

Illawarra Area Health Service

The Wollongong Hospital

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Your Ref

Our Ret MPR/PH

26th February, 1986.

CORONER'S REPORT ON WILLIAM ROONEY

My name is Mason Phillip Ramsay and I hold the degrees of M.B., Ch.B. (University of Otago) and am a F.F.A.R.C.S., employed as Director of Intensive Care by the Illawarra Area Health Service. I first met Mr. William Rooney at about 10 a.m. on the morning of 14.2.86. He was in the Casualty Department of The Wollongong Hospital and I was told that he had been found by the Ambulancemen at the bottom of a series of steps at the back of Globe Lane. He was hypothermic, was unconscious when they found him and was still unconscious when I saw him. He was making some inappropriate noises and flexing to painful stimuli. I was shown x-rays of a skull which showed fractures in the occipital bone extending into the base of the skull and there was blood coming from his left ear. It was decided that he should have a C.A.T. Scan and to perform this I elected to paralyse, intubate and electively ventilate the man. C.A.T. Scan was performed without contrast and was reported by Dr. Herbert Ho. The report was "a plain C.T. head study was performed. Multiple fractures are seen. There is, however, no definite evidence of depression of the fragments. There is a fracture seen through the left petrous temporal bone, another one at the right occipital region. There is another fracture in the left parietal region just before the temporal area. The right posterior parietal region is also seen with a fracture line running through it.

There is extensive subarachnoid haemorrhage. There is a little deviation of the mid line structure to the left side. Cerebral contusion is noted in both frontal lobes with multiple small areas of haemorrhages. There is a thin but rather extensive extra cerebral collection in the right parietal region." I discussed these findings with Dr. Peter Moloney, Neurosurgeon, who was operating at the Illawarra Private Hospital. He decided he did not need any emergency surgery performed, however, it would be reasonable to monitor his intracranal pressure. At about 5 o'clock Dr. Moloney and Dr. McDowell inserted an

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intracranial monitor via a burr hole in the right parietal region. He was maintained on a ventilator and lightly sedated for the next four days and his intracranial pressures were basically stable. On the morning of the 19th between about 7 and 8 a.m. his responses decreased and when I saw him about 8:30 a.m. he was barely responding to painful stimuli on the left with flexion. His pupils were still slightly reacting. He was taken for a second C.A.T. Scan to eliminate the possibility of a surgically correctable lesion. The second C.A.T. Scan was essentially unchanged from the first. During the C.A.T. Scan he had changes of blood pressure, pupils and pulse consistent with deteriorating brain stem function. Dr. Moloney and I further discussed the patient's prognosis at about 2 p.m. and felt that he was probably brain dead. We decided that a relatively reasonable course was to offer no further resuscitation and felt that the patient would probably die overnight if left on a ventilator with no further support. However, the following morning (20.2.86.), although he remained hypotensive, his heart was still functioning and he was hypothermic. He started to deteriorate between 7 and and 8 a.m. His temperature was brought back to within acceptable limits and his carbon dioxide levels were adjusted to the necessary criterion for brain death evaluation and he was formally evaluated as brain dead by Dr. David Serisier and by myself between 1 p.m. and 2:30 p.m. on 20.2.86. At the completion of the second brain death evaluation the ventilator support was terminated. His heart ceased beating at about 14:35 hours.

M. P. RAMSAY, M.B., Ch.B., F.R.A.R.C.S., Director of Intensive Care, I.A.H.S.