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BRAIN DEATH AND PROLONGED STATES OF IMPAIRED RESPONSIVENESS

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INTRODUCTION

The related issues of brain death and prolonged states of impaired consciousness were recently addressed in the significant decision of *Lovato v. District Court*,[1] delivered by Justice Groves of the Supreme Court of Colorado, on October 15, 1979. This article will discuss these and related questions from the perspective of a clinical neurologist active in a university teaching hospital who confronts these issues very frequently and is required to render an operational decision for particular patients. The author wishes to caution readers that many viewpoints in this work are personal and encourage those who may wish to explore these topics further to consult the published proceedings of the New York Academy of Sciences meeting held in November 1978,[2] which is an excellent source for medical, legal, and ethical information.

I. BRAIN DEATH

For clarity, certain terms will be defined now, while others will be explained as they occur later in the discussion. The brain stem is composed of the midbrain (mesencephalon), pons, and medulla. It contains nerve cell groups and pathways that are intimately involved with basic cardiac and respiratory functions, with reflex activities such as eye movements and responses to light and other stimuli, and with those reactions to the environment that are understood by such terms as responsiveness, awareness, or consciousness (though the latter term has philosophical implications that are beyond the scope of this article). Spinal reflexes are reactions that depend only on the functional integrity of the spinal cord and hence can exist in the absence of a functioning brain. The term death is equated to an irreversible loss of function, either in part or whole, of an organ or of the human organism itself. Cerebral death refers specifically to death of the cerebral hemispheres but not of the brain stem, and patients so afflicted may remain in a prolonged state of impaired responsiveness. Clinically, this condition usually equates to the persistent vegetative state, about which more will be said later. An important distinction is that cerebral death is not a synonym for brain death. Brain death, medically and legally, implies irreversible cessation of function of the whole brain, which includes the cerebral hemispheres and the brain stem. The French term, coma dépassé, is an equivalent term

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1. Lovato v. District Court, 601 P.2d 1072 (Colo. 1979).

2. *See generally Brain Death: Interrelated Medical and Social Issues*, 315 ANNALS N.Y. ACAD. SCI. 1-454 (J. Korein ed. 1978).

which encompasses not only absence of clinical function but absence of electrophysiologic activity and cerebral blood flow as well.[3]

In *Lovato*, there was never any doubt about the presence of brain death in the seventeen-month-old child as determined by the attending physician, a consulting neurologist, and a court-appointed neurologist. The issue was whether in Colorado the death of an individual should be determined by the standards of past centuries, or whether contemporary standards could be applied despite the absence of legislative statutes setting forth the criteria for such a determination. Did society in general, and physicians in particular, have to abide by the cardio-respiratory description of death as expressed in Black's Law Dictionary—"A total stoppage of the circulation of the blood and a cessation of the animal and vital functions consequent thereon, such as respiration, pulsation, etc."[4]—or could an additional, not a substitute, definition apply in some instances? Justice Groves announced that the court was adopting as a rule, "until otherwise changed legislatively or judicially," the provisions of the Uniform Brain Death Act. This act was accepted in August 1978 by the National Conference of Commissioners on Uniform State Laws. Its provisions state: "For legal and medical purposes, an individual with irreversible cessation of all functioning of the brain, including the brain stem, is dead. Determinations of death under this act shall be made in accordance with reasonable medical standards."[5]

Inasmuch as physicians in Colorado may now follow "reasonable medical standards" in determining when the brain is dead, it becomes necessary to define, with as much precision as possible, just what these standards might be. While perhaps having some merit in our current litigious society, Sir Peter Medawar's criterion of brain death—that a man is legally dead "when he has undergone irreversible changes of the type that make it impossible for him to seek to litigate"[6]—may not be universally applicable. A vague or poorly drawn set of medical criteria might err in two ways. Life support might be abandoned prematurely in some instances or kept up for an unnecessarily long time in others. Hence, I will discuss the evolution of relevant medical thinking in some detail and will try to define a set of guidelines that all physicians might use.

In the twelve years since the landmark 1968 report of the Ad Hoc Committee of the Harvard Medical School to Examine the Definition of Brain Death,[7] physicians, lawyers, and ethicists have worked hard to develop standards of such accuracy that any possible errors would favor the preservation rather than the cessation of life. These endeavors have been stimulated by the development in recent decades of machines, such as respirators, and therapeutic techniques which allow the maintenance of cardiac and respiratory function for a long time, despite natural failure of heart and lungs. In addi-

3. Mollaret & Goulon, *Le Coma Dépassé*, 101 REVUE NEUROLOGIQUE 3 (1959).
4. BLACK'S LAW DICTIONARY 488 (rev. 4th ed. 1968).
5. Cranford, *Uniform Brain Death Act*, 29 NEUROLOGY 417 (1979).
6. MEDAWAR, THE UNIQUENESS OF THE INDIVIDUAL (1957).
7. Ad Hoc Committee of the Harvard Medical School to Examine the Definition of Brain Death, *A Definition of Irreversible Coma*, 205 J.A.M.A. 337 (1968) [hereinafter cited as *Irreversible Coma*].

tion, the realization by society that its fiscal resources were not infinite provided an economic impetus, often the most compelling of all reasons, to address further this most complex problem.

What the Harvard Committee did was to offer a definition of irreversible coma which they called a new criterion of death. Such a state was defined as one in which "irreversible cardiac arrest will inevitably follow regardless of the maintenance of all resuscitative measures."[8] Subsequent studies have confirmed that the heart will stop beating in an adult within seven days despite any therapy once brain death has occurred.[9] How long the heart of an infant or a very young child may continue to beat following brain death is somewhat less certain. The hearts of most brain dead children will stop beating within two to three weeks.[10] The longest duration of cardiac activity that I have been able to find in the medical literature in a child with clinical findings of brain death and absent cerebral circulation as established by radiographic techniques was thirty-two days.[11] Justice Groves accepted the view that the seventeen-month-old Lovato child would spontaneously develop cardiac arrest "within a month or so" of disconnection from life support systems, and this judgment appears sound. Hence, when we discuss the abandonment of such systems in the case of a brain dead person, we are talking about a step that moves up the inevitable cessation of the heart by only a few days in adults and a somewhat longer period in young children. It is when we consider true states of irreversible impairment of responsiveness or cognition, which differ from brain death and which are exemplified by the condition of Karen Quinlan, or make attempts to predict outcome for the brain and the entire organism within a few days of a catastrophic medical event that we move onto less certain ground.

The 1968 Harvard Committee[12] adopted a set of very stringent medical criteria to identify those patients who had no discernible brain and spinal cord activity, and who despite a beating heart could never return to a feeling, consciously responsive existence. The patient had to be unreceptive of, and unresponsive to, any stimulus including pain. Spontaneous respirations had to be absent for at least three minutes. All spinal reflexes (such as the knee jerk) and all cephalic reflexes (such as pupillary responses to light, the corneal blink reflex, and ocular response to ice water instilled into the external ear canal) had to be absent. An electroencephalogram (EEG), to be repeated at a twenty-four hour interval when the clinical tests were also to be repeated, could be utilized but was not considered to be mandatory. Since central nervous system depressant drugs, such as barbiturates, and hypothermia could introduce possible errors into the assessment, these factors had to be absent. These standards were felt to be consistent with the view stated by Pope Pius XII that "it remains for the doctor . . . to give a clear

8. Korein, *The Problem of Brain Death: Development and History*, 315 ANNALS N.Y. ACAD. SCI. 19 (1978).

9. *Id.*

10. Ingvar, Brun, Johansson & Samuelsson, *Survival After Severe Cerebral Anoxia with Destruction of the Cerebral Cortex: The Apallic Syndrome*, 315 ANNALS N.Y. ACAD. SCI. 184 (1978).

11. *Id.* at 211-14.

12. *Irreversible Coma*, *supra* note 7.

and precise definition of 'death' and the 'moment of death' of a patient who passes away in a state of unconsciousness."[13]

The widespread application of these Harvard standards led quickly to a realization that irreversible coma and brain death were not always synonymous, that some of the given criteria of brain death were not completely accurate, and that modifications of these guidelines to meet particular circumstances were desirable. For example, from approximately one-half of "brain dead" individuals, spinal reflexes may be elicited.[14] A twenty-four hour delay in determining death may jeopardize the viability of organs, such as the kidney, which might be donated to needy recipients. Consequently, alternatives to the Harvard criteria were put forth from Minnesota,[15] Japan,[16] Sweden,[17] and from a collaborative study in this country supported by the National Institute of Neurological Diseases and Stroke.[18] In addition, much investigation has been devoted to rapid laboratory tests that might possibly be infallible arbiters of brain death, such as the use of cerebral angiography[19] and the injection of radioactive tracers as indicators of cerebral blood flow.[20]

With the results of all these studies and investigations in mind, I would offer the following as "reasonable medical standards" for the diagnosis of brain death.

1. *The decision about brain death is ultimately a clinical one*.[21] In most instances, no ambiguity exists in declaring the presence and time of death by traditional cardio-respiratory and/or neurological criteria. Laboratory tests are ancillary procedures that are not required to establish brain death, but they are useful in some instances to resolve uncertainties. These tests should not be regarded as a substitute for the clinical criteria outlined below or for the critical degree of judgment required in interpreting these criteria.

2. *It is essential that all historical information in a case be evaluated and that appropriate diagnostic procedures be carried out, so as to define as specifically as possible the cause or causes of the patient's condition*. In some instances, the use of the CT scanner, the EEG, or other laboratory aids, if available, may be of assistance in identifying the specific diagnosis. All therapeutic procedures appropriate

13. Address by Pope Pius XII, The Prolongation of Life, 4 THE POPE SPEAKS 393, 396 (Nov. 24, 1957).

14. Allen & Burkholder, *Clinical Criteria of Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 70 (1978).

15. Mohandas & Chou, *Brain Death: A Clinical and Pathological Study*, 35 J. NEUROSURG. 211 (1971).

16. Address to the Fifth International Congress of Neurological Surgery (Oct. 7-13, 1973) (paper entitled Clinical Study of Brain Death, by K. Ueki, K. Takeuchi & K. Katsurada).

17. Ingvar & Widen, *Brain Death: Summary of a Symposium*, 69 LAKARTIDNINGEN 3804 (1972).

18. Bickford, *An Appraisal of the Criteria of Cerebral Death: A Summary Statement, A Collaborative Study*, 237 J.A.M.A. 982 (1977); Molinari, *Review of Clinical Criteria of Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 62 (1978).

19. Kricheff, *Angiographic Findings in Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 168 (1978).

20. Braunstein, Korein, Kricheff & Lieberman, *Evaluation of the Critical Deficit of Cerebral Circulation Using Radioactive Tracers (Bolus Technique)*, 315 ANNALS N.Y. ACAD. SCI. 143 (1978) [hereinafter cited as Braunstein].

21. Ad Hoc Colorado General Hospital Committee, Procedure for Brain Death Decision-Making for Patients Whose Organs May Be Transplanted (Aug. 27, 1979) (unpublished).

to the patient's condition also should have been instituted. The details of what is therapeutically appropriate should be determined by the attending physician, in consultation with responsible family members. It is obvious that if the diagnosis of a potentially reversible medical condition is made, therapeutic efforts would be exerted to the maximum degree, whereas if an irreversible event, such as massive trauma to the head, had occurred such efforts might be minimal.

3. *The patient must be in coma and unresponsive in any purposeful way to any stimulus, such as severe pain*. The emphasis here is on the word purposeful, inasmuch as experience has shown that in a few brain dead patients reflex flexor spasms (withdrawal movements) of the legs or reflex decerebrate or decorticate movements may be induced by some stimuli.[22] Not all agree, however, that the presence of spontaneous or stimulus-induced decorticate or decerebrate movements is compatible with a diagnosis of brain death.[23] The decerebrate movement consists of extensor thrusting of arms and legs, while the decorticate movement involves arm flexion and leg extension. Spontaneous motor movements usually are absent in brain dead individuals, but may be present rarely as isolated jerks of a limb.[24]

4. *Spontaneous respiration must be absent and no efforts must be made by the patient to trigger a respirator*. Such a loss of respiration is called apnea. While initially it was thought that testing for apnea would be simple, it has been realized that false observations may occur unless precautions are taken. In addition, an improperly performed test in a patient who is not brain dead may further injure the brain.

Two physiological stimuli basic to reflex respiration are an increase in the carbon dioxide (CO2) content or a decrease of the oxygen content in the blood. Concern has been expressed about the possibility that a patient who had been hyperventilated on a respirator and hence depleted of CO2, while at the same time a high oxygen concentration had been inspired, might have both respiratory drives blunted.[25] In such a situation, when the respirator is disconnected, apnea in a patient not truly brain dead might persist longer than the 3-minute test suggested by the Harvard criteria,[26] or longer even than the 15-minute test suggested by a Northwestern University study.[27] This result would be due to a slow build up of CO2 from a very low blood level and a slow fall of oxygen from a very high blood level. In two actual instances, spontaneous ventilation returned in patients with flat EEG's (no electrical activity noted) after 5 minutes, while in one instance it returned after 9 minutes.[28] An additional problem with this testing for ap-

22. Allen & Burkholder, *supra* note 14, at 76.
23. *See* Sweet, *Brain Death*, 299 NEW ENG. J. MED. 410 (1978).
24. Allen & Burkholder, *supra* note 14, at 76.
25. Posner, *Coma and Other States of Consciousness: The Differential Diagnosis of Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 215 (1978).
26. *Irreversible Coma*, *supra* note 7.
27. Schafer & Caronna, *Duration of Apnea Needed to Confirm Brain Death*, 28 NEUROLOGY 661 (1978).
28. Milhaud, Riboulot & Gayet, *Disconnecting Tests and Oxygen Uptake in the Diagnosis of Total Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 241 (1978).

nea is that a patient who is not brain dead might be injured further by the hypoxia induced by the disconnection of the respirator.

To avoid these problems, simple precautions must be instituted. One test consists of artifically ventilating the patient at a normal rate of 20 breaths/minute with a flow of 7 liters of 100% oxygen/minute for at least 15-60 minutes to wash out 50% or more of the total body nitrogen.[29] This procedure greatly elevates the amount of oxygen in the blood, while maintaining a normal CO2 level. The respirator is then turned off while either a fine tracheal catheter or an endotracheal tube is used to continuously introduce 100% oxygen at a rate in adults of 4-6 liters/minute.[30] The test may be continued for 10 to 15 minutes, during which time the arterial CO2 pressure (which is the most potent stimulator of breathing) will rise to a level which should trigger respiration from an intact respiratory center in the brain stem. During the test period, the arterial oxygen pressure will not fall to hypoxic levels, and no additional brain damage will be sustained. Facilities for the measurement of arterial blood gases and pH are available in almost every modern hospital today. Such measurements should be made before and/or after the disconnection test to provide additional data for its validation. If the pressure of arterial CO2 prior to the test is at least 30 mm Hg, 10 minutes of apnea will always raise it above 60 mm Hg, the minimal level needed before one can say with certainty that respiration is absent.[31] Unless one knows the arterial CO2 pressure at either the start or the termination of the test, there is no way to be certain that the test has been sufficiently provocative with any time period utilized.

5. *Certain cephalic reflexes (light, corneal, oculovestibular, and oculocephalic) must be absent*. Cephalic reflexes traverse a variety of pathways through the brain stem, and thus their presence or absence is of considerable importance in determining whether that structure is intact. Some cephalic reflexes are light, corneal, oculocephalic, oculovestibular, audio-ocular, snout, pharyngeal, swallow, cough, and jaw. Studies have established that the absence of the pupillary light reflex (not necessarily associated with the presence of dilated pupils), oculovestibular reflex (eye movement in relation to ice water in the ear), oculocephalic reflex (eye movement in response to rapid head turning) and corneal reflex (blink) have the greatest degree of correlation with brain death.[32] Only rarely are they present in patients who are apneic, comatose, and without electroencephalographic activity for more than twenty-four hours, and then only transiently. Some of the other cephalic reflexes, such as the audio-ocular, are too sensitive, since they disappear early in patients who are not brain dead. Others, such as the snout and jaw reflexes, are too insensitive and may persist in cases of presumptive brain death.[33]

6. *The absence of spinal reflex activity is not necessary for a diagnosis of brain death*. Such reflexes, particularly in the arms, may be present or absent at

29. *Id*. at 243.
30. Milhaud, Riboulot & Gayet, *supra* note 28; Posner, *supra* note 25, at 217.
31. Schafer & Caronna, *supra* note 27, at 666.
32. Allen & Burkholder, *supra* note 14, at 94.
33. *Id*.

different times following the occurrence of brain death, and, hence, are of no value in the documentation of this entity.[34]

7. *Significant hypothermia must be absent*. This condition may mask other clinical signs, and, consequently at the time of testing, the patient's rectal temperature should be no lower than 97-98 degrees F. The temperature is usually normal to elevated in most brain dead individuals but may fall slightly in some. One study ascertained that approximately 17% of brain dead patients had temperatures below 95 degrees F. and only 2/114 had temperatures below 90 degrees F.[35] Warming of such patients prior to testing would be required.

8. *Drug intoxication must be absent*. Inasmuch as drugs may depress respiration and many of the reflex responses noted above, as well as producing coma, a blood and urine screen for sedatives, narcotics, psychoactive drugs, and alcohol should be obtained, if possible. Where such tests are not possible, a careful inquiry must be made with regard to these agents. The use of deliberately induced barbiturate coma as therapy for certain severe brain injuries and diseases makes it mandatory that blood levels of the drugs used be known before tests for brain death can be considered valid.

9. *The EEG is an aid in the diagnosis of brain death, but its use is not mandatory*. British[36] and American studies[37] of brain death have made this point, but in actual practice the use of the EEG as an ancillary aid is very frequent. It is of particular value when traumatic injury to the head and face makes testing of cephalic reflexes difficult. Unfortunately, and possibly as a way of abdicating personal responsibility, many physicians seem to desire to use this study in place of, rather than in addition to, the clinical criteria listed above. If the test is easily available, if it is done properly for at least 30 minutes by knowledgeable technicians in conformity with the guidelines for recording put forward by the American EEG Society,[38] if it is evaluated by physicians conversant with the possible technical pitfalls of the study and also conversant with the clinical problem at hand,[39] then the test is one which can buttress the clinical impression of brain death and further guard against the possibility of error.

These are several reasons why the EEG should not be used as the sole basis for decision-making. "In the state of brain death, the EEG is always silent; however, ECS [electrocerebral silence] does not always mean brain death."[40] For example, just as drug intoxication and hypothermia may

34. *Id.*

35. *Id.* at 72.

36. *Diagnosis of Brain Death*, 2 LANCET 1069 (1976) [hereinafter cited as *Diagnosis of Brain Death*].

37. Beecher, *After the "Definition of Irreversible Coma,"* 281 NEW ENG. J. MED. 1070 (1969).

38. AMERICAN ELECTROENCEPHALOGRAPHIC SOCIETY, MINIMUM TECHNICAL STANDARDS FOR EEG RECORDING IN SUSPECTED CEREBRAL DEATH (1976).

39. Report of the Ad Hoc Committee of the American Electroencephalographic Society on EEG Criteria for Determination of Cerebral Death, *Cerebral Death and the Electroencephalogram*, 209 J.A.M.A. 1505 (1969).

40. Bennett, *The EEG in Determination of Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 110 (1978).

mask clinical signs of life, these conditions may also cause ECS.[41] In the case of drugs, 24 hours of ECS has been followed occasionally by full recovery. In man, temperatures under 29 degrees C (84.2 degrees F) seem to be necessary to produce significant change in the EEG, but ECS does not likely occur until body temperatures go below 20 degrees C (68 degrees F). Recording of ECS without drug intoxication or hypothermia with subsequent recovery of some electrical activity in the brain, and in a few instances clinical recovery, has been reported. Such cases are very rare and the duration in hours (2 to 8?) of clear ECS which can precede brain and clinical recovery is not known.[42] ECS, however, when recorded in a setting consistent with the clinical criteria of brain death noted above is strong, presumptive, and additional evidence of brain death.

The EEG ordinarily is recorded from scalp electrodes, and, thus, it is possible to have ECS occur from brains that actually have existing deep subcortical electrical activity, both spontaneous and evoked.[43] Such a finding would suggest that the brain stem is not dead, and, hence, such patients would not be legally brain dead. Cases of ECS associated with clinical signs of brain stem function, though rare, have been reported.[44] Such cases might imply that the neocortex (the gray matter of most of the cerebral hemispheres) was dead while the brain stem was alive and raise philosophical questions for the present and legal problems for the future that are beyond the scope of this discussion. It should be apparent, therefore, that sole reliance on electrically silent EEG's to diagnose brain death would be improper. Even more rarely, it seems possible that the combination of a dead brain stem and some cortical electrical activity may occur, again emphasizing the need for clinical judgment to be the final arbiter of brain death.

10. *Additional laboratory tests may be used to provide further data about brain death, but must be correlated with clinical findings*. The evaluation of methods to rapidly and accurately determine the absence of cerebral circulation, a condition incompatible with recovery of electrical activity or clinical function, is under current investigation.[45] A bedside bolus technique using radioactive tracers has been advocated, especially in cases in which drug intoxication may be suspected.[46] It is sometimes difficult, and often time consuming, to clarify the question of drug intoxication, especially when the nature of the drug in question is unknown. In addition, drug intoxication may co-exist with other causes of coma, and, hence, be overlooked. Such a technique may, therefore, prove to be of value. Cerebral angiography,[47] brain scanning techniques,[48] pharmacologic tests (such as one using atropine to deter-

41. *See* Hughes, *Limitations of the EEG in Coma and Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 121 (1978).
42. *Id.* at 124, 126.
43. *Id.* at 127.
44. *Id.* at 129.
45. Walker, *Ancillary Studies in the Diagnosis of Brain Death*, 315 ANNALS N.Y. ACAD. SCI. 228 (1978).
46. Braunstein, *supra* note 20.
47. Kricheff, *supra* note 19.
48. Goodman, *Discussion*, 315 ANNALS N.Y. ACAD. SCI. 259 (1978).

mine viability of brain stem centers that accelerate cardiac rate),[49] and blood gas studies[50] also have been evaluated as measures of cerebral circulation. They may be particularly useful in infants and young children for whom the length of possible cardiac activity in a brain dead state may be more lengthy than that in adults. The current lack of availability to physicians in many hospitals of some of these tests and the lack of extensive validation for others, however, would put them outside the pale of "reasonable medical standards," which should be standards that can be employed readily by the non-neurologist.

11. *With few exceptions, at least six to twelve hours should elapse following the onset of apnea and coma before the final determination of brain death*. A critical question relates to the rapidity with which brain death can be declared, especially in cases in which organ transplantation is contemplated. By contrast with the Harvard criteria,[51] some recent studies have advised waiting only six hours after the onset of coma and apnea,[52] while others advocate waiting at least twelve hours.[53] In those instances where the specific time of onset of the apnea is not known, it has been recommended that at least six to twelve hours pass between two formal evaluations of the clinical and electroencephalographic criteria of brain death.[54] The majority of Colorado neurologists polled recently felt that twelve hours should be the minimum time of observation.[55] It is important to emphasize that a second complete clinical examination for brain death should be performed after the minimum six to twelve hour time period, in order not to overlook the slightest sign of genuine brain activity.

In a few special circumstances, a certain degree of leeway for judgment would also seem practical.[56] When a patient has sustained massive, obviously fatal head trauma, such as from a large caliber gunshot wound through the head, the time interval for observation may be shortened appropriately. By contrast, in other instances it may be necessary to observe the patient for much longer than twelve hours before outcome can be predicted clearly.

12. *All the examinations, both laboratory and clinical, should be documented in the chart not only by date, but by time as well*. The actual time of death should be documented clearly, because of potentially important legal considerations.

13. *No physician engaged in the process of procuring or transplanting organs from the patient involved should be a party to the determination of brain death*. This stricture should be scrupulously observed for obvious reasons.

Adhering strictly to these criteria, which I believe are reasonable and

49. Ouaknine, *Cardiac and Metabolic Alternations in Brain Death: Discussion Paper*, 315 ANNALS N.Y. ACAD. SCI. 252 (1978).

50. Walker, *supra* note 45, at 229.

51. *Irreversible Coma*, *supra* note 7.

52. Bickford, *supra* note 18, at 985.

53. Mohandas & Chou, *supra* note 15, at 212.

54. Suter & Brush, *Clinical Problems of Brain Death and Coma in Intensive Care Units*, 315 ANNALS N.Y. ACAD. SCI. 398 (1978).

55. Interview with E.C. Hutchins, President of Colorado Society of Neurologists (1980).

56. *Diagnosis of Brain Death*, *supra* note 36, at 1070.

which can be carried out by any physician without unduly specialized or costly help, brain death can justly be equated to legal death without fear of error. As Posner has indicated, "there is no differential diagnosis of brain death since the criteria have been so drawn that all patients whose brains are not dead are eliminated from consideration."[57]

II. PROLONGED STATES OF IMPAIRED RESPONSIVENESS

The remainder of this paper will attempt to grapple with issues that are much more difficult than those that relate to brain death. This is because they are less well defined medically, and, consequently, have scarcely been approached legislatively or judicially.[58] In a long footnote, Justice Groves avoided addressing the issue of irreversible coma, inasmuch as it was not applicable to the *Lovato* case.[59] He correctly pointed out that irreversible coma is not synonymous with either death or brain death. The same is true for a number of other states, and, hence, our discussion must examine additional conditions which do not meet the medical definition of true coma (a sleep-like state of eyes-closed, unarousable unresponsiveness, without evident psychological awareness of self or environment).

Patients who survive for long periods in states of impaired responsiveness to their environment sometimes may be thought to be in coma, while others clearly are not. Initially, such patients have stormy courses for days or weeks during which, for a time, brain stem functions may appear to be lost. They then stabilize and persist as described below. A variety of terms such as akinetic mutism, coma vigile, apallic syndrome, dyspallic syndrome, neocortical death, persistent vegetative state, and the locked-in syndrome have been used by authors in various countries in an attempt to bring semantic precision to these complex conditions. None of these labels precisely fit every case, and, hence, a knowledge of each is required to understand why the legal issues surrounding this area are not likely to be resolved for quite some time. Probably the most common nontraumatic cause for these conditions is cerebral anoxia or hypoxia (a complete absence or deficiency of oxygenation of the brain). Events such as sudden heart attacks with cardiac arrest, intoxication with carbon monoxide, hanging, or drowning would be common examples of causation of cerebral anoxia. Other frequent causes of vegetative states are strokes, failure of the liver or other vital organs, pressure sequelae from tumors, or infections.

The number of patients in these terrible conditions appears to be increasing, probably as a result of wide-spread use of resuscitative techniques by both professional and lay people. Fortunately for some and unfortunately for others, emergency resuscitation is attempted for just about any individual who collapses under almost any circumstance. There is no question that many lives are saved in this way and that full recovery of the patient often ensues. An unfortunate by-product, however, is that some brains

57. Posner, *supra* note 25, at 216.

58. Schneck, *Brain Death, the Persistent Vegetative State and Medical Decision-Making*, 11 INT'L SOC. BARRISTERS 201 (1976).

59. Lovato v. District Court, 601 P.2d 1072, 1076 n.6 (Colo. 1979).

are so damaged by the catastrophic event which precipitated the resuscitative action that although the individual survives, there may be permanent loss of cognition and responsiveness. Nerve cells in the brain begin to die after approximately five minutes of oxygen deprivation, and no mechanism exists for their replacement. The cerebral cortex is much more sensitive to oxygen deprivation than is the brain stem, and, hence, survival of the organism with loss of those cognitive functions dependent on the cortex is no surprise. Ideally, if one knew just how long the brain had been deprived of oxygen before a resuscitation was attempted, one might make a judgment as to whether to proceed. Only rarely is this time known accurately (cardiac arrest during an operation would be one example). Likewise, the efficacy of a resuscitative attempt in providing well oxygenated blood to the brain is rarely measured. It seems likely that eager but unskilled volunteers with little practical experience in resuscitation may not be as effective physiologically as might be desired.

The term akinetic mutism, originally coined in England in 1941,[60] describes patients who, while often asleep, at times appear awake; who while not paralyzed, are immobile except for occasional random, slow, and inconstant motor responses to auditory, painful, and other stimuli; who, while not always silent, have no significant spontaneous verbal communications; who rarely follow objects or people with their eyes; and who often give the impression that in some way they are aware of their environment, though not responsive to it. Thus, brain stem functions obviously are intact in these patients. If electroencephalography is performed, the record is usually diffusely slow, but is occasionally near normal unlike the ECS of a brain dead person. It is apparent that these individuals may not always be akinetic or mute, and some have lived in this tragic condition for weeks, months, or years. Rare patients with reversible causes for this state, such as resectable tumors, have recovered with no memory of the time spent in it. A French term, coma vigile, is essentially a synonym for akinetic mutism.

A German author, in 1940, first used the term apallic syndrome.[61] These patients have a complete loss of speech, voluntary motor activity, and emotional and other reactions to their environment, but they retain brain stem functions including respiration.[62] They may respond to some stimuli with involuntary movements and changes in respiration, and they may exhibit chewing and swallowing movements. The EEG is usually quite depressed and sometimes exhibits ECS. Individuals may survive a long time in this condition, with one reported patient existing 17 years.[63] Cerebral blood flow is reduced to about 20% of normal. Incomplete syndromes in terms of their clinical, EEG, and cerebral metabolic features have been called dyspallic. The major pathological change is severe anoxic damage to the cerebral cortical nerve cells while the brain stem remains relatively preserved. Ne-

60. Cairns, Oldfield, Pennybacker & Whitteridge, *Akinetic Mutism With An Epidermoid Cyst of the 3rd Ventricle*, 64 BRAIN 273 (1941).

61. Kretschmer, *Das Apallische Syndrom*, 169 Z. GESAMTE NEUROLOGY PSYCHIATRY 576 (1940).

62. Ingvar, Brun, Johansson & Samuelsson, *supra* note 10, at 184.

63. *Id.* at 203.

ocortical death, an English term,[64] also refers to widespread, irreversible damage to the gray matter of the cerebral hemispheres, while the intact brain stem is able to contribute to prolonged survival of the patient.

The term persistent vegetative state was coined in 1972,[65] and has rapidly gained acceptance in this country and in England. Patients so named have a complete loss of cognitive function and are, in lay terminology, "vegetables." This state differs from coma in that at times the eyes are open, and sleep-wake cycles are present. Spontaneous and apparently reflex eye and limb movements may occur in response to environmental stimuli. Occasionally, the eyes will open in response to verbal stimuli. No comprehensible word or communication is ever expressed, and complex behavior is never initiated. Brain stem functions, such as temperature regulation, intestinal mobility, and respiration, are present. Karen Quinlan exists in this state, "awake but not aware."[66] At no time did she ever meet any of the criteria for brain death, and on examination six months after her initial episode of anoxia, she still possessed a variety of cephalic reflexes.[67] Spontaneous respirations were present, EEG activity was recorded, and normal intracranial circulation confirmed by angiography was found. The differences from brain death are obvious, though the differences between features encompassed by the term persistent vegetative state as compared to the other terms discussed here may at times seem small. In the final analysis of the neuropathologist, a variety of lesions in a variety of locations may produce relatively similar clinical pictures. What is most important to realize, and I hope that the repeated emphasis may be forgiven, is that these patients fit no current criteria for legal death.

Finally, quite distinct from brain death and from states of prolonged unresponsiveness is a condition perhaps described as early as 1875, but definitely named in 1965, as the locked-in syndrome.[68] It has also been called pseudocoma, the de-efferented state or cerebromedullospinal disconnection. Such patients usually have lost all limb and facial movements, but jaw, lid, and eye movements characteristically are preserved and, most importantly, the capacity for awareness and responsiveness is undeniably intact. Meaningful communication may be established through eye blink codes with these unfortunate individuals. Most such patients have suffered strokes, though trauma or demyelination (loss of the fatty insulating covering of nerve fibers) may be the cause in a few. Since such patients are aware, responsive, and alive by every medical and legal criterion, we shall not discuss them further.

A question now to be considered is: Should the legal definition of death cover irreversible loss of cognitive function, whether the condition be called a persistent vegetative state, the apallic syndrome, akinetic mutism, or whatever? In 1975, the New Jersey court dealing with the Quinlan case ap-

64. Brierley, *et al.*, *Neocortical Death after Cardiac Arrest*, 2 LANCET 560 (1976).

65. Jennett & Plum, *Persistent Vegetative State After Brain Damage. A Syndrome in Search of a Name*, 1 LANCET 734 (1972).

66. F. PLUM & D. LEVY, *Outcome From Severe Neurological Illness; Should It Influence Medical Decisions?*, in BRAIN AND MIND 267 (CIBA FOUNDATION SYMPOSIUM 69, 1979).

67. Korein, *Editors Comment*, 315 ANNALS N.Y. ACAD. SCI. 320 (1978).

68. F. PLUM & J. POSNER, THE DIAGNOSIS OF STUPOR AND COMA 6 (2d ed. 1972).

parently made "cognition," not brain death as we have discussed it, an all important point in decision-making.[69] "It formulated a procedure to permit withdrawal of a presumptively life-supporting respirator following a medical determination that cognition was irretrievably lost. The court thus established a legal precedent for terminating care of those adults who, while retaining vegetative neurological functions, lack the capacity to interact with the external environment."[70] This view may create enormous problems for patients, families, physicians, and society. How does one end the life of a patient whose heart beats, whose lungs move adequately, and whose homeostatic mechanisms are preserved, without committing homicide? What does one do if the view of family and physician differ with regard to the degree of effort to be exerted to maintain life, especially when the mere discontinuance of a respirator does not solve the problem (as it did not for Quinlan and as it would not for most such patients)? Inasmuch as only rare patients are known to have recovered to a variable degree from a vegetative state (almost all of whom were post-traumatic),[71] how much credence should be given to those who believe that any chance for recovery, no matter how small, is worth all the cost and effort that goes into the care of such patients? And finally, should the court, a hospital or community committee, or an individual physician have the final decision-making authority in such difficult cases?

Taking the last question first, my personal, pragmatic view is that the physician, and not the court, is in the best position to give advice to the concerned family and to chart a course of action with them. The doctor is best able, utilizing special help from consultants or from a hospital committee of experts if needed, to ascertain the cause of the condition and to predict, within the limits of scientific knowledge, an outcome. He or she can support the family in its grief, answer questions, and sometimes educate or even arbitrate. Flexibility of action with regard to the maintenance or institution of various forms of treatment, as well as their omission, can be agreed upon to the general satisfaction of all. While this methodology will not avoid disagreement in every case, my experience leads me to believe that most rational and caring physicians and families can reach a *modus operandi* that will avoid a Quinlan-type confrontation, in which the family wanted to discontinue support and the physicians refused. The age-old basis on which good physicians have acted is discretionary, and not authoritative, care. It would be wrong, in my opinion, to substitute either legislative or judicial fiat for this type of flexibility. Formalization and standardization of action for patients in prolonged noncognitive states would invade the area of personal belief in a way that would harm freedom of choice. While the patient can

69. Beresford, *The Quinlan Decision: Problems and Legislative Alternatives*, 2 ANNALS NEUROLOGY 74, 75 (1977).

70. Beresford, *Cognitive Death: Differential Problems and Legal Overtones*, 315 ANNALS N.Y. ACAD. SCI. 339 (1978).

71. For a general discussion of the prospects for recovery after coma see A. BRICOLO, *Prolonged Post-Traumatic Coma*, in HANDBOOK OF CLINICAL NEUROLOGY 699 (1976); Levy, Knill-Jones & Plum, *The Vegetative State and Its Prognosis Following Nontraumatic Coma*, 315 ANNALS N.Y. ACAD. SCI. 293 (1978); Rosenberg, Johnson & Brenner, *Recovery of Cognition After Prolonged Vegetative State*, 2 ANNALS NEUROLOGY 167 (1977).

no longer express a choice, families and physicians can. If well documented statements from the patient, either verbal or in the form of a so-called living will, are available, the position so stated can be taken into consideration. My belief is that the end result of such reasoning together will, in most instances, provide an acceptable solution to a tragic problem.

Attempts to decide upon courses of action by courts in such cases likely will prove to be cumbersome, time-consuming, and expensive. In a recent case involving an 83-year-old Marist brother who went into a coma after a cardiac arrest,[72] the Appellate Division of the New York Supreme Court ruled that court approval must be obtained every time physicians want to stop life-supporting treatment for patients unable to make such a decision for themselves. Testimony by the patient's family, and his or her physician, and a hospital committee of three doctors, and a guardian appointed by the court, and the Attorney General or the District Attorney who shall be given the opportunity to have examinations conducted by physicians of his own choosing is to be taken in each case before a court decision is rendered. This ruling was given, despite the plea of the patient's twelve nieces and nephews and his religious superior to terminate mechanical respiration. The patient died after 114 days during the legal proceedings. His medical bill was $87,000, and his legal fees were $20,000, despite donation of some of their time by attorneys.[73] Further appeal is in progress.

A neurologist who has been a leader in attempts to delineate and clarify the persistent vegetative state has stated that "[d]ecisions about who shall live and who shall die cannot be left to doctors alone."[74] He suggests that it is society that decides how physicians should act in these circumstances, taking cognizance of the best scientific information available. The practical problem, however, is that our society has not yet reached a consensus opinion at this moment in time, and all the data that are needed for rational decision-making in every case have not yet been gathered. It appears unlikely to me that any court or legislative body at the present time will be able to define specific standards precise enough to cover all forms of medical and social responses to prolonged states of impaired cognition.

The question as to whether patients who exist in a vegetative state should be considered legally dead is certain to engender strong emotions, regardless of which way it is answered. Personally, I do not believe they should be considered legally dead, since I doubt very much that most physicians, based on the descriptions given above, would consider them medically dead. If this is the case, how might this difficult problem be managed? To begin with, such individuals fall into two groups—those who are in the unstable initial stages of their neurological problem and are not yet fixed in a vegetative state, and those who have survived for about a month and for whom past experience would indicate that there is practically no chance for further improvement of significance.[75]

72. Eichner v. Dillon, 73 A.D.2d 431, 426 N.Y.S.2d 517 (1980).
73. 426 N.Y.S.2d at 550.
74. F. PLUM & D. LEVY, *supra* note 66.
75. Levy, Knill-Jones & Plum, *supra* note 71, at 302.

For the first group, it would be useful if doctors possessed the ability to make reasonably accurate judgments of outcome very early after an acute catastrophic brain event for humanitarian and economic reasons. If such judgments predicted with great validity not only death but also the likelihood of occurrence of a vegetative state, then resuscitative efforts might be abandoned early and with a predictable effect rather than late. Most of the people who finally enter a relatively stable vegetative state do so after passing through a few turbulent weeks of cardiac, respiratory, chemical, and neurologic instability which requires a great deal of therapy, attention, and expense, usually in an intensive care unit, to maintain survival. Ideally, the first few hours would be best for this determination since the longer one waits to make the decision not to treat vigorously, the harder it is for all concerned to implement that decision.[76] The methods that physicians currently use, however, to estimate outcome are not refined enough, particularly in cases of non-traumatic coma, to always accurately predict the future based on observations in this short period of time. In only about one-quarter of such patients can favorable or unfavorable outcomes be predicted in the first six hours with a 95% confidence limit, and accuracy during a twenty-four-hour period is not much better.[77] By contrast, it now appears that coma or a vegetative state at seven days "is rarely associated with return of independence."[78]

Recent evidence suggests that in many cases three days may be the time period needed for valid judgments of prognosis. Of nine patients at the Denver General Hospital who remained unresponsive for at least three days and who survived to be discharged, four remained in a vegetative state, two required total nursing care, and three were unemployable due to major organic mental defects.[79]

A recent study of non-traumatic states, such as coma due to cardiac arrest from myocardial infarction (heart attack), seems of even more practical value.[80] Using four clinical parameters (pupillary light reflex, corneal blink reflex, withdrawal movements of the limbs, and verbalization of any type) in 261 patients evaluated three days following their acute cerebral insult, it was determined that when all four parameters were present 74% of patients made either a good recovery or had only a moderate degree of disability, 26% remained severely disabled, and no patients died or entered a vegetative state. By contrast, if after three days none of these parameters existed, 96% of such patients either died or entered a vegetative state, and the remaining 4% were severely disabled. This careful study, together with the knowledge that patients remaining in a vegetative state for more than a

76. F. PLUM & D. LEVY, *supra* note 66.

77. *Id.*

78. Levy, Knill-Jones & Plum, *supra* note 71, at 303.

79. Yarnell, *Neurological Outcome of Prolonged Coma Survivors of Out-of-Hospital Cardiac Arrest*, 7 STROKE 279, 281 (1976).

80. D. Levy & F. Plum, Evaluating Prognosis in Nontraumatic Coma. Interview with D. Levy, Associate Professor, Cornell University, and F. Plum, Chairman of the Dep't of Neurology, Cornell University, in Boston (Sept. 7-10, 1980), Poster Presentation 58, Meeting of the American Neurological Association.

month following an anoxic event have essentially no chance for recovery,[81] provides an operational approach, for the present, for many patients. If this data is discussed with a family, most families in my opinion, would not wish to continue vigorous resuscitative efforts, such as restarting the heart after a cardiac arrest, antibiotic treatment of significant infections, or the continuance of mechanical respiration. By contrast, some families would desire this type of vigorous therapy, and, if the physician agreed, resuscitative efforts could be continued. If the physician and family disagreed on the course of action, then another physician could be sought to take over the case. Outcomes for very young children and young adults in whom the potential for recovery is much greater remain to be ascertained, but undoubtedly will be forthcoming.

Unlike the absolutes associated with prediction of brain death, it may never be possible to do better than the figures quoted above. Obviously, further studies involving a larger number of patients are needed to corroborate and possibly refine this data. Even if the 4% figure is looked upon as an error rate, it should be realized that such surviving patients are unlikely to be productive citizens and that most will require permanent and near total care. Hence, it could be argued that no real error of prediction of outcome has been made if we are talking about the ability to function compared with an individual's prior state. A much higher error rate of 20% actually was made in the reverse direction when those individuals considered to have a good prognosis after three days later were evaluated.[82] The hardest patients to assess continue to be those who possess only one or two of the clinical signs after three days. At the present time, there is no certain way to tell who among those will live or die, become functional or be vegetative. Whether to continue to treat such patients after three days will have to be decided on an individual basis by the physician and family.

An additional important consideration in assessment of outcome is the cause of the problem itself. Those patients with a metabolic cause, such as liver failure, tend to have better outcomes than do those with structural injury, such as derived from a stroke or hypoxia. In one study of 500 patients who had non-traumatic coma,[83] the chances for the survivors to recover to a fully independent existence was three times greater for those who had metabolic causes when compared to hypoxic patients and four times greater when compared to stroke patients.

For the second group of noncognitive patients, fixed after a month or so in a vegetative limbo, only minimal medical and nursing care, such as feeding, turning, and cleansing, is needed to maintain them in a stable condition. I do not believe that most physicians and nurses would be willing to take the active step of stopping this basal care to end their lives, and I can, in fact, recall the acute discomfort experienced by the medical and nursing staff when a brain dead patient, disconnected from a respirator, continued to have cardiac action for many hours while the family insisted that the giving

81. F. PLUM & D. LEVY, *supra* note 66.
82. Levy & Plum, *supra* note 80.
83. F. PLUM & D. LEVY, *supra* note 66.

of intravenous fluid be discontinued. When a significant infection or other complication occurs, however, which ordinarily would call for further active treatment measures, then the physician and family may again consider a restrained course of action. This decision would be no different, in my opinion, from similar ones made frequently for patients terminally ill from cancer and other serious illnesses. If a collaborative decision is made to treat vigorously, it will be taken in full knowledge of the fact that recovery from a month-long vegetative state essentially never occurs.[84]

Justice Groves, paraphrasing William Cullen Bryant's poem of 1817, "Thanatopsis," spoke of the innumerable caravan moving to their chambers in the silent halls of death.[85] We now know that some individuals tragically pause in limbo during this journey, for what must seem to their families to be an eternity before they can lie "down to pleasant dreams."[86] As with all the difficult questions that face an ever-changing society, time, trial, and experience will eventually clarify the management of these irreversible states of cognitive loss. Operational, rather than legal or legislative methods, probably will solve most such situations. That some errors will be made is undeniable, since all physicians will wish to err on the side of life rather than death. I believe, however, that such errors will be infrequent and in keeping with the injunction carried in the heart of all physicians—above all, do no harm to your patient.[87]

84. Levy, Knill-Jones & Plum, *supra* note 71, at 302.

85. Lovato v. District Court, 601 P.2d 1072, 1075 (Colo. 1975) (citing W. C. Bryant, *Thanatopsis* (1817)).

86. W.C. Bryant, *Thanatopsis* (1817).

87. For further references in the general subject area of brain death, see Black, *Brain Death*, 299 NEW ENG. J. MED. 338, 393-401 (1978); Higashi, Sakata, Hatano, Abiko, Ihara, Katayama, Wakuta, Okamura, Ueda, Zenke & Aoki, *Epidemiological Studies on Patients with a Persistent Vegetaive State*, 40 J. NEUROL. NEUROSURG. PSYCHIAT. 876 (1977); Levy, Bates, Caronna, Cartlidge, Knill-Jones, Shaw & Plum, *Recovery From Nontraumatic Coma*, 103 TRANSACTIONS AM. NEUROLOGICAL A. 104 (1978); Stickel, *The Brain Death Criterion for Human Death. An Analysis and Reflections on the 1977 New York Conference on Brain Death*, 6 ETHICS SCI. MED. 177 (1979).