L M J MAMAYO — APPELLANT

and

THE STATE — RESPONDENT

Judgment by:
NESTADT, JA

Case No 633/89
CCC/

IN THE SUPREME COURT OF SOUTH AFRICA

(APPELLATE DIVISION)

In the matter between:

LUVUYO JUSTICE MTSHIZANA MAMAYO APPELLANT

and

THE STATE RESPONDENT

CORAM: HOEXTER, NESTADT et MILNE JJA

DATE HEARD: 19 NOVEMBER 1990

DATE DELIVERED: 26 NOVEMBER 1990

J U D G M E N T

NESTADT, JA:

At about 2 pm on 30 May 1985, appellant, armed with a knife, entered the farm-house of 84 year-old Herbert Daniels. The farm is situated about 15 kilometers from

East London. Daniels lived there alone. Appellant's motive was to steal money from him. On encountering Daniels, appellant attacked him. He stabbed him a number of times. He also bit off a portion of Daniels' left thumb. Just then some visitors arrived at the house. They raised the alarm. Appellant fled. Daniels was taken to hospital where he underwent treatment for his injuries. He remained there until 16 August 1985 when he died.

These events gave rise, subsequent to appellant's arrest on 18 June 1985, to his indictment on two counts, viz, murder and attempted robbery (with aggravating circumstances). The trial was heard by JENNETT J and assessors sitting in the East London Circuit Local Division. Appellant was convicted on both counts. No extenuating circumstances having been found, he was sentenced to death for the murder. The conviction of attempted robbery attracted a sentence of 15 years'

imprisonment. This appeal, which is brought with the leave of the trial judge, is against the conviction for murder and the death sentence.

The first and main issue, in relation to the appeal against the conviction, is whether the trial court's finding that appellant caused Daniels' death was correct. As has been indicated, there was an interval of some two and a half months between appellant's attack on him and his death. In these circumstances it is not surprising that in the court a quo the State presented detailed medical evidence concerning the injuries inflicted on deceased by appellant, the treatment deceased received and the cause of death. A number of doctors testified in this regard. In outline their evidence (the acceptability whereof is not in issue) was the following. Deceased sustained various injuries as a result of appellant's assault on him. One was an incised wound over the left eye which went through

the eyebrow and penetrated into the corner of the eye. A second was a superficial flesh wound to the left of the mouth. Thirdly, it was found that the tip of the left thumb had been amputated through the base of the nail. The most significant injury, however, was a large gaping wound at the base of the neck on the right hand side which transected the jugular vein. Deceased was admitted to the casualty section of the Frere Hospital. Though conscious, he was found to be in a shocked condition. His blood pressure was extremely low, namely, 60/50. Emergency treatment was given. But this did not suffice. It was decided that the wound to the neck required to be operated upon. This was to deal with a haematoma (an area of internal bleeding) that had formed there. Under local anaesthetic it was evacuated. Dr Zanewezyk performed the operation. Deceased's immediate post-operative course was stable. Two days later (ie on 1 June 1985), however,

whilst he was still in the intensive care unit, he developed a hemiplegia (ie a paralysis) on his left side. Thereafter his condition gradually but progressively deteriorated. He remained immobile. Pressure or bed sores formed. There were several episodes of pneumonia and urinary tract infection. According to the post-mortem report of Dr Wingreen the cause of death was "cerebral infarction". The evidence explained this to mean death of the brain.

Causation is often dealt with under the twin rubrics of factual and legal causation. In relation to the former, the "but for" or sine qua non test is applied. Legal causation, which serves to limit an accused's liability, requires that, in addition, his conduct be the decisive (or substantial, or proximate, or direct) cause of death; negatively put, there must have been no novus actus interveniens (Hunt: South African Criminal Law and Procedure, vol II, 2nd ed (by Milton), 345 seq; Snyman:

Strafreg, 2nd ed, 69 seq).

I turn to the issue of factual causation. As has been said, deceased died as a result of cerebral infarction. The State had to prove that but for appellant's attack on deceased, and more particularly the infliction of the neck injury, this condition would not have occurred. In order to determine whether such onus was discharged, it is convenient to adopt a two-fold enquiry. The first is whether it was established that already on 1 June 1985, deceased suffered a partial infarction of the brain due to the consequences of the neck injury. That he did then suffer such a cerebral infarction is clear. This is evidenced by the hemiplegia which developed. In the words of Dr Wingreen, "(it) indicates that he had death of ... portion of his brain". What led to this was the brain being deprived of oxygen, consequent upon the formation of a thrombosis or clot which

developed because of an inadequate blood supply to the brain. But what was the reason for the inadequate blood supply and thrombosis; is the only reasonable inference that they were due to the neck injury? The evidence refers to certain other possible causes. One is that the clot was the result of the operation itself. This, however, cannot avail appellant. The surgery was properly conducted and if it dislodged a clot this could not be avoided. There is therefore no question of the chain of causation being broken in this way. But difficulty does arise from the fact of deceased's advanced age, and that he was then suffering from atrophy of the brain and hardening of the arteries. This, it was argued on behalf of appellant, might have caused the thrombosis. Reliance was in this regard placed on the following evidence of Dr Unger (in whose care deceased was from after the operation until he died):

"Just to clarify a point that you made that the dislodging of the clot could have been caused by either the injury or the subsequent operation? --- Yes.

COURT: Is there anything else that could have caused it? --- Definitely, yes. There's a wide variety of possibilities, yes.

This is what one concerns me where one is dealing with a man of the age of the deceased who has, as you said, suffered from some hardening of the arteries. --- Yes, it could be a problem related to the arteries themselves. It could be a more remote problem in the body. The clot could originate anywhere. It could originate from the heart as well...

Doctor, are you saying that what you observed on the scan and what you subsequently found at the post-mortem examination could have been caused directly by the drop in blood pressure, not necessarily by an operational procedure? --- It's difficult to say what was the cause. It could have been multi-factorial...

You say it would appear that there are several possible contributory causes for the condition which you observed? --- Definitely so, yes. For that hemiplegia? --- Yes.

COURT: In the normal course - an elderly person suffering from hardening of the arteries, can they die of cerebral infarction? --- Yes."

These views notwithstanding, it seems to me that the weight of the evidence, together with the

probabilities, established that it was the injury to deceased's neck that put in train the events which culminated in the deceased suffering a cerebral infarction on 1 June. This is what the trial court (by implication) found and I think it was right. On a proper reading of the medical evidence, there is a firm factual foundation for it. The injury, described as a potentially fatal one, left a gaping wound. This led to a large amount of blood being lost and, most important, to the sudden, severe and relatively prolonged drop in deceased's blood pressure. Its level was described in the evidence as "very low" and "dangerous for life". It is true that prior to the operation it was restored to a more normal level but by this time, as Dr Wingreen said, "the damage had been done". By this was meant that the supply of blood and thus oxygen to the brain had been adversely affected. Dr Wingreen's explanation of the consequence of the loss of blood

pressure is important. He states:

> "With such a dangerous drop in blood pressure one has straight away the brain is deprived of oxygen and the state of anoxia develops. This results in the death of the brain, which might be selective for certain parts of the brain... I find that as a result of an unnatural drop in his blood pressure he sustained brain damage...
> I can only say that [the hemiplegia] is as a result of the hypotension - the drop in the blood pressure... [The] changes in the brain, can be ascribed to the state of low blood pressure."

And, dealing specifically with the clot, he said:

> "I don't think that one must emphasise the clotting story because what I emphasise here is the necrosis - the death of the brain which arises from the hypotension, whether that's due to associated clotting or not is of no real significance ... Doctor, with regard to the clot to which mention was made, is it then your position that in the scenario which you have identified the clot is a secondary result of the drop in blood pressure, rather than the cause of ...(Intervention)? --- Yes, M'Lord, that was certainly a secondary result."

Already on admission to hospital, deceased was blind in both eyes. To Dr Wingreen this was indicative of oxygen deprivation (to the nerve cells of the retina). So at an

early stage there were clinical signs (as I understand the evidence) of low blood pressure. Further evidence of this is that as he was being taken into theatre, he suffered a respiratory or cardiac arrest. According to the anaesthetist who then rushed to appellant's assistance, "he had a very, very slow pulse which is a sign of anoxia ... which is ... a lack of oxygen".

The cumulative effect of this evidence is, in my opinion, such as to give rise to only one reasonable inference, viz, that the hemiplegia was caused by the loss of blood pressure which in turn resulted from the injury. There is really nothing to refute it. The mere fact that deceased was aged and suffered from a diseased brain and arteries does not suffice. It is true that Dr. Wingreen thought that the fact of deceased's suffering from hardening of the arteries made him "very susceptible to ... oxygen and blood pressure changes". This was particularly

so in view of deceased's age. Dr Wingreen added:

> "Now my experience with old people and treating their blood pressures is that any reduction in blood pressure for any length of time is a very dangerous procedure. One gets certain changes which occur in the brain arising as a result of the anoxia which takes a period of hours to days to develop."

But these susceptibilities cannot operate in appellant's favour. He must take his victim as he finds him. In any event, it would be a most improbable coincidence that within about two days of appellant's assault on deceased, deceased would suffer a cerebal infarction from some cause independent of and unrelated to the injuries he sustained. There was, moreover, no evidence that he had in the past actually suffered from any consequences of his condition. On the contrary, it appears that he was a relatively fit and healthy man for his age. So the possibility that he sustained an independent, naturally caused thrombosis was a theoretical, speculative one and cannot legitimately

operate in appellant's favour.

To sum up so far, I agree with Mr Tyler for the State that the conclusion is unavoidable that in the absence of the attack by appellant, deceased would not have suffered a partial cerebral infarction some two days thereafter. Proceeding on this basis, I turn to the second enquiry, viz, whether, as the State contended, the infarction was correctly found to be the ultimate cause of death. There is ample evidence to support this conclusion. Dr Wingreen, in his testimony, confirmed his post-mortem finding that the cerebral infarction was the cause of death. This was despite the two and a half months interval that elapsed. Deceased's death was, nevertheless, "directly attributable to changes which occurred early on". A cerebral infarction does not necessarily result in immediate death; there could be "a delay from a period of minutes to a period of years". In

this case, deceased having suffered the cerebral infarction, "the pathological process (was) set for a deterioration without any hope of recovery whatsoever". The brain damage which he then sustained, being irreversible:

> "led to a process of infarction or death of that total part of the brain which got worse and took a number of - took a period of time, whether it took a period of days or months is of no significance from a pathological point of view because the brain was dying, one was getting reaction between the normal brain and the abnormal brain, a build-up of pressure, and final death as a result of the original cause, which was the necrosis, the infarction of the whole - not just a small portion of the brain, but a whole parieto-occipital cortex which is more than about 40% of a half of the brain."

Dr Wingreen's conclusion was that "the final cause of death must have been the precipitating death of the parieto-occipital region of the brain."

There can be no question but that on this evidence the cause of death was the cerebral infarction and thus the stab wound of the neck. On behalf of appellant,

however, Mr Lowe, on the basis of certain other evidence of Dr Wingreen, contested this. His submission was that the reasonable possibility of a supervening, independent thrombosis, attributable to natural causes, having caused deceased's death, had not been excluded. Reliance was placed on certain statements by Dr Wingreen which, it was said, constituted a concession to this effect. I quote:

> "COURT: I'm sorry, is that what you were saying, because you seemed to me to be saying because of his bedridden state and the fact of sepsis developing he would be more susceptible to a stroke. --- Yes, more susceptible to form a thrombosis.
> To form a thrombosis, but that doesn't necessarily mean that if he suffered a second stroke that it would necessarily - that it was necessarily attributable to that. It could still be attributable to natural causes. --- To natural causes.
> Possibly. --- Possibly.
> Yes. --- Yes."

Attention was also drawn to a further passage in his evidence, viz:

> "Yes, I don't think one can under-exaggerate or

> under-estimate the effects of this assault, but the question we have to decide is whether beyond reasonable doubt the death was precipitated by the assault, which you think it was? --- Well I would say that the assault very definitely contributed to his being in hospital which gave rise to the original infarction of the brain and which never ever changed. Whether a natural process of pathology then took over - this I am unable - this is the one point which I am totally at a loss."

In the result, so it was contended, a possible novus actus interveniens had not been negatived.

It cannot be gainsaid that the evidence referred to, if read in isolation, lends some support to the argument. When considered in context, however, and in the light of Dr Wingreen's evidence as a whole, I am satisfied that it does not and that the true effect of what he says is, as already indicated, that deceased's death was due to the cerebral infarction originally suffered. His opinion regarding a second thrombosis was on the specific assumption that there was such a thrombosis. But, as he

goes on to say:

> "I myself did not see any further thromboses in any other area of the brain excepting in that one particular part which was obvious... I found no evidence that (a second thrombosis) had in fact occurred - in other words in any other area of the brain."

Plainly, he is referring to the area of the original cerebral infarction. It is true that these statements (and others) seem to acknowledge the possibility of a second thrombosis in the same area. Thus he says "I cannot exclude the fact that there may well have been another little thrombosis - one can't see these things macroscopically". But on a proper reading of his evidence I do not think that this is so. He is still being asked to speculate and that is what he is doing. The possibility in question is therefore no more than a theoretical one. As such it can be left out of account.

The argument based on the passage in which there is reference to the possibility of a "natural process of

pathology (taking) over" and causing death must also be rejected. Dr Wingreen explains what he meant by this. He says:

> "Whether a natural process of pathology then arose - in other words man of 84 plus necrotic brain, unable to move, all the events which are consequential thereafter, this could be due to all factors like his age, his inability to cope with infections of his chest, his inability to cope with bladder infections, his inability to cope with immunity. These are all the features of the ageing process. We are not dealing with a young man with a necrotic brain. We are now dealing with an old man with a necrotic brain and natural pathology then takes over and will lead to his inevitable death... (I)t could be any pathology arising thereafter. His heart become incompetent - and I noted in the notes that he did have cardiac pathology, he did have emphysema. He had a prostatectomy but I noticed he was on a drain and he did have bladder irrigation."

None of these factors can be regarded as interrupting the chain of causation. The original effects of the cerebral infarction remained. This is apparent from Dr Wingreen's further explanation of the significance of the "natural

process of pathology". His evidence in this regard reads:

> "Now if we hypothetically think of the state at which he had been placed - think of what might have transpired thereafter, that process of pathology, as you call it, would the deceased have been able to resist - physically resist this natural process of pathology to the same extent that he would have been able to resist it had he been an active man - in other words not bedridden? --- No.
> COURT: He has weakened considerably. --- He has weakened very considerably. He has no reaction to immunity. He is unable to resist infections. He is unable to move. He is unable to cough and move about to prevent a terminal pneumonia. He is unable to resist an infection of his bladder. He is unable to react even to his cardiac failure which could develop."

As I understand the position therefore, insofar as a natural process of pathology may have precipitated deceased's death, it was itself brought about by the sequelae of the cerebral infarction which he suffered on about 1 June 1985. In other words, he died from causes which, far from being independent or abnormal, flowed from the original injury to the neck and which, but for such

injury, would not then have arisen. The natural process of pathology under discussion cannot therefore rank as a novus actus.

I have perhaps encroached on to the field of legal causation. But is was convenient to deal with it together with the issue of factual causation. For the reasons aforesaid, there is no warrant for disturbing the trial court's finding that both aspects were proved and that appellant was accordingly responsible for deceased's death.

The second issue raised by the appeal against the conviction is whether the trial court was justified in inferring that in assaulting deceased, appellant had the necessary mens rea for murder. The finding in this regard was based on dolus eventualis. In my view, it is unassailable. The nature and circumstances of the attack proclaim this. Deceased, being an elderly man, could

readily have been overpowered by far less lethal means than those actually employed. Instead, appellant attacked him with a knife with which he had previously armed himself and which, in the absence of any evidence by appellant, he must be taken to have intended to immediately use on encountering deceased. This is what happened. And he stabbed deceased three times in the area of the head and neck. These are particularly vulnerable parts of the body. The fact that he also bit off portion of deceased's thumb is a further indication of the ferocity of the attack. The inference that he subjectively foresaw the possibility of deceased's death is irresistible.

In the result, the appeal against the conviction must fail.

It remains to deal with the appeal against the death sentence. Since appellant's trial, the Criminal Law Amendment Act, 107 of 1990, has come into operation. It

is consequently necessary for us to decide whether, in our opinion, the death sentence is the only proper sentence. This must be done with due regard to the presence or absence of mitigating and aggravating factors. There are certainly aggravating factors present. Appellant's victim was an old, defenceless man. The attack on him took place in the privacy of his (isolated) home. One knows how unfortunately frequent this type of crime is. It was not an impulsive one. It was planned. This is evidenced by the fact that appellant had, a few days before, made enquiries about who deceased lived with. On the day in question, therefore, appellant went to deceased's place armed with a knife. As I have indicated, the assault was a savage and ruthless one. Appellant's callousness is shown by his state of mind when his purpose was frustrated. In a written confession to a magistrate he said: "My hart was seer omdat ek nie geld gekry het nie". Appellant is

not a first offender. During the period from 1977 to 1984 he was convicted of unlawful possession of dagga, housebreaking with intent to steal and theft and on two counts of theft.

On the other hand, there are certain mitigating factors. There is some evidence that appellant was accustomed to smoke dagga and abused liquor. As I have said, he was found guilty on the basis of dolus eventualis. The trial court's reasons in this regard contain the following passage:

> "As to the accused's intention when he inflicted the injuries to the deceased, we are not able to conclude on the evidence that the accused directly intended the death of the deceased. If he had, seems to us that he would and could, to put it colloquially, have finished the deceased off there and then."

Appellant's previous convictions are not in respect of any crimes of violence. Moreover, he was not directly sentenced to imprisonment without the option of a fine and

in consequence he may not in fact have served any period of imprisonment. Though aged 24 and therefore not a youth, he is still a comparatively young man. So, as Mr Tyler fairly conceded, this is not a case where it can be said that imprisonment is unlikely to have a rehabilitative effect on appellant. In all the circumstances, and though I regard the case as a borderline one, I have come to the conclusion that the death sentence is not the only suitable sentence. In the words of NICHOLAS AJA in S vs J 1989(1) SA 669(A) and 682 D, it is not "imperatively called for." It seems to me that the substitution of a period of imprisonment will more appropriately achieve the objects of sentence. The period of imprisonment will, however, have to be a lengthy one. The interests of society demand this. In fixing the period of imprisonment, account will be taken of the fact that appellant was an awaiting trial prisoner for some two years.

The appeal against the conviction for murder is dismissed. The death sentence is, however, set aside. In its place there is substituted a sentence of imprisonment of 18 years. The sentence of 15 years' imprisonment for attempted robbery is to run concurrently with it.

NESTADT, JA

HOEXTER, JA)
) CONCUR
MILNE, JA)