Donald I. Templer, PhD, professor of psychology at California School of Professional Psychology-Fresno, received his doctorate in clinical psychology from the University of Kentucky in 1967. He has contributed to more than 100 publications, most often writing in the areas of neuropsychology, schizophrenia, and death. He has over 1000 citations to his credit, with one of his earlier articles being declared a citation classic by Current Contents in 1984. Templer is an author of six assessment instruments. His Death Anxiety Scale has been translated into many languages and used on all six continents. A synthesis of this research is found in the 1986 Lonetto and Templer book Death Anxiety. He is a fellow of the American Psychological Association and the American Psychological Society.

Lawrence C. Hardage, PhD, directs the Augusta Neuropsychology Center in Augusta, GA, and consults to the courts and to rehabilitation hospitals concerning head injuries and their sequelae. He has served as president of the National Academy of Neuropsychology, and of the American Psychological Association division of neuropsychology. His academic appointments have included professor of neurology at the Medical College of Georgia and at Indiana University Medical Center, and Marie Wilson Howell visiting scholar at the University of Arkansas. He has edited the International Journal of Clinical Neuropsychology, Clinical Neuropsychology, and Neuropsychology and Special Education, and served as consulting editor to Archives of Clinical Neuropsychology, International Journal of Psychophysiology, and the American Psychological Association journal of Neuropsychology and Psychophysiology. He is a proponent of the field of neuropsychology, and his research in neuropsychology and special education has been recognized by the American Psychological Society.

W. Gary Cannon, PhD, received his doctorate in clinical psychology from Brigham Young University after completing a neuropsychological rotation at the University of Utah. After completing a postdoctoral fellowship with the Devereux Foundation, and teaching at the University of California, San Diego, he joined the faculty of the Children's Hospital Neuropsychology Laboratory at the University of California, San Diego. He is a fellow of the American Psychological Association and the American Psychological Society.

Brain Health and Brain Vulnerability

Preventable Brain Damage

Editors

W. Gary Cannon
Lawrence C. Hardage
Donald I. Templer
CONTENTS

Contributors vii

Introduction ix

Part I: Impact Damage

1. Brain Injury from Motor Vehicle Accidents 3
   Lawrence C. Hartlage and Gurntal Rattan

2. Contact Sports 15
   Richard H. Drew and Donald L. Tenpler

3. Noncontact Sports 30
   Donald I. Tenpler and Richard H. Drew

4. Accidental Injuries of Children 41
   William B. Miller and Frank D. Miller

5. Brain Injury from Motor Vehicle Accidents
   W. Gary Cannon and Donald L. Temple

6. Assault 72
   Robert Geffner and Alan Rosenbaum

7. Psychosurgery 80
   Donald L. Temple

8. ECT and Permanent Brain Damage 99
   W. Gary Cannon and Donald L. Temple

Part II: Chemical Damage

9. Industrial Toxins 111
   W. Gary Cannon and Donald L. Temple

10. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

11. Psychosurgery
    Donald L. Temple

12. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

13. Noncontact Sports
    Donald L. Temple and Richard H. Drew

14. Assault
    Robert Geffner and Alan Rosenbaum

15. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

16. Psychosurgery
    Donald L. Temple

17. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

18. Noncontact Sports
    Donald L. Temple and Richard H. Drew

19. Assault
    Robert Geffner and Alan Rosenbaum

20. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

21. Psychosurgery
    Donald L. Temple

22. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

23. Noncontact Sports
    Donald L. Temple and Richard H. Drew

24. Assault
    Robert Geffner and Alan Rosenbaum

25. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

26. Psychosurgery
    Donald L. Temple

27. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

28. Noncontact Sports
    Donald L. Temple and Richard H. Drew

29. Assault
    Robert Geffner and Alan Rosenbaum

30. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

31. Psychosurgery
    Donald L. Temple

32. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

33. Noncontact Sports
    Donald L. Temple and Richard H. Drew

34. Assault
    Robert Geffner and Alan Rosenbaum

35. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

36. Psychosurgery
    Donald L. Temple

37. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

38. Noncontact Sports
    Donald L. Temple and Richard H. Drew

39. Assault
    Robert Geffner and Alan Rosenbaum

40. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

41. Psychosurgery
    Donald L. Temple

42. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

43. Noncontact Sports
    Donald L. Temple and Richard H. Drew

44. Assault
    Robert Geffner and Alan Rosenbaum

45. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

46. Psychosurgery
    Donald L. Temple

47. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

48. Noncontact Sports
    Donald L. Temple and Richard H. Drew

49. Assault
    Robert Geffner and Alan Rosenbaum

50. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

51. Psychosurgery
    Donald L. Temple

52. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

53. Noncontact Sports
    Donald L. Temple and Richard H. Drew

54. Assault
    Robert Geffner and Alan Rosenbaum

55. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

56. Psychosurgery
    Donald L. Temple

57. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

58. Noncontact Sports
    Donald L. Temple and Richard H. Drew

59. Assault
    Robert Geffner and Alan Rosenbaum

60. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

61. Psychosurgery
    Donald L. Temple

62. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

63. Noncontact Sports
    Donald L. Temple and Richard H. Drew

64. Assault
    Robert Geffner and Alan Rosenbaum

65. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

66. Psychosurgery
    Donald L. Temple

67. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

68. Noncontact Sports
    Donald L. Temple and Richard H. Drew

69. Assault
    Robert Geffner and Alan Rosenbaum

70. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

71. Psychosurgery
    Donald L. Temple

72. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

73. Noncontact Sports
    Donald L. Temple and Richard H. Drew

74. Assault
    Robert Geffner and Alan Rosenbaum

75. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

76. Psychosurgery
    Donald L. Temple

77. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

78. Noncontact Sports
    Donald L. Temple and Richard H. Drew

79. Assault
    Robert Geffner and Alan Rosenbaum

80. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

81. Psychosurgery
    Donald L. Temple

82. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

83. Noncontact Sports
    Donald L. Temple and Richard H. Drew

84. Assault
    Robert Geffner and Alan Rosenbaum

85. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

86. Psychosurgery
    Donald L. Temple

87. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

88. Noncontact Sports
    Donald L. Temple and Richard H. Drew

89. Assault
    Robert Geffner and Alan Rosenbaum

90. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

91. Psychosurgery
    Donald L. Temple

92. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

93. Noncontact Sports
    Donald L. Temple and Richard H. Drew

94. Assault
    Robert Geffner and Alan Rosenbaum

95. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

96. Psychosurgery
    Donald L. Temple

97. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

98. Noncontact Sports
    Donald L. Temple and Richard H. Drew

99. Assault
    Robert Geffner and Alan Rosenbaum

100. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

101. Psychosurgery
    Donald L. Temple

102. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

103. Noncontact Sports
    Donald L. Temple and Richard H. Drew

104. Assault
    Robert Geffner and Alan Rosenbaum

105. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

106. Psychosurgery
    Donald L. Temple

107. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

108. Noncontact Sports
    Donald L. Temple and Richard H. Drew

109. Assault
    Robert Geffner and Alan Rosenbaum

110. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

111. Psychosurgery
    Donald L. Temple

112. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

113. Noncontact Sports
    Donald L. Temple and Richard H. Drew

114. Assault
    Robert Geffner and Alan Rosenbaum

115. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

116. Psychosurgery
    Donald L. Temple

117. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

118. Noncontact Sports
    Donald L. Temple and Richard H. Drew

119. Assault
    Robert Geffner and Alan Rosenbaum

120. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

121. Psychosurgery
    Donald L. Temple

122. Accidental Injuries of Children
    William B. Miller and Frank D. Miller

123. Noncontact Sports
    Donald L. Temple and Richard H. Drew

124. Assault
    Robert Geffner and Alan Rosenbaum

125. Brain Injury from Motor Vehicle Accidents
    W. Gary Cannon and Donald L. Temple

126. Psychosurgery
    Donald L. Temple
The use of ECT in the United States is decreasing. In fact, there was a 46% decrease from 1975 to 1980. However, even in 1980 there were 33,384 psychiatric patients given ECT. In California, legislation in 1975 severely restricted the use of ECT. Nevertheless, from 1977 to 1980, 17.5% ECT was used in California's prisons. In the patients given ECT (Thompson & Blain, 1987), in California's psychiatric hospitals, the patients were over 70. In fact, it was a 46% decrease from 1975 to 1980. However, even in 1980 there were 33,384 psychiatrists who were more actively involved with ECT. Nevertheless, the issue of ECT is very controversial. It may be comparable to other emotionally laden issues such as ethnic differences in IQ and the bad effects of marijuana. Friedberg (1977), an outspoken critic of ECT, attributed the rise of ECT in the 1930s to the authoritarian political era in which 275,000 inmates in German psychiatric hospitals were starved, beaten, drugged, and gassed to death. On the other hand, Shukla (1981) stated, "Despite abhorrence in some quarters, it is still being practiced as one of the cheapest and safest, and yet one of the most effective, therapeutic techniques in the whole of medicine."

Some questions are still being answered. Consider the following: In an evaluation of ECT in the 1930s, the authorization of ECT in the United States, in which the issue of ECT was highly debated, is considered. A controversy exists as to how effective it is in treating depression. It may be that the effectiveness of ECT is due to the fact that it is a very controversial treatment. It is a topic that is often debated.
It is especially valued in the recalcitrant cases of depression that do not respond to antidepressant drugs. It is not going to become an obsolete treatment unless and until more effective antidepressant drugs are developed. Janicak, Davis, Gibbons, Ericksen, Chang, and Gallagher (1985) published a meta-analysis that showed ECT to be clearly superior to the tricyclic antidepressants.

ECT was used as a form of treatment in the early 1940s. It was developed as a way to control seizures in people with epilepsy. However, it was later discovered that ECT could also be used to treat depression. In the 1950s, ECT was widely used to treat depression in the United States, but its use declined in the 1960s and 1970s as newer antidepressant drugs became available.

The current evidence suggests that ECT is effective for treating depression, particularly in cases where other treatments have not been successful. However, ECT is not without its risks. Some patients experience memory loss and other cognitive impairments after receiving ECT. These side effects are more common with higher doses of ECT. Therefore, it is important to carefully weigh the benefits and risks of ECT before deciding whether to use it as a treatment option.

Animal brain autopsy reports, animal brain studies, the brains of epileptics, spontaneous seizures, psychological test findings in patients with a history of many ECT, CT scan findings, and magnetic resonance imaging have all been used to study the effects of ECT on the brain. Although these studies suggest that ECT can cause permanent brain damage, the extent of this damage is not yet fully understood.

In summary, ECT is a treatment option for depression that is effective but has significant side effects. It is important for healthcare professionals to carefully consider the risks and benefits of ECT before recommending it to patients.
than, although similarly patterned as, animals not convulsed without special measures Meldrum & Brerley, 1913; Meldrum, Vigourocex, & Brerley, 1973.

difficult because of the great variation in stimulus parameters and other properties of the ECT, the different types of animals, and varying sophistication of both animals and humans. It is possible to cause definite permanent brain damage through ECT, and it is possible to administer ECT with minimal or no damage. It is not a matter of whether ECT can produce permanent damage but a matter of in what circumstances it occurs.

HUMAN BRAIN AUTOPSY REPORTS

In the 1940s and 1950s, there were a large number of reports concerning the examination of brains of persons who had died following ECT. Madow (1956) noted that many of these could have been of a potentially reversible nature. Such reversibility was much less with the 12 patients who had neuronal and/or glial pathology. In one case, the author Riese, 1948, in addition to giving the neuronal and glial changes, reported numerous slits and rents similar to that seen after execution. Needless to say, patients who died following ECT are not healthy. Madow concluded, on the basis of these 38 cases and five of his own, "If the individual being treated is well physically, most of the neuropathological changes are reversible. If, on the other hand, the patient has cardiac, vascular, or renal disease, the cerebral changes, chiefly vascular, may be permanent."

An interesting autopsy case report was presented by Llppmann et al. (1984). An 89-year-old woman with a long history of psychiatric illness died in 1982. There was also some unsubstantiated evidence of her having received 800 additional ECTs. The authors stated that the moderate cerebral atrophy was consistent with her age and did not show old focal ischemic lesions or any additional ECTs. They also stated that the examination of the frontal lobes failed to reveal the sites of the cannula used in her prefrontal lobotomy in 1953. The authors concluded that the absence of CT changes cannot exclude damage but that it is encouraging that CT showed no evidence of this occurring with ECT. The authors stated that examination of the brains of persons who had died following ECT is important in understanding the effects of ECT on the brain. It is possible to cause definite permanent brain damage through ECT, and it is possible to administer ECT with minimal or no damage. It is not a matter of whether ECT can produce permanent damage but a matter of in what circumstances it occurs.
respect to time and place, and amnestic for events before the day of ECT. Nevertheless, no CT changes were observed. The findings would appear to point to the safeness of the ECT. However, the present author is willing to entertain an alternative explanation. If the CT did not reflect the massive acute brain syndrome with gross disorientation, then it may not be capable of detecting minor changes in patients months or years after the ECT. Perhaps the CT scan is not the most optimal tool for ruling out brain changes resulting from ECT.

A reasonable generalization may be that CT scans have failed to provide a definitive perspective with respect to the matter of permanent brain damage.

MAGNETIC RESONANCE IMAGING

Coffey and colleagues (1988) reported on magnetic resonance imaging before and after ECT administered to nine depressed patients. Blind raters' assessments showed no significant differences between pre- and post-ECT in cortical atrophy and global comparison. There were also no significant changes in ventricle-to-brain ratios. Furthermore, patients with preexisting brain disease showed no worsening. However, the authors did state: "Still these observations need to be confirmed in a larger number of subjects with techniques that will quantitate even subtle brain changes which might otherwise not be detected by qualitative clinical assessments. Further studies should also include patients with histories of previous ECT to evaluate any potential cumulative effects and should involve long-term follow-up studies including both subjective and objective measures of memory function." (p. 706).

A case report of a multiple sclerosis patient with magnetic resonance imaging before and after ECT is reassuring. There was no evidence of changes in white matter lesions visualized on spin-echo images. (Coffee, Weiner, McCall, & Heinz, 1987).

In summary, the two studies using magnetic resonance imaging did not provide evidence of permanent brain damage resulting from ECT. However, more studies are needed.

PSYCHOLOGICAL TESTING WITH A PAST HISTORY OF MANY ECTS

Goldman, Gomer, and Templer (1972) administered the Bender-Gestalt and the Benton Visual Retention Test to schizophrenics in a VA hospital. Twenty had a past history of from 50 to 219 ECTs, and 20 had no history of ECT. The ECT patients did significantly worse on both instruments, with performance on ECT-imposed (p. 707).

More studies are needed with large patient samples with magnetic resonance imaging.

BRAINS OF EPILEPTICS

ECT and permanent brain damage. (p. 708).

The recent emphasis on psychosurgery suggests that postmortem examination of patients who have sustained brain injuries or strokes might provide useful information about the effects of ECT on brain structure and function. However, it is important to remember that the effects of ECT on brain tissue are not fully understood, and that the relationship between ECT and brain damage is still being investigated.

The findings of the present study suggest that ECT does not cause permanent brain damage. However, further research is needed to determine the extent of brain damage resulting from ECT.

ECT and Permanent Brain Damage

A reasonable generalization may be that ECT does not cause permanent brain damage. However, the relationship between ECT and brain damage is still being investigated.
Gastaut and Gastaut (1976) demonstrated through brain scans that in seven of 20 cases status epilepticus produced brain atrophy. They reasoned, "Since the edema and the atrophy were unilateral and bilateral and related to the localization of the convulsions unilateral or bilateral chronic seizures, the conclusion can be drawn that the atrophic process depends upon the epileptic process and not on the cause of the status" (p. 18).

A common finding in epileptics and ECT patients is noteworthy. Norman (1964) stated that it is not uncommon to find at autopsy both old and recent lesions in the brains of epileptics. Alpers and Hughes (1942) reported old and recent brain lesions associated with different series of ECT.

**Spontaneous Seizures**

The reports of spontaneous seizures, which appeared in the pre-1960s ECT era, probably do not constitute one of the more definitive domains. However, this section is included to increase breadth of perspective.

It would appear that if seizures that were not previously evidenced appeared after ECT and persisted, permanent brain pathology must be inferred. There have been numerous cases of post-ECT spontaneous seizures reported in the literature and briefly reviewed by Blumenthal (1955), Pacella and Barrera (1945), and Karliner (1956). It appears that in the majority of cases the seizures do not persist indefinitely, although an exact perspective is difficult to obtain because of anticonvulsant medication employed and the limited follow-up information. Another difficulty is in all cases, definitively tracing the etiology to the ECT, since spontaneous seizures develop in only a very small proportion of patients given this treatment. Nevertheless, the composite of relevant literature does indicate that, at least in some patients, no evidence of seizure potential existed before treatment and post-ECT seizures persist for years.

An article that is one of the most systematic and representative in terms of findings is that of Blumenthal (1955) who reported on 12 schizophrenic patients in one hospital who developed post-ECT convulsions. Six of the patients had previous EEGs with four of them being normal, one clearly abnormal, and one mildly abnormal. The patients averaged 72 ECTs and 12 spontaneous seizures. The time from last treatment to first spontaneous seizure ranged from 12 hours to 11 months, with an average of 2½ months. The total duration of spontaneous seizures in the study period ranged from 1 day to 3½ years, with an average of 1 year. Following the onset of seizures, eight of the 12 patients were found to have a clearly abnormal, and one a mildly abnormal EEG. Masovich and Catzenelbogen (1948) reported that 20 of their 82 patients had convulsive pattern cerebral dysrhythmia 10 months post-ECT. None had such in their pretreatment EEG. Nine (15%) of the 60 patients who had three to 15 treatments, and 11 (50%) of the 22 patients who had 16 to 42 treatments had this 10-month post-treatment EEG. However, these results are not definitive, and further research is needed.
worse on tests. However, it would be much more difficult to explain away the
p < 0.05. And, as already stated, Hartellus found greater damage, both reversible
and irreversible, in cats that were given 11 to 16 than in those given four ECTs.

EEGs more adversely affected by treatment. Hartellus' statement that patients with pretreatment EEG abnormalities are more
likely to show marked post-ECT cerebral dysrhythmia and to generally show
EEGs more adversely affected by treatment. Hartellius 1952 found significantly more reversible and irreversible brain
damage, both during and after ECT, in patients with pretreatment EEG abnormalities than in those without.

It is recommended that more research be carried out on the safety and the
impact of ECT on the brain. Best practices and guidelines for ECT should be
formulated to minimize the risk of permanent brain damage.

**RECOMMENDATIONS**

It is recommended that more research be carried out on the safety and the
impact of ECT on the brain. Best practices and guidelines for ECT should be
formulated to minimize the risk of permanent brain damage.
The effect of electric shock therapy upon cerebrospinal fluid


Hartelius, H. 1952. Cerebral changes following electrically induced convulsions: An


Goldman, H., Gomer, F. E., & Templer, D. J. 1972. Long-term effects of electroconvulsive


KarIlner, W. 1956. Epileptic states following electroshock therapy. Journal of Hillside

larsen, B. F., & Vraa-Jensen, 13. 1953. lschaemià changes in the brain following


Levy, N. A., Serota, H. M., & Grinker, P. ft. 1942. Disturbance in brain function following


Madow, L 1956. Brain changes in electroshock therapy, American Journal of Psychiatry,


Meyer, 0., & O'Daniel, ft. 1985. 1250 Electroconvulsant treatments without evi


Norman, P.. M. 1964. The neuropathology of status eplleptlcus. Medicine, Science and the

OEET AND PERMANENT BRAIN DAMAGE 107

ECT AND PERMANENT BRAIN DAMAGE 107