Case No: 361/98

IN THE SUPREME COURT OF APPEAL OF SOUTH AFRICA

REPORTABLE

In the matter of:

LEONIDAS SOUZOU MICHAEL	**First Appellant**
THELMA MICHAEL	**Second Appellant**

and

LINKSFIELD PARK CLINIC (PTY) LIMITED	**First Respondent**
DR HUGH M THOMAS	**Second Respondent**

CORAM: **Howie, Farlam JJA and Chetty AJA**

Date Heard: **19 February 2001**

Date Delivered: **13 March 2001**

Medical negligence alleged - approach to expert evidence - whether a case for adverse costs order against successful defendant **.**

J U D G M E N T

THE COURT

INTRODUCTION

[1] This is a tragic case indeed. A boy of seventeen underwent corrective nasal surgery and suffered cardiac arrest while under general anaesthesia. By the time resuscitation had restored heart function he had sustained major brain damage as a result of cerebral anoxia. He has been left in a permanent vegetative state.

[2] His parents sued for damages in the High Court at Johannesburg. The private company owning the clinic where the operation was performed was cited as the first defendant and the anaesthetist as the second defendant. Negligence was alleged on the latter's part in relation to the cardiac arrest and joint negligence was alleged in respect of the resuscitation process. By agreement between the parties the trial Judge (Schabort J) was asked to determine only the question of liability. Having found that none of the alleged

negligence had been proved, the learned Judge dismissed the claim but granted leave for this appeal. For convenience we shall refer to the parties by their trial designations.

THE FACTS NOT IN ISSUE

[3] The following facts are now common cause or no longer realistically disputable. The plaintiffs' son, Minas, ("the patient") had sustained an injury to his nose when taking part in sport. He consulted a plastic and reconstructive surgeon, Dr MS Fayman, who recommended a rhinoplasty. The object was to remove a hump on the dorsal aspect of the nose and to correct a deviated septum.

[4] The operation was arranged for 10:00 on 7 December 1994 at the first defendant's clinic. Dr Fayman was assisted by Dr Grace Rubin and the second defendant, a specialist in anaesthesiology, was the anaesthetist. All

three doctors were in private practice.

[5] Among the first defendant's employees involved in the events of that morning were Sister S Montgomery, the sister in general charge of anaesthetics and recovery, and Sister DE Glaeser who was the anaesthetic sister assigned to this particular operation. They were both registered nurses.

[6] Included in the clinic's emergency equipment was a resuscitation trolley carrying, among other things, a Lohmeier defibrillator. A defibrillator is a portable electronic apparatus designed to restore normal rhythm to a fibrillating heart by way of electric shocks applied to the chest wall. It was among Sister Glaeser's duties to see to it beforehand that this defibrillator was in working order and to use it when called upon by the second defendant to do so. As anaesthetist, he was in overall charge of all necessary resuscitation measures.

[7] At about 9:40 the pre-operative process started. The initial stages

included the insertion into the patient's left hand of an intravenous tube connected to an AFC 123 drip-line and the attachment to his person of leads from items of equipment reflecting, *inter alia*, blood pressure, heart rate and electrocardiographic ("ECG") tracings of heart rhythm.

[8] Anaesthetic induction commenced at about 9:45 employing a combination of inhalants and intravenous drugs. Among the drugs administered intravenously was one milligram of propranolol hydrochloride ("propranolol") which was given to prevent an untoward increase in heart rate during the operation. Propranolol in medical parlance is a beta blocker. It lowers excessive heart rates by blocking the beta adrenergic receptors in the heart which govern heart rate stimulation. It is manufactured in tablet form and also in one milligram (one millilitre) ampoules for intravenous administration. In South Africa it is sold, *inter alia*, under the trade name

"Inderal". The package insert published in November 1993 by the South African distributors of Inderal stated that intravenous administration was for the emergency treatment of cardiac dysrhythmias especially including supra-ventricular tachydysrhythmias. The recommended dose was one milligram injected over one minute which could be repeated at two minute intervals until a response was observed or to a maximum, in the case of anaesthetised patients, of five milligrams.

[9] At roughly 9:50, with the patient now fully generally anaesthetised, Dr Fayman injected a local anaesthetic (lignocaine and adrenaline) into the nose and inserted at the back of each nostril a plug of ribbon gauze soaked in a cocaine solution. The use of cocaine had a two-fold purpose. It is a local anaesthetic and a vasoconstrictor. The blood vessels of the nasal lining bleed very readily and it was necessary to constrict them to ensure a clear field for

the surgeon. Cocaine is widely used for this purpose in ear, nose and throat surgery. The mass of cocaine in the solution was approximately 150 milligrams (being 1,76 milligrams per kilogram of the patient's weight, which was eighty-five kilograms.) The limits of a safe dose are from 1,5 milligrams to 2 milligrams per kilogram. Because not all of the solution was in contact with the inner nasal surfaces only about eighty per cent of the cocaine would have been absorbed.

[10] Cocaine, either in overdose or in patient over-reaction, has cardio-toxic effects which can lead to cardiac arrest. One of these is its local anaesthetic effect, which impairs electrical conduction within the heart and diminishes the contractility of the myocardium - the heart muscle. Another is its propensity to result in coronary vasospasm which leads to myocardial ischemia. Cocaine toxicity exhibits a well-known pattern of heart reaction, first hypertension and

tachycardia, then ventricular arrhythmias, then falling blood pressure and heart rate, then ventricular fibrillation and finally cardiac arrest.

[11] At 10:00 the operation began. The kind of operation in question usually took Dr Fayman about one hour and involved, after an incision in each nostril to enable lifting the soft tissue off the ridge of the nose, operating first in one nostril and then in the other. The surgery encompassed lowering the bony ridge to the desired degree by rasping it from both sides and then trimming the cartilaginous portion of the nose with a scalpel. Dr Fayman completed the rasping process on the left side and went on to operate on the right.

[12] Between 10:15 and 10:28, while surgery was in progress, bleeding in the nose suddenly occurred in the right nostril which obscured the surgical field and brought the operation to a stop. With the bleeding there was a dramatic and alarming increase in the patient's heart rate and blood pressure. In the

evidence this high level of heart rate (tachycardia) and high blood pressure (hypertension) was called “the hypertensive crisis” and the tachycardia itself was identified as a supra-ventricular tachydysrhythmia. The second defendant diagnosed too light anaesthesia as the cause of the crisis. This did not mean inadequate anaesthesia. The difference is that adequate anaesthesia can during surgery become too light by reason, not of reduction in anaesthetic, but of excessive surgical stimulus . He deepened the degree of anaesthesia, and to bring down the heart rate and blood pressure, which presented the risk of cerebral haemorrhage, he injected a further one milligram of propranolol into the drip-line. The heart rate and blood pressure came down as intended but thereafter they continued to decline. At below sixty beats per minute the heart rate became what is called bradycardia. Early in the bradycardia the ECG monitor displayed features of a normal tracing, including the characteristic

peak and lows referred to as the QRS complex. This complex then soon broadened, indicating a symptomatic bradycardia. At about this time the second defendant instructed Dr Fayman to undertake cardio-pulmonary resuscitation ("CPR") by way of external heart massage. (Unless after this there is specific reference to the first dose of propranolol we shall only speak of the later one.)

[13] The second defendant considered that there had been an over-action by the propranolol and to counter it he started administering, in conjunction with the CPR, a sequence of different drugs (ephedrine, isoprenaline and adrenaline) to try to raise the heart rate and blood pressure by removing the beta blockade. All these measures failed and the patient's heart went into cardiac arrest at 10:28.

[14] Shortly before the arrest the second defendant noted that the ECG

tracing had become a flat line. In other words there was no discernible wave. This led him to conclude that the patient's heart was in a state known as asystole, in which there is no electrical activity in the heart at all. Because shocking by defibrillator damages an asystolic heart he considered he was confined in his resuscitation efforts to CPR and drug therapy, those being the only measures by which rhythm can be restored if the heart is in that state. When, after about four minutes, these efforts failed to yield any apparent result, the second defendant's options were to leave the patient for dead or to employ the defibrillator in the hope that if the heart was not in asystole but in ventricular fibrillation a heart beat could be restored by defibrillation. A fibrillating heart is one in which there are electrical impulses but no rhythm and no output. Its energy goes into rapid, random, unco-ordinated contractions, all in complete disorder. What defibrillation does is to shock a fibrillating

heart into momentary asystole and afford it the opportunity for a normal beat to resume spontaneously. (As ventricular fibrillation is the only form of fibrillation which need be mentioned we shall, from now on simply refer to fibrillation.)

[15] The Lohmeier defibrillator ("the Lohmeier") was therefore brought into action. On the second defendant's instructions Sister Glaeser set the device to deliver a charge of 200 joules. When she did so she noticed that the number of joules digitally displayed as reflecting the strength of the required charge did not stay at 200 but started decreasing while she was busy preparing to activate the defibrillator. She nevertheless proceeded to cause delivery of a shock. The patient's body responded but not his heart. For some minutes after that, CPR and adrenaline were repeated. A second shock at 200 joules was ordered. The outcome was the same. Again the number of joules on the

display fell before the shock could be given. After renewed CPR and further adrenaline a third shock was ordered, this time at 360 joules. The heart remained in arrest. Once more the digital display decreased. Because Sister Glaeser and the second defendant thought that the diminishing display indicated that the apparatus was failing to hold its charge and was therefore defective, Sister Montgomery was sent to fetch another defibrillator. CPR and adrenaline were repeated. In addition, bretylium tosylate, sodium bicarbonate and calcium gluconate were injected into the drip-line.

[16] From the Intensive Care Unit Sister Montgomery returned in due course with another make of defibrillator. When programmed to deliver a charge of 360 joules, its digital display remained constant. With the new defibrillator a fourth and fifth shock were given. Both elicited a body reaction and, in addition, a heart beat. The fourth resulted in ventricular tachycardia and the

fifth, sinus tachycardia - a fast but normal rhythm. By the time heart action was restored it was 10:44. Further resuscitation was required in the Intensive Care Unit and so the operation was not completed. The nasal wounds were simply closed and the patient's nose was plugged and splinted.

[17] Prior to the cardiac arrest, and more or less contemporaneously, the second defendant recorded certain data regarding the operation. He used both sides of a stereotyped form which he himself designed and which he had had printed. One side was referred to as his "chart". His recordings were interrupted entirely by the arrest and resuscitation but later that morning he made further entries on the reverse side of the form under the heading "Additional notes". We shall refer respectively to the "chart" and to the "additional notes". Later during the day he spoke to the plaintiffs and, in expressing his regret for what had happened, said of the operation that

everything had been done correctly and that he did not know what had gone wrong.

[18] During the afternoon the first defendant's general manager, Dr Malkin, spoke to Sister Glaeser. In recounting the morning's events, she indicated that in comparison with the second defibrillator the Lohmeier had seemed to be defective. In consequence Dr Malkin wrote to the suppliers of the Lohmeier alleging that the resuscitation had failed because the defibrillator was unable to maintain the required charge and expressing concern that there had been a delay in the resuscitation. This prompted a number of independent tests of the apparatus concerned during the following year, the result of all of which was that it was reported to be in working order. It was also established that in all defibrillators the programmed charge diminishes between the time it is set and the delivery of a shock. This is due to electrical resistance within

the apparatus. Lohmeiers constitute the only make whose digital display reflects that reduction and Sister Glaeser and the second defendant did not know this.

[19] At 19:00 on the day of the operation the patient was examined by a cardiologist, Dr JL Salitan, who performed an echocardiogram. He later reported that the patient's heart was enlarged and its left ventricular contractility significantly reduced. His conclusion was that there was "marked global myocardial dysfunction, probably acute", possibly the result of prolonged hypoxia. Obviously prolonged hypoxia did occur and although it is in dispute precisely by what mechanism the myocardial damage came to be caused, what is not in issue is that hypoxia caused injury to the brain. Brain injury was sustained after the heart went into cardiac arrest and was ongoing for as long as the resuscitation period advanced without restoration of a heart

beat.

THE ALLEGED NEGLIGENCE

[20] Of the many grounds of negligence set out in the particulars of claim (as amended) the following summary reflects those which remain in tenable contention at this stage. As regards the first defendant it is alleged, in effect, that:

1. In respect of the Lohmeier, it failed to have a functional defibrillator immediately available when required.

2. Alternatively, if the Lohmeier was functional, first defendant failed (at a time prior to the date in question) to inform Sister Glaeser about, and to train her in, the workings and manner of operation of the Lohmeier, thereby causing delay in the resuscitation process when the

Lohmeier appeared to her to be defective and to require replacement by a substitute defibrillator.

[21] As regards the second defendant it is alleged in relation to the cardiac arrest that:

1. He failed to take adequate account of the effect which the cocaine would have in conjunction with what he himself administered and to guide Dr Fayman as to the upper dose limits of cocaine.

2. He failed to dilute the propranolol which was given to combat the hypertensive crisis or to administer it in doses of between 100 micrograms and 500 micrograms at a time.

3. The use of propranolol in conjunction with cocaine created the risk of sudden heart failure.

4. He failed to recognise the risk of, or to prevent, life-threatening bradycardia and cardiac arrest.

In relation to the resuscitation it is alleged that:

5. He failed to ensure beforehand that a functional defibrillator was available and that he was reasonably acquainted with its workings. This caused a delay in the resuscitation process when a second defibrillator was sent for.

6. When the patient's heart was in fibrillation he failed to order defibrillation at the earliest opportunity. Alternatively, he attempted defibrillation on an asystolic heart thereby worsening the outcome. In the further alternative he failed to deliver three quick shocks in a

"stacked sequence" in accordance with certain published algorithms approved for emergency cardiac resuscitation.

[22] We must mention for the sake of completeness that on examination by the second defendant on the morning of the operation the patient told him that he had managed over the preceding year to reduce his weight from 114 to 85 kilograms. The second defendant did not enquire whether this appreciable weight loss had possibly involved the use of any drugs harmful or potentially harmful to the heart when used in combination with other drugs. However, the plaintiffs' evidence was that the patient had not used any such substances and no fault was attributed in the pleadings to the second defendant's omission to investigate this possibility. We must therefore ignore it.

THE ISSUES FOR DECISION

[23] The first essential issue is the cause of the cardiac arrest. The

plaintiffs' contention is that it was propranolol and that the hypertensive crisis was occasioned by too light anaesthesia. For the second defendant it is maintained that the cause of both the hypertensive crisis and the arrest was cocaine toxicity. In either event the question then is whether the arrest was foreseeable as a reasonable possibility, meaning a possibility which a reasonable anaesthetist would foresee and guard against. Finally, if the cause of the arrest was cocaine toxicity and the arrest was indeed foreseeable in that sense, the question would then be whether the arrest was reasonably avoidable.

[24] The main subsidiary question allied to the first issue concerns the length of time between the hypertensive crisis and the cardiac arrest and that, in turn, depends on the credibility and reliability of the witnesses who were centrally involved in the operating theatre at the time. For the plaintiffs they were

Doctor Fayman, Doctor Rubin and Sister Glaeser. On the opposite side the second defendant stood alone.

[25] The other subsidiary questions are whether, irrespective of the cause of the arrest and irrespective of the correctness of his conclusions, the second defendant was reasonable in diagnosing too light anaesthesia as the cause of the hypertensive crisis and in giving propranolol as the counter; whether he was at fault in relation to either the size of the dose or the manner of its administration; and whether it was reasonable to diagnose a propranolol over-action as the cause of the bradycardia.

[26] The second essential issue is whether the Lohmeier was defective and, if not, whether the ignorance of the second defendant and Sister Glaeser as to the manner of its workings was culpable and whether their ignorance occasioned an unreasonable delay in the resuscitative process.

[27] Allied questions (accepting that when the fourth shock was given there must unquestionably have been fibrillation present) are whether the heart arrested in asystole or fibrillation; when fibrillation occurred if initially there was asystole; whether fibrillation was immediately amenable to defibrillation and, if not, when it first became amenable. Finally, on the matter of delay, the crucial enquiry is whether the fourth shock (and the fifth if required) would have been given materially earlier had the Lohmeier been in proper working order and had Sister Glaeser and the second defendant known that. The answer to that enquiry entails examination of what resuscitation measures were in progress between the third and fourth shocks and whether the picture would have been different in the absence of their ignorance.

[28] In support of the parties' rival contentions regarding all these points of dispute five expert witnesses testified. The plaintiffs called Professor R

Koorn, Associate Professor of Clinical Anaesthesia at Columbia University, New York and Emeritus Professor D G Moyes, former Academic Head, Anaesthesia, at Witwatersrand University and currently Professor at the University of Adelaide, Australia. First defendant called Professor Pierre Fourie, Clinical Head of the Department of Anaesthesiology at Pretoria University. The expert testimony on behalf of second defendant was given by Professor AR Coetzee, Head of the Department of Anaesthesiology at Stellenbosch University and his counterpart at the University of Cape Town, Professor M F M James.

THE FINDINGS OF THE TRIAL COURT

[29] The trial court found that the cause of the cardiac arrest was in all probability cocaine toxicity. This finding was based, in essence, on the evidence of the second defendant and Professor James and Professor Coetzee. Despite holding that the second defendant had given untruthful

evidence concerning the extent of the dilution and the manner of administration of the propranolol and had also falsely alleged having changed the ECG leads to try to interpret the flat line tracing, the court accepted his evidence in all other respects. In particular, the Judge believed his account of events between the administration of the propranolol and the arrest. In this regard the second defendant testified that within a couple of minutes of the propranolol being given the patient's heart rate, blood pressure and other vital signs reverted to normal levels and remained there till shortly before the onset of bradycardia. On this evidence the court found that there was a so-called period of normality of some six or seven minutes following upon the propranolol being administered.

[30] In coming to this conclusion the Judge relied not only on the second defendant's evidence but also on the evidence of Dr Rubin, preferring what they had to say to the evidence of Dr Fayman that the whole sequence from

tachycardia to bradycardia was "very, very rapid". The Judge expressed that preference notwithstanding the finding that Dr Fayman was honest, objective and a fair observer. The only respect in which Dr Fayman's evidence was preferred to that of the second defendant was in relation to what precise procedure was being performed at the time when the hypertensive crisis arose and whether, as alleged by the second defendant, the cocaine plugs were removed at that stage. As for Sister Glaeser, the court found "on the basis of her demeanour and her testimony as a whole" that she was not reliable concerning the speed with which the patient's heart rate and blood pressure changed.

[31] Following from these findings of credibility the Judge held that the propranolol was injected into the drip line "*statim*" (all at once), that this achieved the period of normality referred to and that the second defendant's chart and additional notes were genuine and basically correct.

[32] Although certain of the experts, including Professor Coetzee at one stage of his evidence, considered a swift injection of one milligram of propranolol to be unreasonable, the trial court accepted Professor Coetzee's later evidence, and the opinion of Professor James, to the effect that neither the speed of administration nor the degree of dilution of a dose of one milligram would have had any adverse effect on the patient. These two experts were, in the Judge's view, independent and objective and their evidence exonerating the second defendant in this and other respects was considered at least as motivated, authoritative and impartial as any condemnatory evidence by the other experts. It was accordingly held on their evidence, and by reason also of relevant concessions by the other experts, that the second defendant's respective diagnoses of too light anaesthesia,of over-action of the propranolol and of the heart's arrested state as asystole, were reasonable.

[33] With regard to the resuscitation, the court found that the measures taken

to combat the diagnosed asystole were appropriate and that it had been reasonable to resort to, and persist with, defibrillation after that. Without a firm finding that the Lohmeier was in working order the Judge nevertheless considered it impossible to determine when the heart become susceptible to defibrillation and therefore held it not proved that the heart was in fact susceptible at any time when the Lohmeier was used. As to culpable ignorance, it was found that the first defendant had done everything reasonable to ensure that its staff was acquainted with the Lohmeier's "idiosyncratic" functioning. No finding was made as to whether the second defendant should have known about the reducing digital display. In these curcumstances the court did not consider whether further defibrillation with a properly functioning Lohmeier would have restored the heart beat and whether such restoration would have occurred any earlier than was in fact the case.

THE APPROACH TO THE EXPERT EVIDENCE

[34] In the course of the evidence counsel often asked the experts whether they thought this or that conduct was reasonable or unreasonable, or even negligent. The learned Judge was not misled by this into abdicating his decision-making duty. Nor, we are sure, did counsel intend that that should happen. However, it is perhaps as well to re-emphasise that the question of reasonableness and negligence is one for the court itself to determine on the basis of the various, and often conflicting, expert opinions presented. As a rule that determination will not involve considerations of credibility but rather the examination of the opinions and the analysis of their essential reasoning, preparatory to the court's reaching its own conclusion on the issues raised.

[35] What must be stressed in this case is that none of the experts was asked, or purported, to express a collective or representative view of what was or was not accepted as reasonable in South African specialist anaesthetist practice in 1994. Although it has often been said in South African cases that

the governing test for professional negligence is the standard of conduct of the reasonable practitioner in the particular professional field, that criterion is not always itself a helpful guide to finding the answer. The present case shows why. Apart from the absence of evidence of what practice prevailed one is not simply dealing here with the standard of, say, the reasonable attorney or advocate, where the court would be able to decide for itself what was reasonable conduct. How does one, then, establish the conduct and views of the notional reasonable anaesthetist without a collective or representative opinion? Especially where the primary function of the experts called is to teach, with the opportunity only for part-time practice. In these circumstances counsel were probably left with little option but to elicit individual views of what the respective witnesses considered reasonable.

[36] That being so, what is required in the evaluation of such evidence is to determine whether and to what extent their opinions advanced are founded on

logical reasoning. That is the thrust of the decision of the House of Lords in the medical negligence case of Bolitho v City and Hackney Health Authority [1998] AC 232 (H.L.(E.)). With the relevant *dicta* in the speech of Lord Browne-Wilkinson we respectfully agree. Summarised, they are to the following effect.

[37] The court is not bound to absolve a defendant from liability for allegedly negligent medical treatment or diagnosis just because evidence of expert opinion, albeit genuinely held, is that the treatment or diagnosis in issue accorded with sound medical practice. The court must be satisfied that such opinion has a logical basis, in other words that the expert has considered comparative risks and benefits and has reached "a defensible conclusion" (at 241 G - 242 B).

[38] If a body of professional opinion overlooks an obvious risk which could have been guarded against it will not be reasonable, even if almost

universally held (at 242 H).

[39] A defendant can properly be held liable, despite the support of a body of professional opinion sanctioning the conduct in issue, if that body of opinion is not capable of withstanding logical analysis and is therefore not reasonable. However, it will very seldom be right to conclude that views genuinely held by a competent expert are unreasonable. The assessment of medical risks and benefits is a matter of clinical judgment which the court would not normally be able to make without expert evidence and it would be wrong to decide a case by simple preference where there are conflicting views on either side, both capable of logical support. Only where expert opinion cannot be logically supported at all will it fail to provide "the benchmark by reference to which the defendant's conduct falls to be assessed" (at 243 A-E).

[40] Finally, it must be borne in mind that expert scientific witnesses do tend to assess likelihood in terms of scientific certainty. Some of the witnesses in

this case had to be diverted from doing so and were invited to express the prospects of an event's occurrence, as far as they possibly could, in terms of more practical assistance to the forensic assessment of probability, for example, as a greater or lesser than fifty per cent chance and so on. This essential difference between the scientific and the judicial measure of proof was aptly highlighted by the House of Lords in the Scottish case of Dingley v The Chief Constable, Strathclyde Police, 200 SC (HL) 77 and the warning given at 89 D-E that:

> "(o)ne cannot entirely discount the risk that by immersing himself in every detail and by looking deeply into the minds of the experts, a judge may be seduced into a position where he applies to the expert evidence the standards which the expert himself will apply to the question whether a particular thesis has been proved or disproved - instead of assessing, as a judge must do, where the balance of probabilities lies on a review of the whole of the evidence."

THE CARDIAC ARREST

[41] We proceed now to consider the issues, commencing with the question

as to what caused the cardiac arrest. The first matter to which attention must be given is whether there was a period of normality, as alleged by the second defendant, after the hypertensive crisis had been resolved and before the bradycardia manifested itself.

[42] The second defendant's evidence on that score, and to the further effect that once the blood pressure and pulse rate declined to within physiologically satisfactory limits Dr Fayman actually resumed surgery for a short while, is in conflict with the evidence of Dr Fayman, who testified that he did not resume surgery. All he did was to suction up some blood and possibly make one or two manoeuvres with his scalpel. There were other important aspects on which the evidence of Dr Fayman differed. The second defendant said that the hypertensive crisis took place shortly after 10:15, that is to say, about a quarter of an hour after the operation commenced, at a time when Dr Fayman, who up to that stage had been working on the right nostril, had just moved

over to the left nostril. According to the second defendant, after the hypertensive crisis was resolved there was a return to normality which lasted for about eight minutes until shortly before 10:25 when the patient's blood pressure and pulse rate rapidly declined, culminating in the cardiac arrest. Dr Fayman, on the other hand, testified that having operated on the left nostril for approximately fifteen minutes he worked on the right nostril for about ten to twelve minutes before he noticed what he called unexpected bleeding in the surgical field. He stated that he commented on this to the second defendant who replied that the blood pressure was high. Dr Fayman said that he then noted a tachycardia beat on the monitor. This changed rapidly "within a very short space of time" into a bradycardia. At about that stage the operation was stopped on the orders of the second defendant, the drapes were torn off the patient and he, Dr Fayman, started a closed cardiac massage which he continued, with interruptions for defibrillation, until the heart beat was restored.

His evidence in cross-examination revealed his difficulty in expressing in minutes his estimate of the period from the tachycardia to the arrest but, he emphasised, "as I recall it, under oath that is my testimony" (i.e., that there was a short period of time, with no return to normality between the tachycardia and the arrest).

[43] There is no reason seriously to question Dr Fayman's evidence, supported as it is by his operation notes made shortly after the events in question, as to the details of the surgery he had performed. He testified that the operation was routine in nature and that he had done it nearly 200 times using the same technique. He therefore had the knowledge and experience to assess how long it took him to perform the surgery he had completed. It follows that his evidence that he had operated for about twenty-five minutes, starting from 10:00, and that he had not just moved to the second nostril, as the second defendant said, but had worked on it for over ten minutes, is likely

to be clearly more reliable than the second defendant's evidence on the point. If that is so, the hypertensive crisis probably occurred close to 10:25,some three minutes before the cardiac arrest at 10:28, and not just after 10:15, as the second defendant said.

[44] Dr Fayman was, on our reading of the record, by far the best of the witnesses who testified as to the events in the theatre culminating in the arrest and subsequent resuscitation of the patient. He gave a basically clear and coherent account of what happened while he readily conceded that his estimates of the time that elapsed during what was clearly a tense and dramatic period might differ from those of other witnesses.

[45] Dr Rubin, who was found by the trial Judge to provide support for the second defendant's allegation that there was period of normality, was very vague and unclear about what had happened. Indeed, she stated in chief that it was "very difficult for me to recall independently what happened in a

chronological sequence or to recall at all because it has been a long time and I have not written anything down". She added, however, "when certain situations have been described to me, I can, in certain circumstances affirm them and certain circumstances say 'no I do not recall that'." Later she stated that what had happened was "very hazy".

[46] She said that she had been reluctant to make a statement to the plaintiffs' attorneys and had initially declined to attend a meeting with them. She gave evidence as a witness called by the plaintiffs after they subpoenaed her. She said that through the discussions which took place at consultations she eventually did attend with the plaintiff's representatives, she had recalled certain things "more or less".

[47] She testified that the first indication she had had that something unusual was happening in the course of the operation was when the resuscitation trolley was brought into the theatre. (It is clear from the evidence that the

resuscitation trolley was brought in on the second defendant's instructions after Sister Glaeser had drawn up the isoprenaline for him. This happened shortly after the bradycardia.) She then busied herself in trying to do a blood test on the patient. When the resuscitation trolley was brought in CPR was being performed.

[48] She said that surgery was being done "for most of the time" until the CPR started. She could not recall who was performing the CPR nor that a second defibrillator was brought into the theatre during the resuscitation.

[49] She testified that she recalled that Dr Fayman had mentioned during the operation that there seemed to be increased bleeding but she could not say exactly how long this was before the resuscitation trolley was brought in - "if it was half an hour, 20 minutes or 10 minutes I cannot tell you, but it was before." Contrary to Dr Fayman's evidence, she claimed that the operation had continued after the bleed had been reported by Dr Fayman until the

resuscitation trolley was brought in. Such continuation was a matter of minutes not seconds, so she said, but she could not estimate how many minutes.

[50] When she was cross-examined by counsel for the second defendant it was put to her that the patient went from a high tachycardia to a bradycardia over a period of "several minutes". She began, "That sounds right but...", counsel for the second defendant then interrupting her by asking , "That is the best you can do, it sounds right?", to which she replied in the affirmative.

[51] The resuscitation trolley was brought in at a stage after the second defendant had administered 25 milligrams of ephedrine to counter the decline in the heart rate and when the blood pressure had fallen to 70 systolic over 45 diastolic, and after he had asked for isoprenaline to be brought to him from the recovery room. Dr Rubin said that she had no recollection of drugs being fetched from outside the theatre before the trolley came in. In our view the

trial Judge erred, with respect, in failing to apportion substantial weight to Dr Fayman's evidence on this crucial aspect and in according undue weight to the evidence of Doctor Rubin.

[52] Turning to the evidence of Sister Glaeser, she testified that she had been present when the anaesthetic was administered to the patient and had then left the theatre. Some time later she was called back by Dr Rubin from the recovery room where she was attending to patients and told that the second defendant needed to see her immediately. From Dr Rubin's voice, said Sister Glaeser, there was an air of urgency about the matter. When she came into the theatre she saw the second defendant standing in front of the cardiac monitor. She could hear by the noise of the monitor that the heartbeat was fast. When she asked the second defendant what the problem was he answered that the patient had a tachycardia and ordered her to get him some propranolol from the anaesthetic trolley in the theatre. She drew up the propranolol in a syringe

and gave it to him. He then asked her to get him some isoprenaline. Because this was kept in a refrigerator she had to leave the theatre. When she came back with the isoprenaline the patient was receiving cardiac massage. She thought the second defendant was applying it but it is clear on the evidence that it was being given by Dr Fayman. She had just put the syringe containing the isoprenaline on the anaesthetic apparatus when the second defendant called for the resuscitation trolley to be brought in. She stayed in the theatre from then on.

[53] Under cross-examination by counsel for the first defendant she said that the fetching of the isoprenaline followed on the drawing up of the propranolol with no long time delay in between. When asked how quickly the patient went from the tachycardia to the low bradycardia, she said it happened "very quickly". In describing how she saw the period of time from the tachycardia to the low bradycardia, she used the word "frightening". Explaining what she

meant, she conveyed it had happened frighteningly quickly. Later she was cross-examined by counsel for the second defendant on the length of the period from the tachycardia to the bradycardia. Her answers appear from the following extract from the record:

> "Are you able to help us in saying how long the time period was from the tachycardia to the bradycardia and how long the time period was from the bradycardia to the cardiac arrest? -- No. From the tachycardia to the cardiac arrest could be ten minutes? -- I do not know.
>
> It could be five minutes? --I do not know.
>
> It could be 15 minutes? -- I do not know.
>
> You do not know. And similarly, sister, the period from the tachycardia to the bradycardia, if I call that the first period, and if I call the period from the bradycardia to the cardiac arrest the

second period, can you tell me whether the first period is longer than the second period? -- No.

Can you tell me whether they were about the same length? -- I do not know."

At another stage of her evidence, she said "I do not remember time".

[54] When one interprets that last comment in the light of the earlier-quoted answers it is apparent that her difficulty really lay in her inability, like Dr Fayman, to recall time in terms of minutes. Over all it seems fair to say that if the period between tachycardia and arrest was truly very short it is not surprising she could not recall the duration of each of its component phases. She would not have had problems, we think, recalling the fact of such a remarkably sudden change if indeed it occurred. The same holds good regarding her recall that she was summoned by Dr Rubin in order to draw up propranolol and that, apart from her going out of the theatre to fetch the

isoprenaline, the second defendant requested she stay there until the arrest.

[55] Sister Glaeser's evidence as to when she was sent for is to be compared with the second defendant's version on the same point. In a report prepared by the second defendant early in 1995 to enable his attorneys to respond to an enquiry by the plaintiffs' attorneys, the second defendant, in dealing with the situation when the patient's condition deteriorated, said the following:

> "The heart rate had deteriorated sequentially 120, 110, 70, 55. I asked someone to call Sister Glaeser to help, and administered 15mg of Ephedrine i v i." [He later stated this was incorrect: in the space of a minute he gave two doses of ephedrine, firstly 10 milligrams and thereafter, when he saw that this was not going to be sufficient, a further 15 milligrams.]

[56] In his evidence-in-chief, by contrast, he said that it was at the time of the tachycardia that he called for Sister Glaeser as he needed assistance and an extra pair of hands to assist him "in drawing up things or fetching things". He thereafter administered the propranolol which was drawn up, he said, into a 10 millilitre syringe. He testified that after that the operation continued for a short

period. Thereafter "over the following several minutes" the heart rate declined. He administered ephedrine to counter the decline in heart rate and blood pressure and then asked for isoprenaline to be brought from the recovery room, which Sister Glaeser fetched. (We shall revert to the matter of the size of syringe in due course, as also the administration of the ephedrine.)

[57] During cross-examination by counsel for the plaintiffs the second defendant said for the first time that he had called for Sister Glaeser at least twice, once at the stage of the tachycardia and later when he administered the ephedrine. When it was put to him that he only summoned her once he said:

> "I recall distinctly Sister Glaeser being called for the tachycardia ... And I recall her being called at the point of the ephedrine, whether she was in theatre at the time I have no specific recollection."

[58] In our view there are no grounds to reject the evidence of Sister Glaeser, however imprecise her recollection of temporal detail, that she was only called into the theatre once and that when she left it thereafter she did so on the

instructions of the second defendant and returned with the isoprenaline. It follows that his assertion, not contained in his preliminary report and raised for the first time in cross-examination, that she was sent for twice - with an interval in between - falls to be rejected. We find that she was called once and that between the propranolol administration at tachycardia and the ephedrine administration at bradycardia, there was good reason for her to remain on hand and not return to the patients in the recovery room. The compelling inference is that she was on urgent standby throughout that period. This ties in with the period having been a short one and, more particularly, it negates a period of normality.

[59] There is a further factor which indicates a strong preponderance of probability in favour of a finding that there was no period of normality between the tachycardia and the bradycardia. The second defendant testified that when the bradycardia took place he ascribed it to an over-action of propranolol.

Therefore he decided to solve the problem by administering isoprenaline which, as he put it, “forms the ideal antidote to propranolol”. It is common cause that propranolol reaches sixty per cent of its peak effect within the first minute of it’s administration. The second defendant testified that it has its peak effect approximately two minutes after injection. One can readily understand how he could have thought that a rapid fall from tachycardia to bradycardia occurring over a period of about two minutes could be caused by an over-action of the propranolol. What one cannot understand is how he could have ascribed to the propranolol the fall that took place from what he called physiologically normal levels to a low bradycardia some six or seven minutes after the propranolol was administered. He would have known that the propranolol had long since reached its peak. His diagnosis of an over-action of propranolol (whether the diagnosis was correct is a question which we shall consider later in this judgment) indicates on the probabilities that the decline

must have taken place during the period when, to his knowledge, the propranolol was reaching its peak or at the latest had just reached its peak.

[60] The trial Judge's finding that there was a period of six or seven minutes of normality after the propranolol was administered was based on several considerations. Firstly, he accepted the evidence of Dr Rubin and the accuracy of two allegedly contemporaneous recordals by the second defendant of monitor readings reflecting relative stability in the patient's condition shortly before the arrest. He also accepted estimates by Professor Coetzee of the various steps which the second defendant said he had taken from the onset of the hypertensive crisis until the administration of the isoprenaline. In the light of those considerations the trial Judge rejected the evidence of Dr Fayman and Sister Glaeser. He also rejected an argument, strongly advanced by the plaintiffs' counsel, based on the fact that the second defendant made entries in the additional notes meaning that there was

tachycardia with normal complex at 10:25, which contradict his recordal of tachycardia shortly after 10:16 on the chart.

[61] While we agree with the trial Judge's reasons for his conclusion regarding the recordal of tachycardia at 10:25 and that the second defendant may mistakenly have written tachycardia when he meant bradycardia, we cannot agree with the Judge's findings regarding the recordal of monitor readings purportedly showing that the patient's condition was relatively stable after the hypertensive crisis. On this aspect of the case the judgment reads as follows:

> "The medical experts commended the chart for its completeness and as a specimen of its kind, allowing, naturally, for the dynamics of an operating theatre. They did not attempt to impugn [the anaesthetic chart] on the ground that the two recordals appeared curious or faked and I can see nothing concerning them evincing that. Expert testimony proving forgery, such as might relate to writing, ink or other physical features of the document, was not adduced. The second defendant attested to an episode of stability and the regular making of these recordals at the time. The occurrence of such an episode was not ruled out by any of the experts as medically not possible and the reality of the readings and contemporaneousness of the recordals were clearly not refuted by the above

> cited excerpts from the testimony of Dr Fayman and Sr Glaeser. The second defendant admitted that he made certain entries on [the chart] after the operation had been completed, *inter alia* about the bradycardia event, and was criticised for not having obtained confirmation from any potential witnesses of monitor readings done after the operation. This did not apply to these recordals, even assuming that he should have had the presence of mind and foresight to have had the other readings witnessed - which in itself is not beyond question."

[62] The Judge did not refer to his own finding that the second defendant had lied in court on a number of aspects, most notably regarding the manner in which the second dose of propranolol was administered. Nor did he refer to the fact that the second defendant clearly lied to the patient's mother on the day of the operation when he said that nothing untoward had happened, when it is clear that he believed that the first defibrillator had not functioned properly and when he believed that, culpably or not, he had given a dose of propranolol which caused the arrest.

[63] It is also clear in our view that he was untruthful about the size of the syringe used in administering the propranolol and also about the administration of ephedrine. As regards the syringe, he maintained till late in the trial that its

size was ten millilitres but when disclosure of his original report to his attorneys was ordered by the Judge consequent upon a strenuously opposed application, it was revealed that he initially referred to a five millilitre syringe. Plainly, he intended his evidence to convey more dilution than in fact was the case. As to ephedrine, in the additional notes he recorded giving 25 millilitres of ephedrine "stat". In evidence, his version was that there were two administrations, 10 and 15 millilitres each, over the best part of a minute. That version was obviously contrived to minimise the urgency of the occasion. Having been prepared to lie to the patient's mother just after the operation and prepared to commit perjury at the trial, he was certainly capable of faking entries on his chart. Moreover, it is not clear why the Judge adverted to the absence of expert evidence proving forgery. The entries concerned were made by the second defendant himself with the pen he was using during the events in question. We do not understand how could it be said that if the entries were

false they would have appeared "curious or faked". The *ipse dixit* of the second defendant, regard being had to what has been said above about his credibility on other issues, takes the case no further. The fact that a period of normality was medically possible does not prove that it occurred and is at best a neutral factor. Nor do the reasonable time estimates for the various actions allegedly taken by the second defendant take the case much further because he himself stated that there was a period of several minutes when the patient's condition was once more stable, when, as he put it, he "limited or downgraded" his vigilance on the patient.

[64] In all the circumstances we are satisfied, on the probabilities, that the second defendant's evidence that there was a period of normality between the hypertensive crisis and the onset of the bradycardia, was adequately shown by the evidence of Dr Fayman and Sister Glaeser, in the respects already discussed, to be unacceptable. His evidence should not have been accepted

and we reject it.

[65] It is now appropriate, on the basis of the factual finding - which we make - that there was no six or seven minute period of normality between the tachycardia and the bradycardia, but rather an interval of only about three minutes of uninterrupted rapid decline, to consider what caused the cardiac arrest. At the trial counsel led prolonged and vigorous debate among the experts on this issue and on related questions.

[66] For the plaintiffs, Professor Koorn said at one point that he could not say whether cocaine toxicity or propranolol was the more likely cause of the arrest but at another stage he identified cocaine as the primary cause and propranolol as only a possible cause. He would have preferred a different beta blocker but conceded that propranolol was not contra-indicated in this case and a dose conforming to the package insert recommendation was not really open to criticism. He also said that despite the drugs administered by

the second defendant the arrest might have occurred in any event. He accepted that the diagnosis of too light anaesthesia was reasonable.

[67] Professor Moyes stated that in his view the cause of the arrest was the propranolol and in particular the manner in which it was given, although he conceded that cocaine toxicity was a possible cause. He ascribed the hypertensive crisis to too light anaesthesia and said that he would not have countered it with any beta blocker but would have merely deepened the anaesthesia. Alternatively, had he used propranolol, he would have administered a dose of 200 micrograms (one-fifth of a milligram) and waited for three or four minutes to observe the effect. In our view the existence of the hypertensive crisis and the risk it posed, coupled with the possibility that cocaine toxicity was the cause of the crisis, render both suggested courses of crisis response illogical and unconvincing. Later in his evidence Professor Moyes said that what he objected to was the use of propranolol at all, not the

one milligram dose that the patient received.

[68] Professor Fourie, on behalf of the first defendant, expressed the opinion that the hypertensive crisis was due predominantly to cocaine toxicity and to a lesser degree to too light anaesthesia. He nevertheless accepted the diagnosis of the latter cause as reasonable. As for the antidote, he had no criticism of the use of propranolol and eventually conceded that the administered dose was reasonable. Concerning the cause of the arrest, his view was that it was chiefly propranolol and to a lesser extent cocaine toxicity.

[69] The two experts who testified on behalf of the second defendant, Professor James and Professor Coetzee, each expressed the view that the hypertensive crisis and the cardiac arrest were both caused by cocaine toxicity, with Professor James adding that the propranolol might possibly have played a small role in bringing about the arrest. In their view it was reasonable to have diagnosed too light anaesthesia, to have used one milligram

of propranolol as a counter and to have diagnosed propranolol over-action as the cause of the arrest. They also considered that without the propranolol the arrest would have occurred in any event.

[70] In view of the conclusion to which we have come it is not necessary to canvass the expert evidence in more detail. A careful study of the record in the light of counsel's arguments has satisfied us that the evidence of Professor Koorn, Professor Moyes and Professor Fourie must defer to the evidence of Professor James and Professor Coetzee.

[71] As regards Professor Coetzee, we take into account that before his eventual detailed exposition on final recall late in the trial (by which stage the issue of a reasonable dose of propranolol had been reduced essentially to one milligram versus half a milligram) he had earlier decried a one milligram dose given all at once as unreasonable. The fact remains that, subsequent to the latter remark, evidence by Professor James and by Professor Coetzee himself,

and yet more evidence by both upon later recall, all of it exhaustively and painstakingly investigated, established that dilution of the dose and the speed of its administration were of no importance and that one milligram given all at once into the drip-line would have had no greater adverse effect on the patient, if any, than half a milligram or even a fifth.

[72] Professor James is an international authority on the management of hypertensive crises and on the use of beta blockers to counter them. Professor Coetzee is South Africa's leading authority on the effects of anaesthetics on the heart on heart injury related to anaesthesia, and on circulatory perfusion in resuscitation. Their respective fields of expertise include all the crucial medical issues in the case. Their eminence is, of course, no guarantee of the acceptability of their evidence and although it does reflect the extent of their knowledge and experience it is the quality of their reasoning that we consider places their evidence on a significantly higher plane than that

of the other experts. Having weighed their evidence and the arguments of counsel, we conclude that their opinions as to cocaine toxicity, as to the diagnoses by the second respondent and his responses to them, and as to his management of the resuscitation, are persuasive and authoritative and, above all, logically reasoned and supported by leading medical literature and by personal research findings and operational experience. Those considerations prevail despite, in the case of Professor Coetzee, his original mistaken impression that an excessive dose of cocaine had been used, and despite the appearance - and it may well be no more than the appearance - of a subjective tendency to fasten on to points even of insubstantial worth just because they could possibly assist the second defendant. No such criticism, or any criticism of moment can in our view be levelled at Professor James's evidence.

[73] Counsel for the second defendant submitted that on the acceptable expert evidence cardiac arrest was caused by cocaine toxicity and not by

propranolol. In particular he relied on five factors, derived from the evidence of Professor James and Professor Coetzee, which may be summarised as follows:

(1) A dose of one milligram of propranolol given could not on its own have caused the arrest Nothing in the abundant literature available on the drug revealed that this size of dose had ever had such a result and Professor Coetzee, who has used propranolol very frequently in heart surgery, said that in his experience any connection between propranolol and the arrest could be ruled out. In addition, the package insert recommended precisely this quantity and not as a maximum dose but an initial one.

(2) The presence of bizarre and broad QRS complexes just before the arrest is part of the classic picture of cocaine toxicity whereas such complexes will not be caused by a beta-blocker such as

propranolol.

(3) The ephedrine and isoprenaline would completely have countered both the initial dose of propranolol given on induction and the second dose given during the hypertensive crisis. If the propranolol had caused the arrest not only the ephedrine and isoprenaline but also the adrenaline would have restored a heart rhythm within the first few minutes after the arrest. The only explanation for the fact that the heart did not start again was the fact that the local anaesthetic effect of cocaine on the heart had not yet worn off.

(4) The fact that crepitations were noted in the patient's lungs by the second defendant before the arrest (as stated by him in his initial report mentioned above) is consistent with cocaine toxicity.

(5) Dr Salitan's echocardiogram established that the heart muscle still

showed a material injury eight hours after the arrest and that it had an ejection fraction of 24% while that of normal persons of the same age as the patient is above 65%. This was consistent with cocaine toxicity and inconsistent with an arrest caused by propranolol because after a propranolol-induced arrest the heart muscle would have recovered to at least 90% capacity within four hours after the arrest.

[74] As far as factor (4), is concerned, (the presence of crepitations before the arrest), we are not prepared to find that the second defendant's uncorroborated statement that there were such crepitations can, in view of the findings made earlier regarding his credibility, be safely accepted. It is true that lung oedema, for which crepitations are a diagnostic sign, was noted by Dr Salitan when he examined the patient on the evening of the incident but it is clear that lung oedema would be present in all cardiac failure, however caused,

and this factor accordingly does not in our view lend support to a finding that the patient's cardiac arrest was caused by cocaine toxicity.

[75] Counsel for the plaintiffs contended that a finding that there was a short period of rapid decline after the hypertensive crisis should lead to the further finding, as the natural and most compelling inference, that propranolol was in fact the cause of the arrest. The second defendant had falsely alleged a period of normality in order to put "a safe distance" between the propranolol he had administered and the arrest because he believed that the propranolol was the cause of the arrest. His lies regarding the manner in which the propranolol was diluted and the use of a 10 millilitre syringe were also explicable on the basis that he thought that the manner in which the propranolol was administered (which Professor Moyes described as "a somewhat cavalier fashion") had also contributed to the arrest.

[76] In our view the second defendant's lies regarding a period of normality

and the manner in which the propranolol was administered do indicate that he believed at the time that the propranolol had caused the arrest. It does not follow, however, that his belief was correct although due weight must be given, to the fact that, as the anaesthetist, he was in a good position to appreciate what was happening at the time and to diagnose accordingly. We have given careful consideration to this point but cannot see how it can overcome the cumulative weight of the other four factors relied on by the second defendant's counsel.

[77] It is clear from the evidence of all the experts and the literature to which they referred that although cocaine is a drug which, as Professor Moyes put it, can "produce almost anything in the heart", even in comparatively small doses, instances of cocaine toxicity under general anaesthetic are indeed very rare. On the other hand there is nothing to show that one milligram of propranolol, even if administered all at once into a drip-line, can cause cardiac

arrest in a patient such as the plaintiff's son who was young, strong and healthy. Moreover, according to Professor James, propranolol had for long been the antidote to cocaine toxicity. He also eliminated the first dose of propranolol from the reckoning, pointing to the fact that its effect had clearly been insufficient to prevent the hypertensive crisis.

[78] Although Professor James and Professor Coetzee were of the view that the second defendant's diagnosis of too light anaesthesia as the cause of the hypertensive crisis was reasonable in the circumstances they were both of the view, with the benefit of hindsight, that the true cause of the crisis was cocaine toxicity . We agree. It will be recalled that according to Dr Fayman, whose evidence on the point we prefer to that of the second defendant, he had already worked for ten to twelve minutes on the second nostril before he noted unexpected bleeding. On the probabilities the anaesthesia, which had been constantly maintained in flow and strength up to that stage, and was adequate

to counter the stimulus of all the rasping which had already been carried out, would not suddenly have become too light. This points to the only other possible cause of the hypertension, namely, cocaine, and in all probability its toxicity continued to be operative until into the resuscitation period. The classic picture of cocaine toxicity leading to cardiac arrest includes ventricular arrhythmia. The latter would have been pre-eminently the warning sign of cocaine toxicity but it was prevented, and so removed, by the effects of propranolol. If, as we find probable, cocaine toxicity was already operative at the time of the hypertensive crisis then a rapid decline of heart rate and blood pressure thereafter was to be expected. It follows that the fact that the arrest was the culmination of a rapid decline from the time the propranolol was administered does not assist the plaintiffs to overcome the cumulative effect of the factors to which we have referred.

[79] It remains to consider whether the arrest was not caused by cocaine

toxicity and propranolol working in combination. Professor James, it will be recalled, was of the opinion that cocaine toxicity was the most probable cause of the events and propranolol only possibly played a role. We can find no basis for differing from that view. Cocaine toxicity could on its own have caused the arrest while propranolol on its own could not. There is no expert evidence which directly or inferentially renders it probable that a combination of cocaine toxicity and propranolol caused the arrest. In the circumstances we have come to the conclusion that the cardiac arrest suffered by the patient was, on the probabilities, caused by cocaine toxicity alone.

[80] The next question to be considered is whether the second defendant was negligent in allowing Dr Fayman to use cocaine or in managing the case as he did when he knew that cocaine had been used.

[81] The first point to make is that the quantity of cocaine used in the solution in which the nasal plugs were soaked, viewed as a dose, was within the limits

of what was widely regarded as safe. It is also regarded as acceptable for cocaine to be used, as it was in the present case, by a plastic surgeon doing a rhinoplasty. Dr Fayman had used it a great many times with no adverse results. Professor Moyes, who originally stated that the quantity concerned was excessive, later conceded, after re-reading the literature, that it was not. Cocaine toxicity - a very rare result, on the evidence, and even more so given the quantity of cocaine actually absorbed - was therefore not at any stage reasonably foreseeable in the sense explained earlier in this judgment. Secondly, all the experts agreed that the second defendant's diagnosis of too light anaesthesia as the cause of the hypertensive crisis was reasonable. We are satisfied, in addition, that there is no basis for finding that his decision to treat it by the administration of one milligram of propranolol, in the manner indicated in the package insert, can be regarded as negligent.

[82] Even if he should have diagnosed the cause of the bradycardia as

cocaine toxicity, cardiac arrest was then virtually inevitable and there was nothing he could have done, on all the evidence, to prevent the arrest in the minimal time available. Therefore, in our view his failure to make that diagnosis, even if it had been negligent, was not causally linked to the arrest.

[83] It remains to point out that even had the evidence justified the conclusion that the arrest was due to propranolol, either alone or in part, it is seriously open to question whether the arrest would have been foreseeable as a reasonable possibility in the legally relevant sense but it is, in the result, unnecessary to pursue that enquiry.

THE RESUSCITATION PROCESS

[84] The only submission for the plaintiffs that the Lohmeier was defective essentially depends on the inference, as the most plausible one which can on all the evidence be drawn, that a heart beat would have been restored had the Lohmeier delivered the required charge when any of the first three shocks was

administered. In this connection counsel for the plaintiffs also sought to place reliance on the evidence of Sister Glaeser and the second defendant that the reaction of the patient's body was "sluggish" whereas when the second defibrillator was used the body's response was unmistakably sharper. The difficulty facing the plaintiffs on the question of the body reaction is the evidence of Dr Fayman. As already stated, we find his account of events in the theatre the most reliable of all. It was his evidence that the bodily response to all the shocks was the same. Bearing in mind that he was required, with the delivery of each shock, to cease applying CPR and to stand back and await developments he would have been well positioned to observe, as well as concerned, to note the patient's reaction, so as to know whether it was going to be necessary for him immediately to resume the cardiac massage. Sister Glaeser said she would not contradict Dr Fayman's evidence on this point and there is the additional consideration that the second defendant had the motive

to blame the Lohmeier and Sister Glaeser, to judge from evidence which it is unnecessary to recount, had the motive to be partisan towards the second defendant. We therefore cannot accept the evidence of these two witnesses in preference to Dr Fayman on the matter of the patient's reaction to the shocks with the Lohmeier.

[85] Reverting to the inference to be drawn from the failure of those three shocks to restore cardiac activity, it is essential to the plaintiffs' case that the heart was at all relevant times not only in fibrillation but in fibrillation amenable to defibrillation.

[86] The evidence is that the ECG tracing of a flat line was present from just before cardiac arrest until the heart beat was restored - that is to say, when a tracing could in fact be seen in the intervals between bouts of CPR. The expert evidence is unanimous that from a flat line one cannot deduce whether the heart's state is asystole or fine fibrillation. According to Professor James,

what fibrillation there was in this case was only ever fine because coarse fibrillation is easily discernible on the monitor and there was no sign of that. Fine fibrillation, on the weight of all the expert evidence, has poor prospects of conversion because it is not always amenable to successful defibrillation. What does, perhaps, tend to favour the plaintiffs is the consideration (also on the weight of the medical evidence) that if cocaine toxicity alone caused the cardiac arrest, the state of the heart when that occurred would probably have been fibrillation, not asystole. Consequently, if the ECG tracing remained the same after that, all the indications are that the heart was in fine fibrillation throughout the period that resuscitation was in progress. The difficulty, however, as the trial court found, is that one does not know when this fibrillation became amenable. In this regard we are left with very little more than speculation. Although Professor James did say at one stage in his evidence that he would "guess on the probabilities" that there was reversible

fibrillation present on at least one occasion when the Lohmeier was used, he later conceded, as we think he had to, that the success of the fourth shock is the only real evidence of the existence of amenable fibrillation. Although it is possible that the ongoing CPR and adrenaline were having a progressively stronger influence in rendering unamenable fibrillation amenable, that possibility is counterbalanced by the recognised fact that if fibrillation is present the best chances of defibrillation are earlier rather than later.

[87] The only other significant factor is the administration of bretylium tosylate ("bretylium") at some time between the third and fourth shocks. The second defendant testified that this drug acts in a manner analogous to injected adrenaline, namely, to enhance the output of the body's own adrenaline. It has some advantage, he said, in cases where one has not got a defibrillator that is working and the patient is in fibrillation. Professor James supported that evidence, saying that bretylium is used to treat what is called intractable

fibrillation, in other words fibrillation which resists conversion despite repeated defibrillation attempts. It is a drug that tends to stabilise the heart rhythm and to improve the chance of successful defibrillation. Although Professor James added that he could not say that bretylium actually made a difference in this case, in our view there exists, on the evidence, a realistic possibility, not overcome by any stronger possibility favouring the plaintiffs, that it was bretylium that converted a stubbornly resistant fibrillation to a state amenable to reversal and that successful defibrillation would not have been possible earlier even with a functional defibrillator.

[88] It follows that it has not been proved that the Lohmeier was defective in the respect contended for.

[89] We come, then, to the alternative argument advanced for the plaintiffs concerning the Lohmeier, namely, that Sister Glaeser and the second defendant's culpable ignorance concerning its features and operation led to an

unreasonable delay in the resuscitation.

[90] We are satisfied that the learned Judge was not entitled to find that enough had been done by first defendant to train its staff in the use of the Lohmeier. Sister Glaeser did not claim to have forgotten what she had once known or been told about the Lohmeier. She was clearly always ignorant of the fact that the reducing digital display was a feature which in no way impeded the efficacy of the apparatus. The inescapable inference is that she had not been properly instructed. Clearly it was the first defendant's responsibility to see to it that she was.

[91] The second defendant, as the anaesthetist, was the person in charge of the resuscitation efforts. He was similarly ignorant. Bearing in mind that a defibrillator is specifically intended for use in an emergency life-saving situation it is plainly a reasonable requirement that the anaesthetist must know how it works. Had Sister Glaeser been so burdened with other tasks that she had

become unavailable to operate the Lohmeier it could very well have been the second defendant's unavoidable duty to do so himself. We find, therefore, that the alleged ignorance was proved and that both defendants were negligent in this regard.

[92] The enquiry is then whether any delay in the resuscitation process resulted from such negligence. This involves comparing what was done between the third and fourth shocks with what, as far as the evidence can show, would have been done had Sister Glaeser and the second defendant not been culpably ignorant and had the Lohmeier not been discarded but used to deliver the fourth and fifth shocks.

[93] An answer favourable to the plaintiffs is only possible if in the latter scenario the fourth shock would have been not only a material degree earlier but successful. (One assumes that if a fifth shock had in that postulated situation been necessary it would have followed very soon after the fourth , as

was the actual position.)

[94] In contending for undue delay, the plaintiffs' counsel placed great emphasis on the estimate given by the second defendant in an early report to Professor Coetzee that it would have taken three to five minutes to fetch and instal the second defibrillator, which time lapse would not have occurred had the Lohmeier been known to be functional. While it is open to the plaintiffs to seek to rely on his estimate as being like an admission against interest (and therefore probably true), it was only ever an estimate and it must be weighed with the other relevant estimates, all of them made in later recollection of events of great urgency which occurred when there was no incentive for anyone to take specific note of time. The evidence of Dr Fayman was that fetching the second defibrillator took only about two minutes and Sister Montgomery - who fetched it - made an estimate of forty-five seconds. Sister Glaeser's evidence was that there was no delay in the defibrillation because she administered the

third shock while the replacement defibrillator was being fetched. In his own evidence, the second defendant said that took about three minutes. He went on to say that while it was being fetched he was busy with all those things which had engaged his attention during the intervals between the first three shocks and that when the next defibrillation was required the second defibrillator was already available.

[95] Under cross-examination he said he treated as for asystole for four minutes and indicated that the intervals between the first three shocks were each three or four minutes, "of that order". Allowing for the time it would have taken to cease massage, prepare and deliver each of the first three shocks and wait for the ECG screen to clear to see the results, it is probable that when the outcome of the third shock was apparent on the screen, the resuscitation period would already have been eleven to thirteen minutes old. One knows that the outcome of the fourth shock was electrically monitored in the sixteenth

minute but whether that shock was actually given late in the fifteenth minute one cannot know. All one can say, therefore, is that the interval between the third and fourth shocks extended for a period of between three and five minutes. Given the in-built uncertainties and the incidence of the onus, it is not possible to find that this interval was actually longer than each interval between the earlier shocks. It was never suggested that the latter intervals (three or four minutes each) were unduly long. On the evidence of the plaintiffs' witnesses and that of the second defendant, the substitute defibrillator was fetched and installed in less time than that or, at best for the plaintiffs, not longer than that.

[96] What is also clear is that in those earlier intervals repeated applications of CPR and adrenaline were essential to try to maintain blood flow to the patient's brain. The same would probably have occurred prior to the fourth shock had it been given with the Lohmeier . However, the question whether in that event the second defendant would also have administered bretylium,

the question as to how long bretylium would have taken to render the heart amenable and the question whether bretylium would have been given any earlier than it was in fact, remain unresolved.

[97] In addition, there is the real possibility that if the Lohmeier had been thought to be fully functional second defendant would not have considered resorting to bretylium. It was partly because he thought the Lohmeier was not working properly that he decided to give bretylium at all. Had he not administered it, the chance that it was bretylium that brought about an amenable fibrillation could never have arisen. In that event it cannot be said that an earlier fourth shock than was actually the case would have restored a heart beat. In our view, therefore, it was not shown on a balance of probabilities that Sister Glaeser and the second defendant's culpable ignorance caused delay in the resuscitation process.

[98] We do not overlook in this connection the letter written by Dr Malkin to

Protea Medical Services in which he complained about the Lohmeier and referred to a delay in the resuscitation. In his evidence, however, he explained that he based the contents of the letter on what Sister Glaeser reported to him on the day of the operation. He testified that she did not allege a delay and it was his inference from what she said about the functioning of the apparatus that there had been a delay. Much as the letter has the appearance of an admission it cannot possibly have the effect of an admission once, as is clear from the evidence, his inference was based not on personal knowledge or anyone else's allegation to him or any other possible factual foundation. That conclusion finally disposes of the case against the first defendant.

[99] It remains to consider the allegation that the second defendant negligently managed the resuscitation by not defibrillating earlier than he did and, in particular, by not administering three quick shocks in what was referred to in the evidence as a "stacked sequence".

[100] As to earlier defibrillation, our finding that it was reasonable in the circumstances for the second defendant to conclude that the cardiac arrest was due to propranolol over-action necessarily means that he was justified in inferring that the heart was in asystole. The expert evidence is clear that that would have been the nature of the arrest had propranolol caused it. In addition, the flat line ECG tracing strengthened that inference. Consequently it was reasonable to resuscitate as for asystole for as long as he did. To defibrillate when that was his diagnosis would have been wrong. Each defibrillation causes some damage to the heart and defibrillation would have rendered an asystolic heart less likely to be susceptible to the effects of CPR and adrenaline.

[101] After some four minutes second defendant decided to defibrillate. He made that decision not because he diagnosed fibrillation but because the alternative was to accept the patient as dead. Indeed, there never was any

indication, but for the fact that the fourth shock elicited a heart beat, that fibrillation was in fact existent. He therefore decided to take the chance that there was not asystole but fibrillation present.

[102] The evidence of Professor Moyes, in reliance upon the algorithms mentioned above, implies that the patient should then have been given three quick "stacked" shocks or nothing at all. This seems to us to be illogical. In the first place the administration of three consecutive shocks without any interval for attempts by way of CPR and adrenaline to maintain circulation to the brain would, as Professor James cogently reasoned, have been dangerous. It would have left the brain without any meaningful circulation for the best part of a minute. And if it was asystole after all, three quick shocks would virtually have eliminated any recovery. Secondly, a decision to leave the patient for dead without even trying defibrillation could have had no reasonable basis whatever. These considerations reinforce the contention of Professor James

that the algorithms are predominantly guidelines for rescue services and that the anaesthetist in the theatre is generally better placed to decide what to do if the patient suffers a cardiac arrest.

[103] Having embarked on the defibrillation route, it was necessary after each unsuccessful shock to maintain circulation and to take some time in order to administer CPR and adrenaline and to let these measures have effect before trying the next defibrillation. It follows that up to the time of the third shock fault has not been proved as regards the second defendant's management of the resuscitation.

[104] As far as events after that are concerned, what has been said earlier in relation to the question of culpable ignorance and delay applies with equal force here. To repeat, it has not been shown that without the second defendant's culpable ignorance in respect of the Lohmeier, the patient's heart beat would have been restored any sooner than it was. The case against the second

defendant was therefore not proved.

THE ORDER AND COSTS

[105] Much as the plaintiffs deserve the sympathy of all for the awful fate that has befallen their son and the profound grief this must have caused them we conclude that the trial Judge was right to dismiss the claim. It follows that the appeal cannot succeed.

[106] On the matter of costs it remains to say that although the Judge said that he deplored the second defendant's untruthfulness in the few respects in which he was found by the trial court to have lied, his dishonesty on our findings went far beyond that. He deviously contrived a false and misleading operation record, he colluded with Sister Glaeser and Sister Montgomery to obtain first defendant's documentation concerning the Lohmeier and he knowingly gave evidence that was false in very numerous respects in an endeavour to eliminate propranolol as the cause of the cardiac arrest and impede a proper investigation

into that event. There is no place for such conduct in an honourable profession.

[107] Although the second defendant's counsel declared during the cross-examination of his client by the plaintiffs' counsel (on 19 May 1997, roughly half-way through the trial) that the defence based on cocaine toxicity depended in no way on there having been a period of normality, investigation of that issue took up a considerable amount of court time and the trial Judge devoted a substantial degree of attention to it before accepting (wrongly in our respectful view) the second defendant's evidence on the point. There can be little doubt that had the second defendant not advanced this false version and not given false evidence in the other respects mentioned above, and had he accepted at the stage of the Rule 37 discussions (if not earlier) that the time from the hypertensive crisis to arrest was as alleged by the plaintiffs and found by this Court, the length of the trial and the scope of the appeal would have been

materially reduced.

[108] Counsel for the plaintiffs did not, either when requesting leave to appeal or in their heads of argument in this Court, seek a special order for costs against the second defendant and only referred to the matter in reply in answer to a question from the Bench. We think that this is an appropriate case to consider whether such an order should be made as a mark of this Court's disapproval of the second defendant's dishonesty, and also by reason of the extent to which the duration of the proceedings was increased. The second defendant has not been heard on the matter of a special order and he must be afforded the opportunity to deal with it. It is also necessary to invite further submissions from the second defendant as to whether this judgment ought to be referred to the Health Professions Council.

[109] The order we make is as follows:

1. The appeal is dismissed as regards the issue of liability.

2. By reason of the terms of paragraph 3 below all questions of costs, both of trial and appeal, will stand over for later determination.

3. The second respondent is called upon to file written submissions on the question as to what costs order should be made by reason of the considerations discussed in this judgment and why an order should not be made referring the judgment to the Health Professions Council for such action as it may consider appropriate.

4. The other parties are at liberty also to file written submissions on the costs question raised in paragraph 3 above in so far as they have an interest in such question.

5. Submissions drawn pursuant to this order must, after mutual exchange of respective drafts between the parties (if applicable),

be filed with the Registrar of this Court within two (2) weeks of the date of this order.

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CT HOWIE
JUDGE OF APPEAL

_____

I G FARLAM
JUDGE OF APPEAL

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D CHETTY
ACTING JUDGE OF APPEAL