became conscious that he had had suffered severe weight loss and was debilitated. He suffered constant stomach pain and nausea and vomiting spells.

67. His General Practitioner treated these symptoms by prescribing Stemetil to control his nausea and vomiting and Pethidine to control his ongoing pain. This medication was not entirely effective and, on the 2nd September, 1998, (two weeks before his wedding) he was admitted to hospital for in-patient treatment where he was detained for six nights.

68. His recovery after his wedding and during the autumn and winter of 1998 was slow and difficult. He continued to have symptoms during the latter part of 1998 and for some time thereafter.

69. He made strenuous efforts to restore his strength at the end of 1998 and in the early stages of 1999. As his strength returned he began to notice a problem with his balance. He found that he needed to use a handrail going up and down stairs and he often swayed and sometimes lost balance.

70. He said that initially he attributed this problem to the aftermath of his pancreatitis and the weight loss and debilitating symptoms which had resulted from that condition. He noted also that by reason of his difficulties with balance and his speech he often appeared to other persons to be intoxicated. He said that he assumed that as his strength returned and as he recovered from the after-effects of pancreatitis these difficulties would disappear.

71. He said that his expectations did not materialise and his difficulties with speech, balance and co-ordination remained permanent and have not improved or recovered in any respect.

72. He also said that his speech, balance and co-ordination difficulties have not worsened since he first became conscious of them in the months following his discharge from hospital.

73. Professor Colm O'Morain, a Consultant Gastroenterologist, examined the plaintiff in October, 2002 and January, 2003, for the purpose of providing to the defendant an opinion as to whether or not the performance of the ERCP upon the plaintiff by the defendants was warranted and appropriate. He took the view that it was warranted and was appropriate.

74. Dealing with the plaintiff's neurological symptoms he noted that *"he has managed to put on weight, initially he was seven stone, Mr. Ward is now ten stone. He is not involved in any physical activity. He did try indoor football but was unable to participate. I don't think this is a consequence of the pseudo-cyst of the pancreas"*.

75. In January, 2005, Dr. Jeremy Stern who is a Consultant Neurologist in Frimley Park Hospital in Surrey, England, examined the plaintiff. He said that the plaintiff told him that *"about six months after discharge, others noted that his walking was a little wobbly. He feels his speech is slurred at times and this is intermittent. His gait is certainly affected when he tries to carry something and will be present on a daily basis although much of the time he feels fine. He says his problem is static and not progressive"*.

76. He found that the plaintiff's speech was *"mildly dysarthric. He is slightly unsteady on his feet with a wide based gait and poor tandem walking. There is very mild finger and nose ataxia but definite disidiadochokensia more on the left than the right"*.

77. Stating that *"I suspect the ERCP is not related to the neurological problems"* he said that his opinion *"would be pending neuro-imaging"* and he recommended investigation with an MRI brain scan, lumbar puncture, metabolic screen among other tests.

78. On 7th July, 2004, the plaintiff was seen by Dr. Patrick Connor, a Consultant Gastroenterologist and General Physician at Finlay Park Hospital in Surrey.

79. Although Dr. Connor was investigating the relationship between the plaintiff's pancreatitis and the ERCP which preceded it, he noted in his report that *"what struck me when I met him for the first time was his relative lack of balance. I also noted that throughout the interview with him his speech was rather dysarthric and a very limited and focused examination of his nervous system with regard to cerebellar function showed gross distortion of his co-ordination. Past pointing was abnormal as was dysidiadocokinesis and he had normal nystagmus. As I mentioned . . he appeared dysarthric and his gait and balance were abnormal"*.

80. He recommended to the plaintiff's General Practitioner, Dr. Coyne that an MRI scan should be undertaken in respect of the brain and cerebellum and that other neurological reviews should be undertaken.

81. He concluded *"it is striking that these abnormalities have been present for the last six years and he tells me that people comment that he seems drunk and his speech is compatible with that. His wife has noticed this as has his family but he has never sought any help for this as he thought this was just part of the problems associated with his illness back in 1998. It may well be that this is all related to the severity of that illness but my concern would be that there has been some damage to cerebellum and clearly this needs further investigation. Its temporal relationship to the events of that time is striking"*.

82. Three consultant neurologists testified in these proceedings. They were:

(1) Professor Leslie J. Findley, who is a Consultant Neurologist at Essex Centre for Neurological Sciences in Queens Hospital, Romford, Essex, England,

(2) Professor Michael Hutchinson who is a Consultant Neurologist in St. Vincent's Hospital, Elm Park, Dublin 4; and

(3) Dr. Donal Costigan who is a Consultant Neurologist in the Mater Private Hospital, the Bon Secours Hospital and the Blackrock Clinic.

83. They were in agreement that:

1. The plaintiff suffers from a condition called "mitochondrial cytopathy" which is a pre-morbid mitochondrial disease. This disease can include abnormal eye movements and muscle dysfunction (myopathy). This mitochondrial disorder is associated with a disorder of eye muscle control. The plaintiff has investigational evidence of mild structural changes in muscle consistent with a mitochondrial disorder. He was investigated as a child for small stature but there was no specific finding on that issue.
2. Between childhood and the date when he first demonstrated the symptoms which ultimately resulted in his contracting acute pancreatitis, the plaintiff enjoyed excellent health. He was employed in a manually demanding occupation. He exercised energetically and participated in football and athletic pursuits vigorously and successfully. He did not and does not smoke or drink. His parents are well and he has four siblings who are all blessed with healthy children. He visited his General Practitioner on only four occasions during several years immediately preceding 1998. He presently has one child, a daughter, who is in good health.
3. Within six months or thereabouts after he contracted acute pancreatitis in 1998 he exhibited symptoms which included ataxia and dysarthria. He still suffers from those symptoms.
4. Consequent upon the development of acute pancreatitis, he required in-patient treatment in hospital for more than three months during which the pancreatic cyst which had developed was drained and he required an external drain to assist in his treatment. His clinical situation was complicated by severe infections requiring repeated courses of antibiotics intravenously. He had major nutritional problems and profound weight loss including the requirement of total parenteral nutrition via a central intravenous line.
5. He had recurrent anaemia during his treatment in hospital which required blood transfusions. He also required narcotic analgesia at that time and after his discharge.
6. During the time which is material to these proceedings he has been examined repeatedly on numerous occasions by a number of medical experts (probably in excess of fifteen). Those experts included Consultant Gastroenterologists, Endocrinologists, Neurologists, Physicians and Surgeons.

A variety of different conflicting opinions have been expressed by these expert witnesses on the various issues which arose in these proceedings. There was (and remains) almost total disagreement between the expert witnesses as to what medical condition or disease caused the initial symptoms which gave rise to the performance upon the plaintiff of an ERCP.

The most frequently expressed (and most plausible) expert view on that issue is that the medical condition or disease which caused those symptoms cannot be established with any degree of conviction. I accept that view.

7. Professor Findley was of the firm opinion that the plaintiff was metabolically challenged during his treatment for pancreatitis and that he suffered damage to the brain resulting in ataxia and loss of co-ordination. He said the combination of intravenous antibiotics, blood transfusions and total parenteral intravenous feeding probably resulted in the alteration of the function of the blood brain barrier which is a protective mechanism separating the brain from the rest of the body. He thought it was unlikely that the plaintiff had developed a mitochondrial encephalopathy as a result of his pancreatitis.
8. Professor Hutchinson disagreed with Professor Findley. He said that a patient who is toxic and has an infection may be confused but will not develop a cerebellar ataxia. He said he knew of no evidence that the type of ataxia from which the plaintiff suffers can be caused by the mechanisms described by Professor Findley. He said that he has never encountered in practice or in medical literature evidence that parenteral nutrition has in the past been discovered as a cause of cerebellar ataxia.

He placed considerable emphasis upon his conclusion that the plaintiff's mitochondrial cytopathy condition has been progressive and has worsened significantly during the last nine years. He based that conclusion on a comparison between his examination of the plaintiff and the reports of Dr. Stern and Dr. Hawkins, who had examined the plaintiff on earlier occasions.

Dr. Hawkins' findings, (which were contained within a one-page letter), demonstrated that he had completely failed to appreciate that the plaintiff was suffering from symptoms consistent with ataxia and dysarthria. The plaintiff was actually suffering from those symptoms for a very long time before April, 2006, (when he was examined by Dr. Hawkins). That fact has been acknowledged by all of the parties to these proceedings and by all of the witnesses who have testified

Dr. Hawkins inexplicably diagnosed a condition known as myasthenia gravis as the cause of the plaintiff's earlier symptoms. It is acknowledged by all of the Neurologists and other expert medical witnesses who testified in these proceedings that that was a wholly mistaken diagnosis.

Professor Hutchinson acknowledged that the plaintiff suffers from ataxia and dysarthria and that his symptoms commenced shortly after the acute pancreatitis from which he suffered in 1998. His reliance upon findings of Dr. Hawkins is, accordingly, questionable.

Professor Hutchinson said that the ataxia and dysarthria were due to a mitochondrial disorder and not to pancreatitis. He agreed that the plaintiff was acutely ill as a result of his pancreatitis but said that if the plaintiff had developed a cerebellar disorder due to the pancreatitis he, (Dr. Hutchinson), would have expected his symptoms to present in the hospital and not to develop six months after discharge.

Although he acknowledged that the plaintiff's ataxia would not necessarily have been evident immediately after he left hospital (because he was in a wheelchair and his gait would not have been noticeable), he felt that the plaintiff's slurred speech should have been evident to properly qualified medical practitioners. He said that thiamine deficiency was unlikely to have caused brain damage in the manner suggested by Professor Findley.

9. Dr. Donal Costigan, in evidence, was of the opinion that the plaintiff's neurological complaints were unrelated to the pancreatitis from which he suffered in 1998.

84. He said that the MRI scan carried out on the plaintiff did not rule out mitochondrial disorder as the source of the plaintiff's ataxia. He agreed with Professor Hutchinson that thiamine deficiency was unlikely to have caused brain damage and consequent ataxia to the plaintiff because, he said, it could be assumed that the dieticians in Mercy Hospital would not have omitted an appropriate level of thiamine within the parenteral feeding.

85. He said that the history given by the plaintiff ... *"and the only available inferential landmarks in lifestyle do not furnish a clear history of a major prerogative ataxia. I would concede that absolutely - but all I am saying is that because the acute illness contains no acute insult that I can find in the clinical record or even in his own account to give me an alternative starting point, I am forced to conclude what the obvious alternative mechanism is. We may never know. I do not think the plaintiff has been able to demonstrate..(that).. during the course of that illness he experienced some agreed insult to the brain and it is all very well to say the blood brain barrier was opened from time to time in animals but it's quite another matter to say you have shown some significant blow to this man's CNS (central nervous system) that occurred in that hospital"*.

Decision

86. In summary, the evidence adduced on behalf of the plaintiff has established that he suffered from a pre-existing neurological condition, (a mitochondrial cytopathy), which has a variable prognosis and progression. The nature of the condition is such that it will usually remain static indefinitely but can manifest itself in a variety of different forms, including the development (and possibly progression) of ataxia and dysarthria.

87. The plaintiff's condition was static prior to the development of his pancreatitis in 1998. The evidence has established that he developed ataxia and dysarthria some months after he had suffered a very severe bout of severe and acute pancreatitis between May and September, 1998 and thereafter.

88. Professor Findley, on behalf of the plaintiff, contends that the ataxia and dysarthria were caused when the plaintiff's blood brain barrier was breached either; (i) by the intravenous antibiotics or, (ii) the parenteral intravenous feeding (including a possible thiamine deficiency), or (iii) by the blood transfusions resulting from anaemia or, (iv) by the repeated intravenous narcotic analgesia required to treat the plaintiff's illness, or, (v) by a combination of all of those factors.

89. On behalf of the defendants it is contended that intravenous antibiotics and parenteral intravenous feeding (including total parenteral intravenous feeding), blood transfusions and intravenous narcotic analgesia are frequent treatments required in hospitals throughout the country and no similar brain damage has been reported or can be adduced in evidence by way of comparison.

90. It is argued that the development of ataxia and dysarthria by the plaintiff is as a consequence of the mitochondrial disorder from which he suffers and that his development of those symptoms soon after his severe pancreatitis was coincidental in nature.

91. There is a clear conflict between the views expressed in evidence by Professor Findley, on the one hand, and by Professor Hutchinson and Dr. Costigan, on the other.

92. Differences of opinion between eminent medical practitioners on issues such as those which have arisen in these proceedings are by no means unusual. I have no doubt whatever that the views expressed are conscientiously held by the expert witnesses who expressed them. Each of the witnesses, in evidence, provided cogent and logical reasons in support of the views which they expressed.

93. It is well established that where negligent medical treatment is alleged against a doctor and there is an honest difference of opinion between two eminent doctors as to which is the better way of treating a patient, it is not negligent for a doctor to accept one view rather than the other (see *O'Donovan v. Cork County Council and Others* [1967] I.R. 173, *Daniels and Another v. Heskin* [1954] I.R. 73 and *Dunne (an infant) v. National Maternity Hospital* [1989] I.R. 91).

94. In the instant case, it has been acknowledged that the plaintiff was subjected by the defendants to treatment which was negligent. The issue of negligence is not in contention.

95. What falls to be determined is whether admitted negligent medical treatment gave rise to particular medical consequences for the plaintiff.

96. Insofar as that issue must be determined, I take the view that it should be determined by this Court applying the ordinary civil standard of proof, that is to say, by requiring that the plaintiff to establish what he seeks to prove by way of evidence and on the balance of probabilities.

97. I found the plaintiff to be a conscientious and truthful witness and I accept his evidence in relation to his neurological symptoms and the treatment which he required, without qualification.

98. Undeniably, the plaintiff enjoyed robust good health until the events which commenced in February, 1998. He had enjoyed a healthy and active childhood and youth and was in constant full-time employment from the age of eighteen years.

99. It is not without significance that he chose and was well capable of manual employment as a young man and was working as a manual labourer when he suffered the illness which has given rise to these proceedings.

100. He was an active and enthusiastic footballer during his youth and early twenties and I accept his evidence that he was still playing indoor football regularly and without difficulty up to February, 1998.

101. It is not disputed that, after the plaintiff was discharged from hospital in August, 1998, his weight was recorded at fifty-seven kilograms and he was weak and severely debilitated.

102. He had to be readmitted to hospital on a number of occasions and was an in-patient from 2nd September, 1998, for six nights. When he was married two weeks later, he was still debilitated and had to remain seated during the wedding ceremony. He had to cancel his honeymoon and was readmitted to hospital in January, 1999 and in May, 1999 for treatment of symptoms arising out of his pancreatitis.

103. I accept the plaintiff's evidence that as his strength returned during the months after his discharge from hospital, he became increasingly conscious of problems with his balance and co-ordination. He noticed that he needed to use a handrail going up and down stairs and often swayed and sometimes lost balance.

104. He found that he was unable to carry a cup of tea from one room to another without causing a spillage. He was unable to return to the indoor football, in which he had actively and enthusiastically participated up to February, 1998.

105. He noted that by reason of his difficulty with balance and his slightly slurred speech, he often appeared to other persons to be intoxicated.

106. I accept his evidence that initially he attributed his impaired balance and speech to the aftermath of his pancreatitis and to the weight loss and debilitating symptoms which had resulted from that illness.

107. I also accept his evidence that his expectations did not materialise and his difficulties with speech, balance and co-ordination remained permanent and have not improved or recovered in any respect.

108. I further accept his evidence that his speech, his balance and his co-ordination difficulties have not worsened since he first became conscious of them in the months following his discharge from hospital.

109. It follows that, almost immediately after very lengthy and intensive treatment for the effects of pancreatitis, the plaintiff exhibited symptoms consistent with ataxia and dysarthria. He had no such symptoms before April, 1998.

110. Professor Hutchinson and Dr. Costigan, in evidence, expressed the view that there was no connection between the plaintiff's treatment for pancreatitis and the fact that he developed ataxia and dysarthria immediately after that intensive treatment. They said that the temporal relationship between the two events was coincidental in nature.

111. Dr. Connor described the temporal relationship as "*striking*" and recommended that it be investigated. Professor Findley was firm in his opinion that the plaintiff's ataxia and dysarthria were the direct result of the very severe and intensive treatment which he required for the acute pancreatitis from which he was suffering.

112. As I have indicated, I am satisfied that I am required to determine this issue on the evidence and on the balance of probabilities.

113. In short, Professor Hutchinson and Dr. Costigan took the view that the plaintiff's pre-existing mitochondrial disorder, which had been dormant for almost thirty years, manifested itself, coincidentally, in the autumn of 1998, almost immediately after the plaintiff had been subjected to more than three months of very severe and intensive treatment for the effects of acute pancreatitis.

114. In support of that conclusion, they relied upon the findings; (a) that the plaintiff's symptoms of ataxia and dysarthria were progressive and have worsened significantly during the last nine years, and, (b) that damage to the blood-brain barrier arising from this type of medical treatment has never been recorded in the past.

115. As I have already indicated, I am satisfied on the evidence and on the balance of probabilities that the plaintiff's symptoms of ataxia and dysarthria were not progressive and have not worsened significantly during the last nine years.

116. Professor Findlay, in evidence, insisted that the plaintiff's condition resulted from his intensive treatment for pancreatitis. In evidence he gave a detailed explanation for his opinion.

117. I am satisfied that, whilst it is possible that the development by the plaintiff of ataxia and dysarthria immediately after his treatment for pancreatitis was coincidental, it is probable that the explanations provided by Professor Findlay are correct and the plaintiff's condition resulted from the very lengthy and severe treatment which he required arising out of his acute pancreatitis.

118. It follows that the plaintiff had discharged the onus of proving, on the evidence and on the balance of probabilities, that the symptoms of ataxia and dysarthria from which he has suffered since the autumn of 1998, and from which he will suffer into the future, was caused by reason of negligence and breach of duty on the part of the defendants, their servants and agents.

Damages

119. The consequences of the ataxia and dysarthria from which the plaintiff suffers, and has suffered for almost ten years, have been detailed in evidence.

120. He has dealt with his ongoing injury and symptoms commendably and has overcome many of the adverse consequences of such a condition. He has secured employment and been promoted to a senior position within his employer's company.

121. He has built a home and family with admirable energy and resourcefulness.

122. However, he has been unable to return to his favourite sport and suffers embarrassment as well as disability on a daily basis and will do so for the remainder of his life.

123. His disability is of some significance, notwithstanding his success in overcoming many of its effects. He has suffered on an ongoing basis over the past ten years and will continue to do so indefinitely into the future.

124. I am satisfied that he is entitled to recover the sum of €50,000 by way of General Damages to compensate him for the suffering, restriction, discomfort, embarrassment inconvenience and disruption of his life which he has suffered between September, 1998 and the date of trial.

125. I would assess the General Damages which he is entitled to recover to compensate him for the further suffering, restriction, embarrassment, discomfort, inconvenience and disruption of his lifestyle from the date of the trial into the future, at €100,000.

126. To these awards must be added the sum of €75,000, already awarded to the plaintiff by way of General Damages to compensate him for the consequences of his pancreatitis. There will, therefore, be a judgment for the plaintiff in the amount of €225,000.