Dr. Liu Fuchs

marked 11/30/81

Y. KING LIU

CURRICULUM VITAE

I. Personal Data

Born in Nanking, China on May 3, 1934. Married, two children. U.S. Citizen.

II. University Education

B.S.	(M.E.), Bradley, University, Peoria, IL	1955
M.S.	(M.E.). University of Wisconsin, Madison, WI	1959
Ph.D.	(Biomechanics), Wayne State Univ., Detroit, MI	1963

III. Licensure

Certified Acupuncturist, State of California, License No. AC737 1978

IV. Teaching and Research Experience

Milwaukee School of Engineering, Milwaukee, WI
Assistant Professor, Mechanical Engineering 1956-1956

Wayne State University, Detroit, MI
Instructor, Engineering Mechanics 1960-1963

University of Michigan, Ann Arbor, MI
Lecturer, Engineering, Mechanics 1963-1964
Assistant Professor, Engineering Mechanics 1964-1968

Stanford University, Stanford, CA
Visiting Assistant Professor, Aeronautics and Astronautics 1968-1969

Tulane University Schools of Medicine and Engineering, New Orleans, LA
Associate Professor, Biomechanics 1969-1972
Professor and Director, Biomechanics Laboratory 1972-1978

University of Iowa College of Engineering and Medicine, Iowa City, IA
Professor and Director, Center for Materials Research 1978-Date
Co-Director, Rehabilitation Engineering Center 1979-Date

V. Industrial Experience

a. Employment
J.I. Case Company, Bettendorf, IA
Product Design Engineer 1955-1956

b. Consulting
Kurz and Root Company, Cedarburg, WI 1958-1961
Vincon Corporation, Detroit, MI 1962
Various attorneys 1978-Date
Biokinetics & Assoc., Ottawa, Ontario, Canada 1981

DEFT. EX. DD- Liu Exh. 1
DATE: 11/30/81
REPORTER: ALBERT J. GASDOR

VI. Honors and Awards

Full Tuition Scholarship, Bradley University	1952-1955
Sigma Xi, Full Member	1961-Date
American Men and Women of Science	1962-Date
Special Research Fellow, National Institutes of Health	1968-1969
Research Career Development Award, NIH	1971-1976
Visiting Professor of Biomechanics, Karolinska Institute Stockholm, Sweden	1972

VII. Current and Past Research Grants and Contracts

*indicates project is current

1. UI-00025-01,02,03 PHS Grant "The Human Body Under Time-Dependent Boundary Conditions," Principal Investigator, University of Michigan, Ann Arbor, MI, September, 1966-August, 1969.

2. 43-67-1136 PHS Contract with NINBS, Head Injury Project, Principal Project Analyst, University of Michigan, Ann Arbor, MI, September, 1967-August, 1968.

3. EC-00402-01 PHS Grant, "The Human Body Under Time-Dependent Boundary Conditions," Principal Investigator, Tulane University, New Orleans, LA, September, 1969-August, 1970.

4. FD-00055-08 PHS Grant, "Experimental Acceleration Injury," Co-Principal Investigator, Tulane University, New Orleans, LA, September, 1969-September, 1971.

5. F33615-72-C-1212 Research Contract with Aerospace Medical Research Lab., U.S. Air Force Systems Command, Wright-Patterson AFB, entitled, "A Continuum Model of Primate Body Response to Impact," Principal Investigator, June, 1970-March, 1973, $72,964.

6. DABCOL-73-1068 Research Contract with U.S. Army Aeromedical Research Lab., Ft. Rucker, AL, entitled "The Effect of Initial Configuration on Pilot Ejection," Principal Investigator, January-July, 1973. $9,968.

7. GK 32047 National Science Foundation Research Grant, "The Structural Biomechanics of the Human Spine," Principal Investigator, January 15, 1972-December 31, 1974. $79,996.

8. NS 10517 National Institute of Neurological Diseases and Stroke, NIH Research Grant, "Acceleration Injuries to the Spinal Cord and Brainstem," Principal Investigator, May, 1972-December, 1974. $63,299.

9. DAMD 17-74-G-9384 Research Contract with U.S. Army Aeromedical Research Lab., Ft. Rucker, AL, entitled, "The In Vivo Dynamic Material Properties of the Spinal Cord - A Feasibility Study," Principal Investigator, September, 1973-August, 1974. $4,991.

10. GM-40723 NIH Research Grant,"The Biomechanics of the Musculoskeletal System," Research Career Development Awardee, May, 1971-April, 1976. $119,500 for 5 years.

11. GM-19107 National Institute of General Medical Sciences, NIH Research Grant, "The Traumatology of the Head and Spine," Principal Investigator, June, 1972-January, 1976. $118,555.

12. Edward G. Schlieder Educational Foundation, Research Grant, "Localization of Acupuncture Points in Experimental Animals," Principal Investigator, July 1, 1974-June 30, 1976. $36,983.

13. NS-10517, National Institute of Neurological Diseases and Stroke, NIH Research Grant, "Acceleration Injuries to the Spinal Cord and Brainstem," Principal Investigator, May, 1975-September, 1978. $115,509.

14. DABT-01-75-C-0237, Research Contract with U.S. Army Aeromedical Research Lab., Ft. Rucker, AL, "The In Vivo Dynamic Material Properties of the Spinal Cord," Principal Investigator, April, 1975-March, 1976. $16,363.

15. GM-19107-06 Research Grant with National Institute of General Medical Sciences, NIH, "The Traumatology of the Head and Spine," Principal Investigator, February 1, 1976-January 31, 1979. $181,542.

16. F33615-76-C-0526 Research Contract with Aerospace Medical Research Lab., U.S. Air Force Systems Command, Wright-Patterson AFB, "Investigation of Cervical Spine Dynamics," Principal Investigator, September, 1976-December, 1979. $48,552.

17. DABT-01-77-C-0015 Research Contract with U.S. Army Aeromedical Research Lab., Ft. Rucker, AL, "The In Vivo Dynamic Material Properties of the Canine Spinal Cord," Principal Investigator, November, 1976-October, 1977. $24,239.

18. DABT-01-C-0086 Research Contract with U.S. Army Aeromedical Research Lab., Ft. Rucker, AL. "The In Vivo Dynamic Material Properties of the Canine Spinal Cord," Principal Investigator, February, 1978-December 1979. $27,632.

19. GM-26608-01, Research Grant with National Institute of General Medical Sciences, NIH, "The Traumatology of the Head and Spine," Principal Investigator, April 1, 1979-March 31, 1981. $169,598.

20. 13-627, Grant from Rehabilitation Services Administration, Rehabilitation Engineering Center Grant, September, Co-Director, September, 1979-August, 1980. $54,700 out of $400,000 center budget earmarked for biomechanics of the spine.

21. Grant from the American Osteopathic Academy of Sclerotherapy, "A Biomechanical Study of Connective Tissue Reaction to Irritating Substances, Principal Investigator, December, 1978-July, 1980. $6,486.

22. 23-P-59176, Grant from National Institute for Handicapped Research, Department of Education, Rehabilitation Engineering Center Grant, Co-Director, September 1980-August 1981. $90,000 out of $45,000 budget earmarked for engineering analysis of the spine.

23. *DAMD-17-81-C-1186, Contract from the U.S. Army Aeromedical Research Lab., DOD, Ft. Rucker, AL, "A Finite-Element Model Analysis of the Protection Provided by Army Aviator Helmets to the Human Head and Neck," Principal Investigator, September 28, 1981-May 28, 1982. $39,821.

24. *23-P-59176, Grant from National Institute for Handicapped Research, Department of Education, Rehabilitation Engineering Center Grant, Co-Director, September 1981-August 1982. $109,825 out of $434,200 center budget earmarked for basic studies of low-back pain.

VIII. Current and Past Training Grants and Contracts

1. 01007-03-04 U.S. Public Health Service Training Grant, "Multiphasic Research Training in Injury Control," Coordinator, July 1, 1969-January 31, 1972.

IX. Other Relevant Research Activity

a. Grants and Contracts Review
   1. Member, Safety and Occupational Health Study Section, National Institute for Occupational Safety and Health, (NIOSH), Public Health Service. Term: July, 1970-July, 1972.
   2. Clinical B Study Section, NIH, 1979-1981

b. Consultancy on Biomechanics to
   1. U.S. Army Aeromedical Research Lab., Ft. Rucker, AL
   2. V.A. Hospital, New Orleans, LA
   3. Dept. of Neurological Surgery, University of Chicago, IL.

c. National Committees
   1. Chairman, Subcommittee on Animal Research, Head and Neck Injury Research Workshop, Dept. of Transportation, 1978
   2. Chairman, Working Group on Biomechanics Data Bank, Committee on Hearing Bioacoustics and Biomechanics (CHABA), National Research Council, National Academy of Sciences, 1977-1979
   3. Advisor-at-Large, Committee on Hearing Bioacoustics and Biomechanics (CHABA), National Research Council, National Academy of Sciences, 1979-present

X. Publications

a. Books and monographs

1. Liu, Y.K, and Wickstrom, J.K., "Estimation of Inertial Property Distribution of the Human Torso From Segmented Cadaveric Data," Perspectives in Biomedical Engineering, R.M. Kenedi, Ed., University Park Press, Baltimore, MD, 1972, pp. 203-213.

2. Liu, Y.K., "Mechanics, Evaluation, Prognosis and Management of Head Injury," Invited Chapter in Perspectives in Biomechanics (Reul, Ghista, and Rau, Eds.), Vol. 1, Part B., Harwood-Academic Press, 1980., Chapter 11, pp. 573-599.

3. Liu, Y.K., "Spinal Injury; Mechanisms, Modelling, Systems for Minimizing Trauma," Chap. in book, Biomechanics of Physiological Mechanism and Body Dynamics, D.N. Ghista, Ed., Oxford Univ. Press (in press).

4. Hosey, R., and Liu, Y.K., "A Homeomorphic Finite Element Model of the Human Head and Neck", Chapter 18, Finite Element Methods in Biomechanics, John Wiley & Son (in press).

b. Papers already published and accepted for publication in technical journals or proceedings with rigorous review procedure.

1. Denman, H.H. and Liu, Y.K., "A Graphical Procedure for the Approximation of the Period of Non-Linear Free Oscillations," Journal of Applied Mechanics, Vol. 31, No. 4, December, 1964, pp. 718-719.

2. Denman, H.H. and Liu, Y., "Application of Ultraspherical Polynomials to Non-Linear Oscillations, II - Free Oscillations," Quarterly Applied Mathematics, Vol. 22, No. 4, January, 1965, pp. 273-292.

3. Denman, H.H. and Liu, Y.K., "Application of Ultraspherical Polynomials to Non-Linear Systems with Step-Function Excitation," Journal Industrial Mathematics Society, Vol. 15, Part 1, 1965, pp. 19-35.

4. Liu, Y.K., "Application of Ultraspherical Polynomials to Non-Linear Forced Oscillations," Journal of Applied Mechanics, Vol. 34, Part 1, March, 1967, pp. 223-226.

5. Liu, Y.K., "Towards a Stress Criterion of Injury - An Example in Caudocephalad Acceleration," Journal of Biomechanics, Vol. 2, Part 2, May, 1969, pp. 145-149.

6. Engin, A.E. and Liu, Y.K., "Axisymmetric Response of a Fluid-Filled Spherical Shell in Free Vibrations," Journal of Biomechanics, Vol. 3, Part 1, January, 1970, pp. 11-22.

7. Orne, D. and Liu, Y.K., "A Mathematical Model of Spinal Response to Impact," Journal of Biomechanics, Vol. 41, Part 1, January, 1971, pp. 49-71. (ASME Preprint No. 70-BHF-1)

8. Liu, Y.K., Laborde, J.M. and Van Buskirk, W.C., "Inertial Properties of a Segmented Cadaver Trunk: Their Implications in Acceleration Injuries," Aerospace Medicine, Vol. 42, Part 6, 1971, pp. 650-657.

9. Liu, Y.K. and von Rosenberg, D.U., "A One-Dimensional Continuum Model of Direct-Closed Head Impact," Proceedings of the Conference on International Research Committee on Biokinetics of Impacts, Amsterdam, Holland, June, 1973, pp. 285-301.

10. Chan, H.S. and Liu, Y.K., "The Asymmetric Response of a Fluid-Filled Spherical Shell - A Mathematical Simulation of a Glancing Blow to the Head," Journal of Biomechanics, Vol. 7, Part 1, January, 1974, pp. 43-59.

11. Liu, Y.K. and von Rosenberg, D.U., "The Effects of Caudocephalad ($+G_z$) Acceleration on the Initially Curved Spine," Computers in Biology and Medicine, Vol. 4, Part 1, June, 1974, pp. 85-106.

12. Liu, Y.K., Ray, G. and Hirsch, C., "The Resistance of the Lumbar Spine to Direct Shear," The Orthopaedic Clinics of North America, Vol. 6, Part 1, January, 1975, pp. 33-48.

13. Liu, Y.K. and Chandran, K.B., "The Exact Solution to the Translational Acceleration of Inviscid Compressible Fluid in Rigid Spherical Shells," Mathematical Biosciences, Vol. 24, 1/2, June, 1975, pp. 1-16.

14. Liu, Y.K. and Chandran, K.B., "Package Cushioning for the Human Head I. Analytical Considerations," Journal of Applied Mechanics, Vol. 42., 1975, pp. 541-546. (ASME Preprint No. WA/Bio-10)

15. Liu, Y.K., Chandran, K.B. and von Rosenberg, D.U., "Angular Acceleration of Viscoelastic (Kelvin) Material in a Rigid Spherical Shell - A Rotational Head Injury Model," Journal of Biomechanics, Vol. 8, No. 5, September, 1975, pp. 285-292.

16. Liu, Y.K., Varela, M. and Oswald, R., "The Correspondence Between Some Motor Points and Acupuncture Loci," American Journal of Chinese Medicine, Vol. 3, Part 4, October, 1975, pp. 347-358.

17. Chandran, K.B., Liu, Y.K. and von Rosenberg, D.U., 'The Exact Solution of the Translational Acceleration of a Low Modulus Elastic Medium in Rigid Spherical Shells - Implications for Head Injury Models," Journal of Applied Mechanics, Vol. 42, Part 4, December 1975, pp. 759-762.

18. Liu, Y.K., Nikravesh, P. and Beck, C., "Optimal Protection in Direct-Closed Head Impact," Institute of Electrical and Electronics Engineers, Inc., Transactions of Biomedical Engineers Vol. BME-23, No. 1, January, 1976, pp. 29-35.

19. Cramer, H., Liu, Y.K. and von Rosenberg, D.U., "A Distributed Parameter Model of the Inertially-Loaded Human Spine," Journal of Biomechanics, Vol. 9, Part 3, March, 1976. pp. 115-130.

20. Chandran, K.B., Liu, Y.K, and von Rosenberg, D.U., "Stress Wave Propagation in Maxwell Fluid Contained in Rigid Spherical Shells," Journal of Sound and Vibration, Vol. 47, Part 1, July, 1976, pp. 107-114.

21. Liu, Y.K. and Chandran, K.B., "An Experimental Study of Package Cushioning for the Human Head," Journal of Applied Mechanics, Vol. 43, Part 3, September, 1976, pp. 469-474.

22. Van Buskirk, W.C., Watts, R.G. and Liu, Y.K., "The Fluid Mechanics of the Semi-Circular Canals," Journal of Fluid Mechanics, Vol. 78, Part 2, December, 1976, pp. 87-99.

23. Lin, H.S., and Liu, Y.K. and Adams, K.H., "Mechanical Response of Lumbar Intervertebral Joint Under Physiological (Complex) Loading," Journal of Bone and Joint Surgery, Vol. 60:A1, January, 1978, pp. 41-55.

24. Liu, Y.K. and Ray, G., "Systems Identification Scheme for the Estimation of the Liner Viscoelastic Properties of the Inter-Vertbral Disc," Aviation, Space and Environmental Medicine, Vol. 49, Part 1, January, 1978, pp. 175-177.

25. Liu, Y.K., "Biomechanics of Closed-Head Impact," Journal of Engineering Mechanics Division, American Society of Civil Engineers, Special Volume on Biomechanics, T.K. Hung, Ed., Vol. 104, No. EM1, Proc. Paper 13540, February, 1978, pp. 131-152.

26. Lin, H.S., Liu, Y.K., Ray, G. and Nikravesh, P., "Systems Identification for Material Properties of the Intervertebral Joint," Journal of Biomechanics, Vol. 11, 1978, pp. 1-14.

27. Domer, F., Liu, Y.K., Chandran, K.B. and Krieger, K.W., "The Effect of Hyperextension-Hyperflexion (Whiplash) on the Function of the Blood-Brain Barrier of Rhesus Monkeys," Experimental Neurology, Vol. 63, 1979, pp. 304-310.

c. Invited papers published in Proceedings, Congresses, Symposia

1. Liu, Y.K. and Murray, J.D., "A Theoretical Study of the Effects of Impulse on the Human Torso," Biomechanics, American Society Mechanical Engineers, Y.C. Fung, Ed., November, 1966, pp. 167-186.

2. Liu, Y.K., "Distributed-Parameter Dynamic Models of the Spine," Proceedings of the Workshop in Bioengineering Approaches to the Problems of the Spine, Washington, D.C., September, 1970, pp. 51-58.

3. Liu, Y.K., "The Biomechanics of Spinal and Head Impact: Problems of Mathematical Simulation," Proceedings Symposium on Biodynamic Modelling and Its Applications, Dayton, OH, AMRL-TR-71-29, October, 1970, pp. 701-736.

4. Liu, Y.K., Chan, H.S. and Nelson, J.A.,III, "Intracranical Pressure Wave Propagation in Head Impact," Proceedings of Summer Computer Simulation Conference, Boston, MA, July, 1971, pp. 984-994.

5. Liu, Y.K. and Chandran, K.B., "The Exact Solution to the Rotational Acceleration of Elastic Material in Rigid Spherical Shells," Recent Advances in Engineering Science, Proceedings 10th Annual Meeting of the Society of Engineering Science, Vol. 8, Raleigh, NC, November, 1973, pp. 255-264.

6. Chandran, K.B., Liu, Y.K. and von Rosenberg, D.U., "Brain Injury Simulation - Translational Acceleration of Maxwell Medium in Rigid Spherical Shells," Proceedings of the 11th Annual Meeting of the Society of Engineering Science, Vol. 9, Durham, NC, November, 1974, pp. 182-183.

7. Pontius, U.R. and Liu, Y.K., "Neuromuscular Effects in a Dynamic Model of the Cervical Spine," Proceedings of the NSF Workshop of the Biomechanics of Volitional Effort, University of Florida, Gainesville, FL, April, 1975, pp. 147-175.

8. Pontius, U.R. and Liu, Y.K., "Neuromuscular Model of the Cervical Spine," Society of Automotive Engineers, Special Issue No. 412, October, 1976, pp. 21-30 (Paper No. 760770).

9. Hung, T.K., Skalak, R., Bugliarello, G., Liu, Y.K., Patel, D.J. and Albin, M.S., "Perspectives in Biomechanics Research and Education for Next Decade," Journal of Engineering Mechanics Division, American Society of Civil Engineers, Special Volume on Biomechanics, T.K. Hung, Ed., Vol. 104, February, 1978, pp. 3-9.

10. Liu, Y.K., "Biomechanics and Biophysics of Central Nervous System (CNS) Trauma," Chapter 3 in CNS Trauma Research Status Report, Sponsored by The Stroke and Trauma Program, National Institutes Neurological, Communicative Diseases and Stroke (NINCDS), National Institutes of Health, 1979, pp. 36-52.

d. Letters-to-the-Editors

1. Liu, Y.K. and Chandran, K.B., "Comment on 'Mathematical Model of a Head Subjected to an Angular Acceleration'," G.N. Bycroft, Journal of Biomechanics, Vol. 7, 1974, pp. 319-321.

2. Liu, Y.K., "Discussion of Measurement of Angular Acceleration of a Rigid Body Using Linear Accelerometers'," A.J. Padgaonkar, K.W. Krieger and A.I. King, Journal of Applied Mechanics, Vol. 43, 1976, pp. 337-338.

3. Liu, Y.K., "Response to Comments on Motor Points and Acupuncture Loci" by C.M. Godfrey in American Journal of Chinese Medicine; Vol. 4, 1976, pp. 408-409.

4. Liu, Y.K., "Discussion of 'Mathematical Modelling, Simulation and Experimental Testing of Biomechanical System Crash Response'," by A.I. King and C.C.Chou, Jour. of Biomechanics, Vol. 10, 1977, pp. 149-152.

6. Liu, Y.K., Cowin, S.C., von Rosenberg, D.U. and Adams, K.H., "A Continuum Model of the Primate Body Response to Impact," Research Report, AMRL-TR-71-99, Aerospace Medical Research Lab., Wright-Patterson, AFB, 1971.

7. Liu, Y.K. and Ray, G., "A Finite Element Analysis of Wave Propagation in the Human Spine," Research Report, AMRL-TR-73-40, Aerospace Medical Research Lab, Wright-Patterson, AFB, 1973.

8. Liu Y.K., Cramer, H. and von Rosenberg, D.U., "A Distributed Parameter Model of the Inertially-Loaded Human Spine: A Finite Difference Solution," AMRL-TR-73-65, Aerospace Medical Research Lab, Wright-Patterson, AFB, 1973.

9. Liu, Y.K., Pontius, U. and Hosey, R., "The Effects of Initial Spinal Configuration on Pilot Ejection," Final Report on Research Contract DABC01-73-C-1068, U.S. Army Aeromedical Research Lab, Fort Rucker, AL, 1973.

10. Liu, Y.K., Chandran, K.B., and Van Buskirk, W.C., "The In Vivo Dynamic Material Properties of the Spinal Cord - A Feasibility Study," Final Report on Research Contract No. DAMD-17-74-G9384, U.S. Army Aeromedical Research Lab., Fort Rucker, AL, 1974.

11. Liu, Y.K., "The Localization of Acupuncture Points in Experimental Animals," Annual Report to Edward G. Schlieder Education Foundation," November, 1974.

12. Liu, Y.K. and Edisen, A.E.U., "Effects of Electroacupuncture Stimulation on the Thalamus and Cortex of the Cat," Annual Report to Edward G. Schlieder Education Foundation, Dec., 1975.

13. Liu, Y.K., Jarrott. D., Chandran, K.B. and Krieger, K.W., "The In Vivo Dynamic Material Properties of the Feline CNS," Final Report on Research Contract DABT01-77-C-0015, U.S. Army Aeromedical Research Lab., Fort Rucker, AL, October, 1977.

14. Liu, Y.K., Krieger, K.W., Njus, G., Ueno, K., Connors, M., Wakano, K., and Thies, D., "Cervical Spine Stiffness and Geometry of the Young Human Male," Final Report of Contract F33615-76-C-0526, AFAMRL-TR-80-138, U.S. Air Force Aerospace Med. Research Lab., Wright-Patterson AFB. OH, September 1981.

g. Journals,publishers, and research support reviews
Journal of Biomechanical Engineering
Journal of Biomechanics
Journal of Applied Mechanics
Journal of Sound and Vibration
Int'l Journal of Neurological Sciences
Medicine and Science in Sports and Exercise
CRC Review of Bioengineering
National Science Foundation
National Institutes of Health

h. Abstracts in the Proceedings of Scientific Meetings

1. Liu, Y.K., "Application of Ultratranspherical Polynomials to Nonlinear Forced Oscillations," Proceedings 5th U.S. National Conference of Applied Mechanics, American Society of Mechanical Engineers, Minneapolis, MN, 1966, p. 144.

2. Liu, Y.K., "A Nonlinear Problem in Parametric Excitation," Proceedings of the Canadian Conference on Applied Mechanics, Canadian Congress of Applied Mechanics, Quebec City, Canada, Vol. 1, 1967, p. 1-192.

3. Liu, Y.K., "Towards a Stress Criterion of Injury," Proceedings Annual Conference on Engineering in Biology and Medicine, Alliance for Engg. in Medicine and Biology, Houston, TX, Vol. 10, 1968, p. 328.

4. Liu, Y.K., "On the Nonlinear Steady-State Vibrations with N-degrees of Freedom," Proceedings of the 2nd Canadian Conference on Applied Mechanics, Canadian Congress of Applied Mechanics, Waterloo, Ontario, Canada, 1969, p. 7.

5. Liu, Y.K., "Intracranial Pressure and Pressure Gradient in Head Impact," Proceedings 8th International Conference on Medicine and Biology in Engineering (ICMBE) and the 22nd Annual Conference on Engineering in Biology and Medicine (ACEMB), Alliance for Engineering in Medicine and Biology, Chicago, IL, July, 1969, Abs. 18-12.

6. Liu, Y.K. and Chan H.S., "Propagation of Pressure Waves in Head Impact," Proceedings 23rd Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Washington, D.C., Vol. 12, 1970, p. 60.

7. Liu, Y.K., "The Biodynamic Response of the Spine to Impact," Proceedings of the 24th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Las Vegas, NV, Vol. 13, 1971, p. 294.

8. Pontius, U.R., Liu, Y.K. and Van Buskirk, W.C., "Effect of the Cervical Neuromusculature on the Dynamics of Whiplash," Proceedings of the 25th Annual Conference of Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Bal Harbour, FL, Vol. 14, 1972, p. 239.

9. Cramer, H., Liu, Y.K. and von Rosenberg, D.U., "A Distributed Parameter Model of the Inertially-Loaded Human Spine," Proceedings of the Biomechanics Symposium of the American Society of Mechanical Engineers Joint Applied Mechanics and Fluid Engineering Meeting, American Society of Mechanical Engineers, Atlanta, GA, June, 1973, pp. 47-48.

10. Chan, H.S. and Liu, Y.K., "The Asymmetric Response of a Fluid-Filled Spherical Shell - A Mathematical Simulation of a Glancing Blow to the Head," Proceedings of the Biomechanics Symposium of the American Society of Mechanical Engineers Joint Applied Mechanics and Fluid Engineering Meeting, American Society of Mechanical Engineers, Atlanta GA, June, 1972, pp. 93-94.

11. Liu, Y.K., Wickstrom, J.K., Saltzberg, B. and Heath, R.G., "Subcortical EEG Changes in Rhesus Monkeys Following Experimental Whiplash," Proc. 26th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Minneapolis, MN, 1973, Vol. 15, p. 404

12. Lin, H.S., Liu, Y.K. and Adams, K., "The Response of the Intervertebral Joint under Complex Loading," Proceedings of the 27th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Philadelphia, PA, 1974, Vol. 16, p. 373.

13. Liu, Y., Varela, M. and Oswald, R., "The Localization of Some Important Acupuncture Points in Experimental Animals," Proceedings of the 7th Annual Meeting of The Neuroelectric Society, New Orleans, LA, November 20-23, 1974.

14. Liu, Y.K. and Chandran, K.B., "A One-Dimensional Experimental Model for Direct-Closed Head Impact," Proceedings American Society of Biomechanics Symposium, American Society of Mechanical Engineers, Troy, NY, June, 1975, pp. 149-152.

15. Liu, Y.K., Nikravesh, P.E. and Beck, C.H., "Optimal Protection in Direct-Closed Head Impact," Proceedings of the 28th Annual Conference of Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, New Orlean, LA, 1975, Volume 17, p. 167.

16. Ray, G., Liu, Y.K. and Davids, N., "Wall Stress in Curved Aorta in Blunt Chest Trauma," Proceedings of the 28th Annual Conference on Medicine and Biology, Alliance for Engineering in Medicine and Biology, New Orleans, LA, 1975, Vol. 17, p. 412.

17. Liu, Y.K. and Edisen, A.E.U., "Effects of Electroacupunctural Stimulation on the Evoked Potentials in the Thalamus and Cortex of the Cat," Proceedings 1st Annual Neural Science Symposium, Neuroscience Society, New Orleans Chapter, October, 1975.

18. Liu, Y.K., Lin, H.S., Nikravesh, P. and Wickstrom, J.K., "The Identification of Material Properties of an Intervertebral Joint Under Experimental Complex Loading," Proceedings of the 22nd Annual Meeting of the Orthopaedic Research Society, (ORS), New Orleans, LA, January, 1976, Vol. 1, p. 66.

19. Chandran, K.B., Liu, Y.K. and Wickstrom, J.K., "Stress Analysis of Spondylolisthesis," Proceedings of the 22nd Annual Meeting of the Orthopaedic Research Society (ORS), New Orleans, LA, January, 1976, Vol. 1, p. 68.

20. Ray, G., Liu, Y.K., Chandran, K.B., Ghista, D.N. and Sandler, H., "Potential Energy Distribution Prior to Ejection as a Basis for Post-Surgical Evaluation of Left Ventricular Efficiency," Proceedings of 29th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Boston, MA, 1976, Vol. 18, p. 126.

21. Liu, Y.K., Chandran, K.B. and Wickstrom, J.K., "In Vivo Dynamic Material Properties of the Canine Spinal Cord," Proceedings of the 29th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Boston, MA, 1976, Vol. 18, p. 48.

22. Ray, G., Liu, Y.K., Chandran, K.B. and Wieting, D., "Pressure-Gradient Changes in Experimental Acute Myocardial Infarction in Dogs," Proceedings of the American Society of Mechanical Engineers Symposium on Biomechanics, Yale University, New Haven, CT, June, 1977, p. 129-131.

23. Liu, Y.K., Jarrott, D., Chandran, K.B. and Krieger, K., "In Vivo Determination of the Cerebro-Spinal Specific Elastance in Cats," Proceedings of the 30th Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, Loas Angeles, CA, 1977, Vol. 19, p. 236.

24. Liu, Y.K. and Krieger, K.W., "Quasistatic and High Strain Rate Material Properties of Young Cervical Spines in Axial Loading and Bending," Proceedings of the First International Conference on Mechanics in Medicine and Biology, Aachen, Germany, Sect. 5, July, 1978, pp. 145-148.

25. Liu, Y.K. and Krieger, K.W., "High-Strain Rate Material Properties of Cervical Spines in Torsion," Proceedings of the 31st Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, 1978, Vol. 20, p. 255.

26. Liu, Y.K. and Krieger, K.W., "High-Strain Rate Material Properties of Human Cervical Spines in Shear," 25th Orthopaedic Research Society Meetings, San Francisco, CA, February, 1979, Vol. 4, p. 28.

27. Hosey, R.R. and Liu, Y.K., "A Finite-Element Model of Brain Damage in Head Impact," Proceedings of the 32nd Annual Conference on Engineering in Medicine and Biology, Alliance for Engineering in Medicine and Biology, 1979, Vol. 21, p. 131.

28. Hosey, R.R. and Liu, Y.K., "A Homeomorphic Finite-Element Model of Impact Head and Neck Injury," Proc. International Conference on Finite-Elements in Biomechanics, Tucson, AZ, February, 1980, pp. 851-870.

29. Liu, Y.K. and Njus, G., "Is Segmental Instability of the Lumbar Spine a Form of Fatigue Failure?" Proc. of the Conf. of International Society of the Study of the Lumbar Spine, New Orleans, LA, 1980.

30. Edisen, A.E.U. and Liu, Y.K., "Regional and Lateral Specificity in the Transfer of Electroacupuncturally Induced Analgesia by Blood Plasma," Federation Proceedings, 40, March, 1981, p. 284.

31. Liu, Y.K. and Gross, C., "A Method for the Rigid Fixation of an Accelerometer Package onto the Skull of Primates," Proc. of 9th Annual International Workshop on Human Subjects for Biomechanical Research, San Francisco, CA, September 1981.

32. Liu, Y.K., Tipton, C.M., Matthes, R., Bedford, T.G. and Walmer, H.C., "The Influence of Sodium Morrhuate Injections on Ligaments and Their Junctions," Abstracts of 25th Annual Osteopathic Research Conference, published in J. of the American Osteopathic Assoc., 80, 11, 1981, p. 764.

33. Liu, Y.K., Buckwalter, J., Njus, G. and Wakano, K., "Radiographic and Morphologic Correlates of Compressive Fatigue Failure of Lumbar Intervertebral Joints," Conf. of the International Society of the Study of the Lumbar Spine, Paris, France, 1981.

34. Liu, Y.K., Njus, G., Connors, M., Wakano, K. and Thies D., "Loading Rate Effects on Lumbar Intervertebral Joints in Tension, Compression and Shear," Proc. of 5th Annual Conference of The American Society of Biomechanics, Cleveland, OH, 1981.

Documents reviewed

Report by Mr. Edwards
Report by Dr. Turner
" " Dr. Marain
" " Dr. Gaume
" " Dr. Connors (Data compiled)
Composite of Troop compartment injuries.

DEFT. EX. DB- Liu Exh. 2
DATE: 11/30/81
REPORTER: ALBERT J. GASDOR

rehabilitation specifically provided by a multidisciplinary team is essential for the future well-being of the plaintiff. Dr. Novack will discuss the problems created by plaintiff's condition from the educational viewpoint and the different modes and necessities caused by this condition in relationship to the family structure. (45 minutes)

Y. King Liu, Ph.D.
Professor of Bioengineering
University of Iowa

The witness will testify in detail about his evaluation and research with monkeys indicating the different forces and movements that can cause brain injury from deceleration and impact. Dr. Y. King Liu will testify that the accident environment of the C5A deceleration and impact is reasonably simulated by laboratory environment produced by him and his research team in their evaluation of brain function in monkeys subjected to accidents. Dr. Liu will testify that in his opinion the injuries in monkeys subjected to impact and deceleration and the long-term effect of such injuries correlate the central nervous dysfunction (MBD) in humans. In describing his research in this field, Dr. Liu will display a movie produced by him on monkeys subjected to trauma. Dr. Liu will also discuss his consultation and evaluation of the accident environment of the C5A with Drs. Snyder, Busby and Mr. Cromack. (45 minutes)

DEFT. EX. DD- LIU #3
DATE: 11/30/81
REPORTER: ALBERT J. GASDOR

COMPOSITE OF

TROOP COMPARTMENT

INJURIES

[CREW]

NEILL, Harriet Goffinet:	Fractured clavicle
	Burns on ear
	Bruises all up and down backs of legs
	Lacerations - right leg and thigh
	Big hematoma on leg and under right arm
	Pulled muscles in back
TATE, Marcia Wirtz:	No physical injuries, but suffered from extreme fright -- remembers only one impact, so may have lost consciousness
AUNE, Regina:	4 broken bones in right foot
	Puncture wound on leg
	Decompression fracture of L-3 in back
	Laceration on elbow
	Cuts and scrapes
	Multiple black and blues
GMEREK, Gregory:	Fractured ribs
	Lacerations on forehead and chin
	Lost consciousness for a time
	Great difficulty breathing

PARKER, William	DIED: Chronic brain syndrome Fracture left femur Hemophorax left partial (See Certificate of Death, attached)
BOUTWELL, Olen:	Unknown, other than hurt ribs ("minor injury")[1]
DOUGHTY, Peter:	Unknown ("minor injury")[1]
PERKINS, Howard:	STATEMENT[2] is missing, but known to have dislocated right shoulder and knee
HADLEY, James:	No injury

[CIVILIANS]

ADAMS, Barbara:	DIED: Crushed and bruised chest Multiple bruises and lacerations all over body (See Autopsy Report, attached)
ADAMS, Linda:	Whiplash Torn knee pads, muscles stretched in both legs Cut on foot that left scar
THOMPSON, Thelma:	Full extent of injuries not known -- Received stitches but not known

DERGE, Susan:	Lost her ear
	Back broken in 4 places
STARK, Merritt, M.D.:	No apparent injuries
LIEVERMANN, Christie:	Multiple bruises and scratches on arms and legs
	Pulled muscles -- right leg and arm

[1] "Minor injury" : see "status" category of Tab 55 to Collateral Report (attached)

[2] Statement given to Collateral Board post-crash.

NAME	RANK	SSAN	CREW POSITION	LOCATION DURING RAPID DECOMP	LOCATION AT IMPACT	STATUS
TRAYNOR, DENNIS	CAPT	[redacted]	Pilot	Left pilot's seat	Same	Minor injury
HARP, TILFORD	CAPT		Copilot	Copilot's seat	Same	Minor injury
LANGFORD, JOHN	CAPT		Navigator	Navigator's seat	Same	Minor injury
ENGELS, ALLEN	TSGT		Flt Engineer	Flt Engr Seat	Same	Minor injury
MALONE, KEITH	CAPT		Pilot	Crew rest area	IP seat	Minor injury
MELTON, EDGAR	CAPT		Pilot	Crew rest area	Cargo compartment	Fatal injuries
WALLACE, WILLIAM	MAJ		Navigator	Crew rest area	Same	Minor injury
DIONNE, DONALD	SSGT		Flt Engineer	Cargo compartment	Thrown from aircraft at rapid decompression	Fatal injuries
McATEE, LYNN	MSGT	[redacted]	Flt Engineer	Cockpit	Crew rest area	Minor injury
DOUGHTY, PETER	TSGT		Loadmaster	Troop compartment	Same	Minor injury
AGUILLON, FELIZARDO	TSGT		Loadmaster	Cargo compartment	Same	Fatal injuries
PAYNE, WENDLE	MSGT		Loadmaster	Cargo compartment	Same	Fatal injuries
BRADLEY, PERCY	TSGT		Loadmaster	Cargo compartment	Crew rest area	Minor injury
SNEDEGAR, RAY	SMSGT		Loadmaster	Crew rest area	Same	Minor injury
PERKINS, HOWARD	SMSGT		Loadmaster	Aft ladder	Troop compartment	Dislocation right shoulder and knee
PARKER, WILLIAM	TSGT	[redacted]	Loadmaster	Troop compartment	Same	Fatal injuries
AUNE, REGINA	LT		Flight Nurse	Troop compartment	Same	Fracture right foot
WIRTZ, MARCIA	LT		Flight Nurse	Troop compartment	Same	Minor injury
GOFFINET, HARRIET	LT		Flight Nurse	Troop compartment	Same	Fracture clavicle
JOHNSON, DENNING	TSGT		Med Tech	Cargo compartment	Same	Fatal injuries
HADLEY, JAMES	SSGT		Med Tech	Troop compartment	Same	No injury
GMEREK, GREGORY	SGT		Med Tech	Troop compartment	Same	Fractured ribs
WISE, PHILLIP	SGT		Med Tech	Cargo compartment	Same	Concussion
BOUTWELL, OWEN	MSGT		Med Tech	Troop compartment	Same	Minor injury
KLINKER, MARY	CAPT		Flight Nurse	Cargo compartment	Same	Fatal injuries
PAGET, MICHAEL	SSGT		Med Tech	Cargo compartment	Same	Fatal injuries
CASTRO, JOE	MSGT		Photographer	Cargo compartment	Same	Fatal injuries
NANCE, KEN	SGT		Photographer	Cargo compartment	Same	Fatal injuries
WILLIS, WILLIAM	LTCOL		ACM	Cockpit	Cargo compartment	Fatal injuries

1. DATE OF OCCURRENCE (Day, month and year)	2. VEHICLE(S) TYPE, MODEL AND SERIAL NO. (designation and serial no. if applicable)	3. (Base Code and Report Serial No.)
4 April 1975	C-5A SN 68-213	--

4. PLACE OF OCCURRENCE: STATE, COUNTY, DISTANCE AND DIRECTION FROM NEAREST TOWN. IF ON BASE, IDENTIFY. IF OFF BASE GIVE DISTANCE FROM NEAREST BASE.	5. HOUR AND TIME ZONE LOCAL	6.
2NM NE OF RWY 25L TAN SON NHUT AB, RVN	1630 H	[X] DAY [ ] NIGHT [ ] DAWN [ ] DUSK

7. ORGANIZATION POSSESSING/OWNING VEHICLE OR MATERIEL AT TIME OF MISHAP

MAJOR COMMAND	SUBCOMMAND OR AF	AIR DIVISION	WING	GROUP	SQUADRON OR UNIT	NAME AND BASE OF
MAC	22AF	--	60MAW	--	--	TRAVIS AFB XDAT

8. (List organizations of second vehicle, if they differ from Item 7 above)

9. ORGANIZATION AND BASE SUBMITTING REPORT (Do not abbreviate)

MILITARY AIRLIFT COMMAND, SCOTT AIR FORCE BASE, ILLINOIS

10. LIST OF PERSONNEL DIRECTLY INVOLVED

(See AFM 127-2 and AFR 127-4 for specific instructions)

LAST NAME, FIRST NAME, MIDDLE INITIAL	GRADE	SSAN	ASSIGNED DUTY	AERO RATING	DEGREE INJURY[1] (Use Abbr)	DAYS LOST
TRAYNOR, DENNIS W.	CAPT	[redacted]	FP	PILOT	N	
HARP, TILFORD W.	CAPT	[redacted]	CP	PILOT	N	
MELTON, EDGAR R.	CAPT	[redacted]	ACM	PILOT	F	
MALONE, KEITH D.	CAPT	[redacted]	ACM	PILOT	N	
WALLACE, WILLIAM G.	MAJ	[redacted]	AN	SR NAV	N	
LANGFORD, JOHN T.	CAPT	[redacted]	NN	NAV	N	
MCATEE, LYNN F.	MSGT	[redacted]	FE	SR CM	N	
ENGELS, ALLEN R.	TSGT	[redacted]	FE	CM	N	
DIONNE, DONALD T.	SSGT	[redacted]	FE	CM	F	
PERKINS, HOWARD C.	SMSGT	[redacted]	LM	SR CM	TT	90
BRADLEY, PERCY D.	TSGT	[redacted]	LM	CM	N	
AGUILLON, FELIZARDO C.	TSGT	[redacted]	LM	CM	F	
DOUGHTY, PETER P.	TSGT	[redacted]	LM	CM	N	
PAYNE, WENDLE L.	MSGT	[redacted]	LM	SR CM	F	
PARKER, WILLIAM A.	TSGT	[redacted]	LM	SR CM	F	
SNEDEGAR, RAYMOND F.	MSGT	[redacted]	LM	SR CM	N	

[1] (Enter applicable letter(s) in DEGREE INJURY column. None-N; Temporary Total-TT; Permanent Partial-PP; Permanent Total-PT; Fatal-F; Missing-M)

11. NARRATIVE DESCRIPTION OF ACCIDENT: Give a detailed history of flight, or chronological order of facts and circumstances leading to the mishap, the results of investigation and analysis to include discussion of all cause factors listed, findings, and recommendations, and any corrective action taken.

ATTACHED

12. AUTHENTICATION

CERTIFICATION BY (Title)	TYPED NAME AND GRADE	SIGNATURE	DATE
INVESTIGATION BOARD PRES	NEWBY, WARNER E., MGEN	Warner E. Newby	23 May

MIDDLE INITIAL	GRADE	SSAN	DUTY	RATING	INJURY	FT ONLY

MAC Mission Observer:

Willis, William S.	LtCol	244-44-3250	MMO	Cmd Plt	F	

Medical Crew Members

Wirtz, Marcia	1Lt	[redacted]	FN	N	
Aune, Regina	1Lt		FN	TT	90
Wise, Philip	Sgt		MT	TT	120
Gnerek, Gregory	Sgt		MT	TT	14
Hadley, James A.	SSgt		MT	N	
Goffinett, Harriet	1Lt		FN	TT	21
Johnson, Denning C.	TSgt		MT	F	
Boutwell, Olen	MSgt		MT	N	
Klinker, Mary T.	Capt		FN	F	
Paget, Michael G.	SSgt		MT	F	

AAVS Photographers

Castro, Joe (NMI)	MSgt	[redacted]	ACM	F	
Nance, Kenneth E.	Sgt		ACM	F	

Passengers:

A manifest of passengers was received from the Defense Attache Office in Saigon. The manifest contained the names of 44 DOD personnel and dependents. Of the personnel on the manifest five are known to have survived the crash. An additional list of 10 attendants was received from the Defense Attache Office. Of these attendants, three are known to have survived the crash. There was no manifest of orphans aboard the aircraft; however, Saigon officials reported 247 were aboard. The aircrew interviews confirmed 145 orphans were located in the troop compartment of which 143 are believed to have survived. Approximately 102 orphans were located in the cargo compartment. Six were known to have survived. The chaos which followed the crash, the number of hospitals to which injured survivors were dispatched, the multi-agencies which accepted remains and the unstable political situation in the Republic of Viet Nam made a total accurate accounting impossible. The following is the final best estimate furnished by a representative from Air Force Military Personnel Center and concurred in by the investigation board.

	ON BOARD	SURVIVED	DECEASED
Flight Crew	16	11	5
Med Crew	10	7	3
Photographers	2	0	2
MAC Observer	1	0	1
Orphans (Troop Compt)	145	143*	2*
Attendants (Troop Compt)	7*	6*	1
Orphans (Cargo Compt)	102*	6	96*
Others (Cargo Compt)	47*	2	45*
Totals	330	175	155

Note: Asterisk indicates figures which were difficult to verify.

CASUALTY REPORT

The following data was compiled from crew testimony, existing manifests, and discussion with JCRC (Saigon) and Army pathologists at Camp Samae San, Thailand. It is believed that the figures given are very accurate although, in some categories, not precise (those with an asterisk). Imprecision of some of the figures was unavoidable despite the exhaustive effort of CWO Scott to compile this casualty/survivor list for several reasons:

a. There was no manifest for the orphans.

b. There was conflicting crew testimony in some categories.

c. Civilian attendants were moving about the aircraft while a head count was being done. Therefore, some may have been counted twice or not at all.

d. There were several American children on board and it could not be ascertained if they were counted as orphans or civilian attendants.

e. After the crash, the exact number of survivors could not be counted because they were hurriedly taken to many hospitals and orphanages throughout Saigon.

	ON BOARD	SAVED	DECEASED
1. Flight Crew	16	12	4
2. Med Crew	10	7	3
3. Photo	2	0	2
4. Observer	1	0	1
5. Orphans Troop Cmpt	145	143*	2*
6. Attendants Troop Cmpt	7*	6*	1

	ON BOARD	SAVED	DECEASED
7. Orphans Cargo Cmpt	102*	6	96*
8. Others Cargo Cmpt	47*	2	45*

1. Data concerring flight crew, medical crew, photographers, and observer are definitely correct.

2. The number of orphans in the troop compartment is exact as determined by crew testimony. However, there was the possibility that there were three rather than two fatalities.

3. The number of attendants in the troop compartment is an estimate according to the crew. The count was confused because some of the attendants were going to and from the cargo compartment. However, the figure seven is probably realistic. It was definitely ascertained that only one of the attendants sustained fatal injury.

4. JCRC Office in Saigon got figure of 247-253 orphans aboard. Crew was sure 145 were in troop compartment. Sgt Wise, survivor from cargo compartment believes at least over 100 children in cargo compartment. This figure (102) with six survivors appears realistic. Six survivors was count by crew.

5. Sgt Wise said at least 50 adults in cargo compartment. Some adults moved up and down from troop compartment. With number of adults manifested/nonmanifested thought to be aboard, 47 appears accurate.

6. Central Identification Laboratory has reported they have probable remains of 47/46 adults (excluding crew). They report having remains of 93 children with possibility of more. Difficult to determine at this time (18 April) because of status of remains.

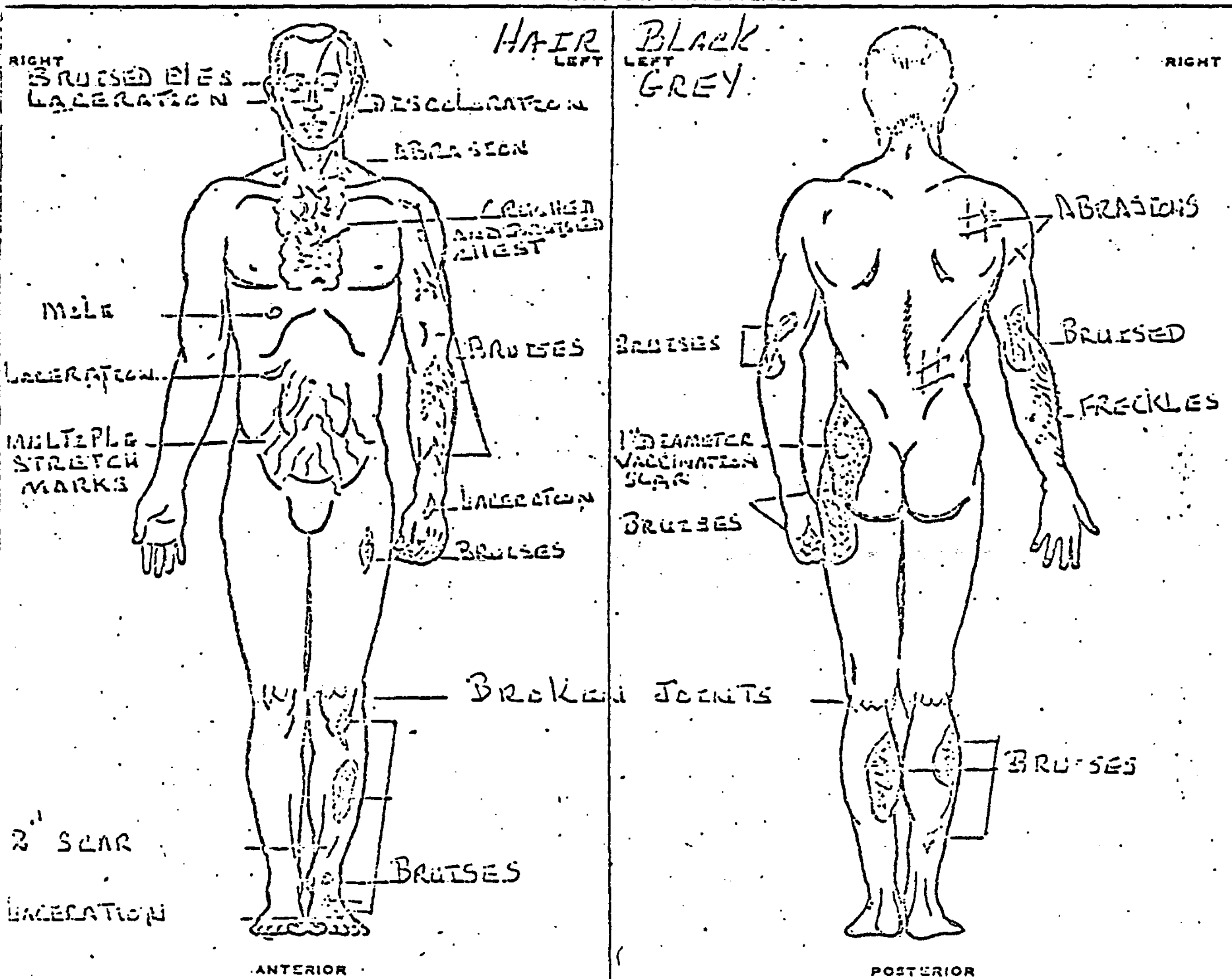
RECORD OF IDENTIFICATION PROCESSING
ANATOMICAL CHART

ICIL - 0082-75 | 10 APRIL 1975

NAME - FIRST NAME - MIDDLE INITIAL (or unknown number)	GRADE	SERVICE NUMBER
ADAMS, Barbara E.	DNC US CIV	292 21 9451

NAME OF CEMETERY, EVACUATION NUMBER, OR SEARCH AND RECOVERY NO.	PLOT	ROW	GRAVE	ESTIMATED AGE (Yrs)	ESTIMATED HEIGHT
					67 1/2

BLACK OUT PORTIONS NOT RECOVERED



CONDITION OF REMAINS (Check pertinent blocks)

[X] INTACT   [ ] DECOMPOSED   [ ] SEMI-SKELETAL   [X] FLESH COVERED   [ ] BURNED (Degree: [ ] 1st [ ] 2d [ ] 3d)

REMARKS (Continue on reverse if additional space is required)

Body - Complete
Race - Caucasian female
Est Ht. - Table measurement 67½"
ID tags - None received
ID card - None received
Clothing - None received

Both hands fingerprinted at USA CIL-THAI.
All prints match those on FD 258 for ADAMS,
Barbara E., [redacted]

NAME OF PREPARING OFFICIAL (Print or type): HENDERSON K. AHLO, SR.

SIGNATURE: Henderson K. Ahlo Sr

FORM 1 FEB 55 008

☆ GPO: 1969 O—343-753/577

INSTRUCTIONS: 1. Prepare in triplicate and distribute as follows:
a. Original to CCoI Cpt S, Attn: Mortuary Division.
b. Copy to Army Command.
c. Copy retained at preparing installation.

2. This statement will be supplemented by signed copies of appropriate Records of Identification Processing (DD Forms 890 through 894).

NAME OF DECEASED (Last, First, Middle)	GRADE	SERVICE NUMBER
ADAMS, Barbara Carol Emerson		[redacted]
BRANCH OF SERVICE	ORGANIZATION AND BASE	
US Civilian	DAO/Saigon, Vietnam	
DATE OF DEATH	PLACE OF DEATH	
4 April 1975	UTM XS 855 985, Go Vap Dist., Gia Dinh Prov., RVN	

CONDITION OF REMAINS (Describe briefly in Remarks)

X	RECOGNIZABLE		EVIDENCE OF DECOMPOSITION
	NOT RECOGNIZABLE	X	MANGLED OR MUTILATED
	COMMINGLED		EVIDENCE OF BURNS

MEANS OF IDENTIFICATION

(Check all appropriate boxes and indicate appropriate inclosures. Specify supporting data in Remarks.)

	IDENTIFICATION TAGS		INCLOSURES
	PERSONAL EFFECTS	X	DD FORM 890
	DENTAL COMPARISON	X	DD FORM 891 (if applicable)
X	SKELETAL AND ANATOMICAL COMPARISON	X	[illegible] DD FORM 893
X	FINGERPRINTS	X	DD FORM 894 and FD 258
	VISUAL RECOGNITION		
	OTHER (Specify in Remarks)		

REMARKS (If additional space is required, continue on separate sheet)

CIRCUMSTANCES:

On 4 April 1975, Barbara E. ADAMS was manifested aboard the C-5A Galaxy flight from Tan Son Nhut Air Base in Saigon, RVN to CONUS. At 1640 hours (local) the aircraft departed TSN with 77 manifested US passengers and creumembers aboard. The aircraft experienced inflight trouble approximately twenty minutes out and tried to return to TSN Airbase for an emergency landing. The aircraft crashed approximately 2 miles NE of TSN Airbase.

On 5 Apr 75, 76 human remains pouches were received in two increments and each pouch assigned a consecutive evacuation number. These remains were assigned THCIL Evac No 0082-75 and when received the pouch was tagged BTB Barbara E. ADAMS.

CONDITION OF REMAINS:

These are the complete remains of an adult Caucasian female exhibiting multiple bruises, lacerations and a crushed chest.

PRIMARY METHOD OF IDENTIFICATION:

The physical and dental anatomy of these remains were thoroughly studied and charted on the applicable forms. The race, height, and hair color compare favorably with the recorded physical data for Ms. ADAMS (See Summary).

Dental records are not available for comparison.

Post mortem fingerprints were taken from these remains at this command. Fingerprints match those on FD 258 for ADAMS, Barbara E.

TO THE BEST OF MY KNOWLEDGE AND BELIEF, THE STATEMENTS MADE HEREIN ARE CORRECT AND TRUE.

DATE	TYPED NAME, GRADE AND TITLE OF IDENTIFYING OFFICER
17 April 1975	JOHN G. ROGERS, DAC, GS-12, JCRC, AREA DESK OFFICER
	SIGNATURE OF IDENTIFYING OFFICER [signature]

DEPARTMENT OF THE AIR FORCE
WASHINGTON, D.C. 20330

REPORT OF CASUALTY

1. REPORT NUMBER AND TYPE: R-159 - Final
Completes Report Number R-137

2. DATE PREPARED: 6 May 75

3. SERVICE IDENTIFICATION (Name, Social Security Number, Grade or Rate, Component, Branch)

PARKER, William Monroe, FR [redacted] TSgt, Regular, USAF

4. CASUALTY STATUS a. [ ] BATTLE [X] NON-BATTLE b. COMMENCED TOUR DATE

c. STATUS: [X] DEATH [ ] KIA [ ] MISSING [ ] MISSING IN ACTION [ ] CAPTURED [ ] OTHER _____

d. DATE: 21 Apr 75 e. PLACE Tripler Army Med Ctr, Honolulu HI

f. CAUSE Injuries received as a result of military aircraft accident (C-5A Loadmaster)

5. a. DATE AND PLACE OF BIRTH	b. RACE	c. SEX	d. RELIGIOUS PREFERENCE
11 Dec 38 - Caddo OK	N/A	N/A	N/A

6. DATE AND PLACE OF LAST ENTRY ON ACTIVE DUTY IN CURRENT STATUS AND HOME OF RECORD AT TIME
N/A

7a. PAY GRADE	b. BASIC PAY	c. INCENTIVE/ADDITIONAL PAY	d. CHECK IF APPLICABLE
E-6	N/A	[ ] YES [ ] NO N/A	[ ] CREW [ ] PASSENGER

8. DUTY STATUS
Retired - Physical Disability (Chronic brain syndrome; Fracture right femur; Hemophorax left partial) effective 17 Apr 75#

9. INTERESTED PERSONS (Name, Address, Relationship)

Mrs Shirley J Parker	1318 Marshall Rd, Vacaville CA 95688	wife
Gina I Parker	same	dau
Connie J Parker	same	dau
Christie G Parker	same	dau
Randall J Parker#	same	son*

10. REPORT FOR VA TO FOLLOW	11. REPORTING COMMAND AGENCY AND DATE REPORT RECEIVED IN DEPARTMENT
[ ] YES [X] NO	HICKAM AFB HI - 22 APR 75

12. PRIOR SERVICE DATA
[X] YES [ ] NO AF18509745

13. REMARKS

*Mrs Minnie E Parker 913 E Stanford, Lubbock TX 79403 mother

SGLI: BY LAW LUMP SUM $20,000

NOTE: THIS FORM MAY BE USED TO FACILITATE THE CASHING OF BONDS, THE PAYMENT OF COMMERCIAL INSURANCE, OR IN THE SETTLEMENT OF ANY OTHER CLAIM IN WHICH PROOF OF DEATH IS REQUIRED

FOOTNOTES:
1. Adult next of kin.
2. Beneficiary for gratuity pay in event there is no surviving wife or child—as designated on record of emergency data.
3. Beneficiary for unpaid pay and allowances—as designated on record of emergency data.

#Indicates change

14. DISTRIBUTION
NOT USED

15. BY ORDER OF THE SECRETARY OF THE AIR FORCE

Dean M. Murphy

DEAN M. MURPHY, Lt Colonel, USAF
Ch, Casualty Rprtg & Survivor Assistance Br

DEPARTMENT OF THE AIR FORCE · OFFICIAL · HEADQUARTERS U.S. AIR FORCE

DD FORM 1 FEB 73 1300

REPLACES DD FORM 1300, 1 MAR 60, WHICH IS OBSOLETE.

Revised Version

A16

SUBCORTICAL EEG CHANGES IN RHESUS MONKEYS FOLLOWING EXPERIMENTAL HYPEREXTENSION-HYPERFLEXION (WHIPLASH)†

Y. King Liu*

K. B. Chandran*

R. G. Heath**

F. Unterharnscheidt***

*Biomedical Engineering Program, The University of Iowa, Iowa City, IA 52242

**Psychiatry and Neurology, Tulane University School of Medicine, New Orleans, LA 70112

***Naval Aerospace Medical Research Laboratory, Michoud Station, New Orleans, LA 70129

†The work reported herein was performed while all authors were members of the faculty of Tulane University School of Medicine, New Orleans, LA 70112. The first author was a NIH Career Development Awardee (Grant No. GM 40723-05) during part of this investigation. The support provided by the National Institutes of Neurological and Communicative Diseases and Stroke (Grant No. NS GM 10517) is gratefully acknowledged.

Running Title:

SUBCORTICAL EEG CHANGES IN POSTWHIPLASHED RHESUS

Key Words:

Whiplash, Rhesus, Electroencephalography, Electrocorticogram, Hippocampus, Spiking

Abbreviations:

EEG - electroencephalogram; ECoG - electrocorticogram; EMG - electromyogram; RETIC - reticular formation; A SEP - anterior septum; CBL FAS - cerebellum fastigus nucleus; HIP - hippocampus; L - left; and R - right

Please send proofs to:

Y. King Liu, Ph.D.
Biomedical Engineering Program
College of Engineering
The University of Iowa
Iowa City, IA 52242

ABSTRACT

A controlled study, involving EEG recordings from the scalp and chronically implanted electrodes in the cortex (ECoG) as well as from selected subcortical nuclei, was undertaken to investigate the neurophysiologic effects on rhesus monkeys following experimental whiplash (hyperextension of the head and neck). Sixteen animals, equally divided into four groups, were studied through the following protocol: two animals within each group were whiplashed and then deep electrodes were implanted into the brain while the second two animals were implanted with deep electrodes and then one was whiplashed after healing. Weekly EEG follow-ups showed hippocampal spiking in 3 of the 4 whiplashed and then electrode-implanted animals and in 1 of the 4 electrode-implanted and then whiplashed animals.

Several results deserve attention. (1) Prior to the onset of spiking, i.e., 6 to 8 weeks post-whiplash, practically all scalp, cortical and subcortical EEG recordings were normal. (2) When hippocampal EEG spiking did take place, only normal and mildly abnormal changes were seen in either the ECoG or scalp EEG. (3) The growth and development of this trauma-induced hippocampal spiking followed the classic sequence for the spread of an epileptogenic focus. This subclinical form of post-traumatic epilepsy may be a combined phenomenon, i.e., the whiplash plus the subcortical electrode placements further decrease the already well-known low-spiking threshold of the hippocampi.

INTRODUCTION

After a front-to-rear automobile collision, the occupants of the automobile hit from behind may suffer a "whiplash injury", a term attributed to Crowe (1958) to describe an injury to the neck in such accidents. Phenomenologically, the term comes from a description of the violent motion of head and neck in which the head is suddenly thrown backward resulting in a hyperextension of the neck (the whip phase) and immediately afterwards is recoiled in the opposite direction forcing the neck into hyperflexion (the lash phase). The use of the term "whiplash injury" by Braaf and Rosner (1963) and States et al. (1970) have been critized by other authors, e.g., Hamel and James (1962) and Farbman (1973), because it described the motion rather than the pathology. In spite of the criticism, the term is entrenched in the lay, medical and legal literature. Whiplash will be the term used hereafter in this paper to describe this special type of head and neck injury instead of the more cumbersome "hyperextension-hyperflexion injury".

The clinical problem associated with whiplash injury has been well stated by McNab (1969):

> Everyone is well acquainted with the common story associated with whiplash injuries. Despite the history of pain the the neck, pain between the shoulder blades, pain the back of the head, pain down the arm, dysphagia, tinnitus, vertigo, and intermittent blurring of vision, there is remarkably little to find on examination. The usual x-ray films do not commonly show any gross abnormalities, even though there may be an alteration in the normal cervical curve. Despite the frustratingly negative examination, the patient's aches and pains fail to respond to the Brownian movements of routine medical therapy. As the months roll by, it becomes increasingly apparent that the patient is grossly emotionally disturbed.
>
> There is general agreement on the type of symptoms to be expected, but widely divergent views are held on the significance of these symptoms. These divergent viewpoints, rigidly held and hotly contested, are firmly based on impressions only. In an endeavor to make these impressions more factually significant, the progress of 575 patients has been carefully followed. Many physicians believe

that the 'whiplash syndrome' is demonstrated only by a group of hysterical, neurotic, if not frankly dishonest, people. However, there are certain disturbing features apparent from an analysis of the case histories available that make it difficult to accept the belief that litigation neurosis is the sole explanation of the long drawn out disability in every instance.

He proceeded to show, quite conclusively, that two or more years after settlement of the court action, 121 out of 266 or 45% continued to have symptoms.

In a recent paper using insurance claim data, O'Neill et al. (14) have stated, that despite some reduction in head and neck injuries due to the requirement of head restraints, the number of whiplash injuries in the U.S. may be "considerably in excess of one million." The reason given for the limited success of what appears to be a rational solution for the whiplash problem is that the adjustable head restraints were usually improperly positioned. The authors warned:

an adjustable head restraint if left in its lowest position actually may increase the chances of severe whiplash injuries by acting as a fulcrum for the head, especially for taller people.

Torres and Shapiro (17) have investigated the EEG of post-whiplashed patients. In a population of 45 patients, 46% had electrical abnormalities of moderate or marked degree while the rest had either mildly abnormal or normal EEGs. Those patients who had more than one EEG showed progressive deterioration instead of improvement in their follow-up records. The authors concluded that:

It is highly improbable that the association of these abnormalities, of higher degree and much higher incidence than any series of normals, with the history and clinical findings of whiplash injury might be exclusively due to chance factors. We conclude, therefore, that the electrical abnormalities were probably caused by the injury.

Gibbs (5) has reassessed the use of clinical EEG as an objective indicator of brain disorder in whiplash injury. He compared the percentage incidence of abnormal findings among 178 medicolegal cases of whiplash with

the percentage incidence of these same EEG findings among asymptomatic control subjects. He found that 11% had EEGs with focal slowing and 5% had paroxysmal slowing, two abnormalities almost never found in asymptomatic control subjects. Though age related, practically all the other abnormalities are found in whiplashed patients as well as asymptomatic controls. It should be emphasized, however, that the incidence of such abnormalities in the asymptomatic controls are found less frequently than in the whiplashed patients. In the adult group between ages of 20 to 60, which accounts for 148 of the 178 cases studied, approximately 65% of these patients with symptoms show normal EEG. Gibbs (5) has further contended:

> A patient who has symptoms and whose EEG shows mild abnormalities might deserve consideration, even though his complaints and abnormalities are the same as those of many other persons.

Scalp EEG work based on animal studies has been reported by Wickstrom et al. (22) Thirty-six EEGs were recorded from rhesus monkeys before and after controlled whiplash. The recorded EEGs were interpreted by an electroencephalographer. Of the 36, 7 had abnormal EEGs. Of the 7 with abnormal EEGs, 6 also had abnormal behavioral symptoms under clinical examination, yet only 4 had identifiable histopathology after necropsy. Of the remaining 29 with "normal" EEGs, examination subsequent to sacrifice revealed that 3 had spinal cord lesions, 2 had brain injuries and 2 others had both brain and spinal cord pathology. In short, approximately 24% of the monkeys with definite neurohistological lesions had no EEG abnormalities by the simulated clinical procedure. Thus, the abnormal scalp EEG is a rather poor indicator of nervous tissue injury. Conversely, the presence or absence of neurohistological lesions cannot be depended upon as the indicator of EEG abnormality either.

Koshino et al. (9) have utilized megimide activation of the EEG to study whiplash and head injury patients with stubborn subjective symptoms, but who showed neither objective abnormalities of the neck nor abnormal clinical EEG. In a clinical study involving 35 whiplash and 45 head injury patients, it was found that 57% of the whiplash group and 73.3% of the head injury group had abnormal activated EEGs after injection of less than 100 mg of megimide. The mean doses of megimide required for the activation of irregular spike and wave complex in the whiplash and head injury groups were 80 mg and 72 mg respectively. Wada (1966) has shown that the threshold of megimide activation of EEG in the normal person is more than 150 mg, and generally an abnormality is indicated if a threshold of less than 100 mg of megimide activated the EEG. Furthermore, the activated abnormal EEGs were provoked at almost the same rate irrespective of the duration from the time of injury to that of examination.

As far as we are aware, the only previous study of subcortical EEG changes following experimental whiplash was the work of Tsuchiya et al. (18) Rigid electrodes were implanted into the following areas of the rabbit brain: neocortex, hippocampus, premammillary area, internal capsule, preoptic area, lateral hypothalamus, ventromedial hypothalamus, central gray matter, and the reticular formation. The results can be summarized as follows:

1) In the neocortex, low amplitude fast waves which were observed immediately after the impact returned almost to the control pattern within 2 to 6 days,

2) In the hypothalamus, normal EEG pattern was observed soon after trauma. However, abnormal EEG, such as the high amplitude slow waves or spikes, were observed 7 to 45 days after the impact and continued for some period. The regions from which

the abnormal waves were recorded were: the medial preoptic area, ventromedial hypothalamic nucleus, posterior hypothalamus, premammillary nucleus, lateral hypothalamic nucleus and lateral preoptic area.

3) In the midbrain reticular formation, sporadic sharp spikes were observed within 2 to 3 days after the whiplash injury, with the EEG apparently returning to normal thereafter.

Besides the obvious objection to the use of rigid electrodes, the experiments of Tsuchiya et al. (1968)[18] were incompletely controlled. The combination of rigid electrodes implanted in the subcortex together with a high rotational acceleration during whiplash obviously contributed to the injury and the subsequent EEG changes. This illuminates the need for improved controls in spite of this study's importance and originality. This study by Tsuchiya, et al. (1968)[18] has laid the difficult foundations upon which our research is based.

1. Experimental Design

The experimental protocol called for the use of four matched groups of rhesus monkeys of the same sex, each weighing about 4 kgs. The study began with at least two baseline scalp EEG recordings per animal. In each series, after ascertaining the scalp EEG to be normal, two of the four animals were whiplashed in the acceleration facility. After the post-whiplash baseline scalp EEG was recorded, one (Animal A) of the above two experimental animals was randomly chosen to act as the scalp control, while the other had flexible electrodes implanted in its brain (Animal B). The third (Animal C) had electrodes implanted in its brain and then whiplashed after the wounds were allowed to heal. The fourth (Animal D) had electrodes implanted in the same sites to act as the electrode control for the series. The protocol is summarized as follows: Animal (A), whiplashed and scalp EEG followed; Animal (B), whiplashed and then followed by deep electrode implantation in the brain; Animal (C), deep electrode implantation in the brain and then whiplashed; and Animal (D), deep electrode implantation in the brain only.

One of the main concerns in the above experimental protocol was: how can one be reasonably sure that, for Animal (C), the whiplash head injury was not the result of presence of deep flexible electrodes in the brain interacting with the high angular acceleration during the whiplash? To insure that the flexible electrode used did not excessively damage the brain during the acceleration pulse, we simulated our experiment in a gel-filled transparent half skull. Electrodes were inserted into the gel as during in vivo implantation into the brain. Indian ink was then injected into the

electrode track until it was filled. A small plastic wedge was used to fix and seal the electrode at the skull, just as used in an actual implantation procedure. The ensemble was whiplashed in a similar fashion as in a test, although the angular acceleration levels achieved were much lower. The procedure was recorded on high speed film and then analyzed. No (Indian) ink left the tracks.

2. Flexible Electrode Implantation

The stereotaxic brain implantation was done in the manner of Heath (7) using flexible bipolar electrodes with silver-balled tips of approximatly 0.5 mm diameter and located 1 mm apart. The electrode implantation was performed under approximately 120 mg of phenobarbitol administered i.v. Because patients with whiplash symptoms very often become emotionally disturbed with time, we postulated the probable involvement of the limbic system, which has been theorized by Papez (5), Maclean (10), Nauta (13), and Zanchetti (23), as the regulator of emotional expression. Heath (8) has reported on data collected over a 20-year period which showed functional connections between certain sensory relay nuclei (ventro-postero-lateral nucleus of the thalamus and the fastigius nucleus of the cerebellum) and brain sites (primarily the septal region and hippocampus), where physiologic activity has been shown to be correlated with emotional expression. Thus, the deep sites chosen for chronic electrode implanation were: the left and right hippocampi (L HIP and R HIP), right reticular formation (R RETIC), right anterior septum (RA SEP) and right cerebellum fastigius nucleus (R CBL FAS). Epidural cortical leads were placed over the frontal, temporal and occipital cortex, to obtain the electrocorticogram (ECoG). The scalp leads used were F7, T3, T5, T8, T4, and T6, i.e., the midtemporal chain in the international

10-20 system. The animals received antibiotics upon completion of their electrode implantation to minimize infection at the surgical sites. Ten days to two weeks were allowed for wound healing.

3. Hyperextension-hyperflexion Trauma

On the day of the whiplash, the animal was tranquilized with phencyclidine hydrochloride (Sernylan® 2 mg/kg) intramuscularly. The torso of the rhesus was then restrained with a jacket and placed sitting on a miniature cart with its head and neck unrestrained. Briefly, the experimental setup consisted of a compressed air pump and reservoir connected to a 152 mm diameter circular cylinder with an associated piston having a 914 mm stroke length. The magnitude of the acceleration exerted on the cart was controlled by the pressure of the compressed air in the reservoir. The duration of acceleration of the vehicle can be varied somewhat by the placement of polyurethane pads of different thickness onto the impacting piston. During the acceleration phase, the cart triggers three pulsed x-ray units (Fexitron No. 730 Hewlett-Packard Corp.) through microswitches to record the changing relationship during the various stages of the hyperextension of the head and neck. A typical radiograph of an animal implanted with deep electrodes being whiplashed is shown in Figure 1. The event is also recorded on a Hycam (Redlake Corp.) high speed camera at 1,000 frames/sec against a preset grid. The acceleration procedure outlined above was previously reported in Domer et al. [3].

4. Data Acquisition

Whenever possible, at least one EEG per week per animal was monitored and tape recorded. The EEG technician kept a continuous log of each animal

during the recording session. Information, such as the beginning and end of sleep, wakefulness, and any abnormalities during these recording sessions was carefully noted and correlated to a time-code generator signal in order to facilitate the later signal analysis.

RESULTS

Typical cart and linear accelerations of the skull as well as the computed angular accelerations have been illustrated previously in Domer et al. (1979)[3]. In the interest of continuity, the numerical results are reiterated: (1) The maximum linear acceleration of the cart was about 35 g's, occurring at about 30 msec after the onset of the impact. (2) The maximal calculated angular acceleration, achieved during hyperextension, was approximately 40×10^3 rad/sec^2 which occurred 50 msec after impact.

None of the monkeys lost consciousness following the whiplash as evidenced by the lack of eye closure and the maintenance of muscular tone. Furthermore, no blood issued from any orifice.

A synopsis of the electroencephalographic changes, if any, for all of the animals in the 4 series is given in Table I.

The animals with hippocampal spiking are: I-B, II-B, IV-B and IV-C. The time course of development of the hippocampal spiking activity in all 4 animals warrant individual attention and are summarized below:

Follow-up on Animal I-B

Two weeks post-whiplash, this animal developed occasional paroxysmal focal slowing during light sleep in F4-C4 and F3-C3 scalp leads. Since focal slowing is a form of scalp EEG abnormality that is almost never seen in unwhiplashed rhesus monkeys, we chose this animal for depth-electrode implantation on the notion that the depth correlates of the surface phenomena could be found. The electrode implantation took place 3 weeks post-whiplash. Two weeks were allowed for the surgical trauma to heal before the first

post-implant (5th week post-whiplash) recording was taken and shown in Fig. 2. Note the absence of abnormalities in the scalp, cortical and depth leads. Figures 3 through 6 recorded the serial events as the weeks passed. Figure 3 illustrates the incipient spiking activity in the right hippocampus 6 weeks post-whiplash during light sleep. The frontal cortical lead, which showed no abnormality, was dropped to make room for the right hippocampus in the first 6 channels. This was done to ease the signal analysis problem anticipated, i.e., the problem of detecting the subcortical spikes at the scalp. By the 8th week, shown in Fig. 4, the left hippocampus had developed a mirror focus. Figure 5 shows that during the 11th week, the left hippocampal activity during sleep was less dependent on the original right hippocampal focus, indicating the near completion of the epileptiform mirror focus. The temporal cortical lead was dropped to make room for the R HIP lead. By the 13th week, the right hippocampus of the animal was in complete "seizure-like" activity but only during sleep. By the 16th week, both hippocampi were continuously spiking practically at all times. Figure 6 is taken from the record of the 18th week showing continuous spiking acitivity in the hippocampi. Weekly tape-recordings continued until the 27th week when the animal knocked out its plugs by accident. The animal was sacrificed at this time. The last recording made showed a slight diminution of the spiking activity.

Follow-up Details on Animal II-B

This animal was whiplashed and then its subcortex implanted with electrodes two days later. Its first EEG record was taken 2 weeks after trauma. The 7th week post-whiplash record showed, during sleep, an occasional single spike in

the left hippocampus. When compared to the corresponding record (8th week post-whiplash) in the previous series shown in Fig. 4, the amplitude of the spikes are of equal magnitude but the frequency of occurrence is much lower. The frequency increased in the next two weekly recordings and reached a maximum of about 3 to 5 spikes for each ten seconds of sleep record as shown in Fig. 7. No mirror focus developed in the right hippocampus nor did the spiking reach the "seizure" level found in Series I.

Follow-up Details of Animal IV-B

This animal was whiplashed and its subcortex implanted with electrodes 10 days later. The first post-implant record was taken 10 days following electrode implantation. The 8th week post-whiplash record showed sharp waves and an occasional spike in the right hippocampus. The activity increased in the right hippocampus as the weeks passed and by the 11th week, a mirror focus had developed in the left hippocampus. By the 18th week, both hippocampi were showing continuous spiking activity. The recordings were continued for 52 weeks with only a slight attenuation in activity. The results were very similar to animal I-B.

Follow-up Details of Animal IV-C

This animal was implanted with electrodes and then monitored for 16 weeks and no abnormalities developed in the EEG. The animal was whiplashed at this point. The post-whiplash recordings were started 4 days later. Seven weeks post-whiplash, the record showed random sharp waves and occasional spikes in the left hippocampus with delta and theta wave activity in the right hippocampus. By the 10th week, the left hippocampus had

developed random bursts of sharp waves and spikes and the right hippocampus had developed a mirror focus. By the 14th week, both sides were in complete "seizure-like" activity with the left hippocampus slightly more active. Continuous spiking activity occurred in both hippocampi thereafter. This was the first C type animal which developed spiking activity. The results were again very similar to animal I-B.

Histologic Controls and Pathological Findings

A standard intracardiac perfusion-fixation technique was used at the time of sacrifice. A laminectomy was performed on the cervical spine to expose the cervical spinal cord. A craniotomy was next performed and the brain and spinal cord removed in toto. Any gross pathology was described and photographed. Following coronal section, the blocks were embedded in celloidin. The sections were stained with either hematoxylin-eosin or cresyl-violet or both techniques. The histological evaluation was done in the manner of Unterharnscheidt and Higgins (19). In our evaluation, special attention was given to the exact locations of the electrode tracts and their endpoints described. The neurohistology has been completed in Series I and II.

All animals showed traumatic lesions associated with the electrode implantation. In the interest of brevity, only the microphotographs associated with the spiking nuclei are displayed. Figures 8 and 9 show the right and left Ammon's horn formation in animals I-B and II-B respectively. In Fig. 8, the endpoint of the electrode tract in Sommer's sector (h_1) is labelled a. Only a few neurons are visible in the region surrounded by the arrows. Figure 9 shows typically the marked local glial and mesenchymal reaction surrounding the electrode tract labelled a. All the neuro-

histology completed thus far confirms the electrode placement for all 8 animals. There were no primary or secondary traumatic lesions elsewhere in the brain or spinal cord.

population of rhesus done by Wickstrom et al. (1969)[22] did show a few such lesions.

Walker et al. (1944)[21] have described experimental results to support the concept that the traumatic unconscious state is very similar to the aftermath of a generalized epileptic convulsion. Subcortical spiking at localized nuclear sites of the brain, however, seldom leads to a clinical seizure. In the rhesus monkey brain, with our particular depth leads, the point has been made by Heath (1972)[8]:

> In some experiments (with cobalt induced epileptiform activity), when these epileptiform discharges spread to encompass the entire brain, the monkey had a clinical epileptic seizure. Behavioral changes were minimal or absent when the epileptiform discharges were limited to one or more nuclear sites without affecting the septal region. But when the septal region was involved, either alone or in concert with epileptiform activity at other deep nuclear sites, the monkeys invariably displayed gross behavioral aberrations in the form of impaired emotionality and reduced awareness which progressed to catatonia.

No gross motor behavioral changes, such as clinical convulsions, were observed in any of the 4 animals even when the spiking discharges in the hippocampi were continuous.

The mirror focus phenomenon, seen in animals I-B, IV-B, and IV-C have been seen in all experimentally induced epilepsy using different agents on the cortex. Morrel (1959)[12] has characterized the growth and development of the mirror-focus as being similar to the learning curve. The growth and development of our trauma-induced hippocampal focus closely parallels an artificially induced local epileptiform activity. Thus, the hippocampal spiking exhibited by whiplashed primates might be considered as a subclinical form of post-traumatic-epilepsy.

The hippocampal spiking activity is most probably an additive phenomenon, i.e., the combination of the whiplash and the traumatic presence of the

DISCUSSION

Numerous means have been found to induce epileptiform activity in the brain. These include a range of different agents such as cobalt, penicillin, metrazol, ethylene chloride, dry ice and others. As far as we are aware, this is the first time that subcortical epileptiform activity has been exhibited in a controlled whiplash primate experiment. Post-traumatic epilepsy occurs in 10% of all severely closed head-injured patients with a clinical diagnosis of contusion. In the present experiment, the physical event initiating trauma was a whiplash movement of the head and neck and not a direct-contact impact of the scalp and skull. If one accepts cerebral concussion as the dramatic, sudden loss of consciousness as a result of trauma, then none of the whiplashed animals in this study were concussed.

Torres and Shapiro (17) have compared the clinical EEG of 45 head injury patients with 45 patients suffering from whiplash injuries. In their patient selection procedure, they showed that while both groups had the same subjective complaints, there was one major difference: 23 of the patients with contact head injuries suffered from cerebral concussion, or amnesia but this was totally absent in the whiplashed group. Thus, the fact that our animals were not concussed is, therefore, not surprising. Traumatological investigators differ widely on the question of whether traumatic unconsciousness and whiplash injuries are accompanied by histologic changes. Our neurohistological findings for Series I and II show no definite primary or secondary traumatic lesions as a result of our measured whiplash input although previous results on a much larger

electrode is enough to drive the sensitive neuronal system and/or the neurons themselves, beyond its spiking threshold; whereas, each by itself is insufficient to accomplish the same. The low spiking threshold of the hippocampus is well-known and as such it is not surprising that it is the only site in our experimental study to exhibit such a phenomenon.

When subcortical spiking does occur, why is it confined mainly to the animal which was whiplashed and then had electrodes implanted in its brain, i.e., animal B in each series? We sought the answer to the above question through its control, animal C, which had electrodes implanted in its brain and then whiplashed. The only difference between B and C, other than the normal variations of the repeatability of the experiment, is the presence of the deep electrodes during whiplash. Our tentative conjecture is that the presence of the electrodes tether the brain, so that the relative motion between the skull and brain is less than would be the case if the electrodes were absent. The brain material is almost incompressible with a bulk modulus K = 2.07 giga pascals (300,000 psi) and a Young's modulus E = 68,900 pascals (10 psi). On the other hand, its shear modulus is 1,378 pascals (G = 0.2 psi). In whiplash, the mechanical input is primarily a rotational acceleration and secondarily a translational acceleration of the head and neck. Both inputs induce a graded set of shear strains, maximum at the subdural cortex and decreasing towards the diencephalomesencephalic core. The flexible electrodes, which are anchored by plastic wedges on the skull, have an order of magnitude higher shear modulus. The brain, being a viscoelastic material, begins to compress the implanted electrode soon after insertion. The frictional forces generated are such that no discernable movement of the electrodes were detected

from either the flash x-rays or from the neurohistology subsequent to sacrifice.

When the spiking activity was continuous within the hippocampi, the EEG activity of the remaining scalp leads, which simulated the clinical EEG, was only mildly abnormal or normal. However, if the scalp EEG records from Figs. 2 through 6 were read serially, it is possible to discern a progressive deterioration. This mild abnormality is in agreement with the results of Torres and Shapiro (17) on human whiplash patients.

In spite of the relatively small experimental sample size, some conclusions can be drawn from the above reslts. The most important point to be made concerns the number of electrode controls, i.e. D animals, which on the basis of the experimental protocol given above is only four. In fact, however, Heath (8) has implanted deep electrodes into the various nuclei of the limbic system of the rhesus over the past 20 years (using the same technique as described here). In over 200 animals in which our chosen sites were implanted as electrode controls, none have developed subcortical spiking during long-term follow-up using identical electrodes and procedures as in the present study. If in 200 normally implanted animals no spiking occurred, but that as a result of the given whiplash, 3 our of 4 of the B animals and 1 out of 4 C animals developed spiking, then heuristically, one would assume that the whiplash episode was a significant event.

It is premature to come to any definite conclusions concerning the interesting neurohistologic lesions observed. Many more extensive and detailed experiments are needed before one can decide whether these lesions are caused by the whiplash or electrode trauma or one additively or synergistically acting on the other. The neurohistology will constitute the content of a future paper.

CONCLUSION

1) The immediate post-whiplash scalp EEG can be considered as a normal baseline recording if free from any previous abnormalities. So far, when the whiplash animals do develop abnormalities, they do so at least 6 weeks post-whiplash.

2) When subcortical EEG changes did take place, only normal or mild abnormal tracings were observable in either the ECoG or the scalp EEG during the same time period in this series of experiments.

3) The spiking activities in the hippocampi occurred mainly in animals which were whiplashed and then had electrodes implanted in their brains, i.e., B animals. The presence of spiking activity was observed in only one animal which had electrodes implanted in its brain and then whiplashed. These results suggest that not only is there minimal interaction between the brain matter and the flexible electrodes due to the whiplash but that these electrodes may have had a tethering effect in reducing the rotation displacement of the brain.

4) The hippocampal spiking activities is most likely a combined phenomenon, i.e., the spiking is the result of either the additive or the synergistic combination of the invasive presence of the recording electrodes and the hypersensitivity of the hippocampi to whiplash. Previous subcortical electrode implantation studies (by one of the authors (R.G.H.)) in more than 200 rhesus monkeys over a 20 year period have shown that the invasive placements of these flexible electrodes in these limbic system sites by themselves did not produce spiking activity even in long term (up to 26 weeks) EEG follow-ups. Hence, the hippocampal spiking seen in our small sample constitutes a statistically significant event.

Our experimental results suggest the following alternative management of potential medicolegal whiplash patients:

(a) Should the patient exhibit a reduction of lordosis of the cervical spine together with severe muscle spasm during the acute phase of the syndrome then at least one, preferably two, clinical EEGs should be ordered.

(b) If subjective complaints persist after 6 weeks, e.g. radicular symptoms, further serial EEGs, in addition to the traditional EMG, should be ordered. Based on the data of Torres and Shapiro (1961) and Gibbs (1971), in 40% of these patients, EEG abnormalities should appear, thus establishing a prima facie clinical diagnosis.

(c) For all patients with a reduction of cervical lordosis, anticonvulsant drugs such as phenytoin and/or phenobarbital may be simultaneously prescribed on an experimental basis as a prophylactic measure even though the definitive experimental and clinical studies are still lacking. These drugs have already been approved for use in direct-impact closed head injuries and therefore, should be suitable for a clinical trial in whiplash patients.

Acknowledgment

The authors wish to express appreciation to Mr. Herbert Diagle for the technical assistance provided in electrode implantation and EEG monitoring. The generous bioinstrumentation assistance provided by Dr. K. W. Krieger during the second half of this study is hereby acknowledged. The advice and encouragement of Dr. J. K. Wickstrom, immediate past Chairman of Orthopedic Surgery, Tulane University School of Medicine, throughout the course of this research is very much appreciated.

TABLE I

ANIMAL	WEIGHT and SEX	EEG CHANGES
I-A	38 N F	No post-whiplash abnormalities in scalp EEG for 27 weeks.
I-B	45 N F	Two weeks post-whiplash occasional paroxysmal focal slowing appeared in F4-C4 and F3-C3 during light sleep. Hippocampal spiking developed 6-weeks post whiplash on L HIP which spread to the right side as a mirror-focus. Lost electrode plug on 27th week.
I-C	38 N F	No post-whiplash abnormalities in scalp and depth EEG in 27 weeks.
I-D	38 N F	No ~~post-whiplash~~ abnormalities in scalp and depth EEG in 27 weeks.
II-A	36 N M	No post-whiplash abnormalities in scalp and depth EEG in 11 weeks.
II-B	35 N M	No scalp EEG abnormalities post whiplash. Seven weeks post-whiplash, occasional left hippocampal spiking occurred. Spiking continued to 11th week. Lost electrode plug in 11th week. No mirror focus phenomenon.
II-C	37 N M	No EEG abnormalities post-whiplash in 11 weeks.
II-D	38 N M	No scalp (or depth) EEG abnormalities in 11 weeks. ~~post-whiplash~~.
III-A	38 N M	No scalp EEG changes for 10 weeks. Recording interrupted for 6 weeks. Resumed recording for 2 more weeks. No scalp EEG changes.
III-B	29 N M	Post whiplash EEG recording for 10 weeks with no changes. No recording for 6 weeks for technical reasons. Resumed recording for 2 more weeks. No discernable changes.
III-C	36 N M	Post whiplash EEG recording for 10 weeks with no changes. No recording for 6 weeks for technical reasons. Resumed recording for 2 more weeks. No discernable changes.
III-D	36 N	No EEG changes in either scalp of depth leads for 10 weeks. Recording interrupted for 6 weeks. Resumed recording for 2 more weeks with no discerning changes.

TABLE I (cont.)

IV-A	37 N M	No scalp EEG changes in 22 weeks.
IV-B	40 N M	Left hippocampal spiking 8 weeks post-whiplash. Mirror-focus fully developed on right hippocampus by the 10th week. Continuous spiking in both hippocampi from 12th week on. Sacrificed on 52nd week.
IV-C	44 N M	Right hippocampal spiking 7 weeks post-whiplash. Mirror-focus fully developed on left hippocampus by 11th week. Continuous spiking thereafter. ~~Given phenytoin on the 28th week. By the 31st week, the spiking activity markedly decreased on the left hippocampus but less so on the right hippocampus~~. Lost plug at 42nd week.
IV-D	36 N M	No scalp or depth EEG changes for 22 weeks.

Caption for Table I

Synopsis of EEG follow up on all animals in the experimental protocol. Animal weight given in newtons (N).

LIST OF FIGURES

Fig. 8(a) Ammon's horn formation. Endpoint of electrode tract is Sommer's sector (h_1) labelled a. Traumatic necrosis of the entire area of Sommer's sector (h_1). Only a few neurons are visible in the region surrounded by the arrows. Spielmeyer's sector (h_2), and Bratz' sector (h_3) are intact. Cresyl-violet; 30:1. Animal I-B.

Fig. 8(b) The electrode tract shows hemosiderin laden macrophages and a marked glial-mesenchymal scar. The area of the traumatic necrosis contain debris laden macrophages and gitter cells. A few remaining neurons of the Sommer's sector (h_1) are shrunken and hyperchromatic. Cresyl-violet; 50:1. Animal I-B.

Fig. 9(a) Ammon's horn formation. Electrode tract extending into Sommer's sector(h_1). Marked local glial and mesenchymal reaction surrounding the tract labelled a. Neurons in Bratz sector (h_3), Spielmeyer's sector (h_2) and Sommer's sector (h_1) intact with the exception of a minimal loss of neurons with glial reaction in the immediate surrounding of the electrode tract in (h_1). Hematoxylin-eosin; 30:1. Animal II-B.

Fig. 9(b) The glial-mesenchymal scar surrounding the electrode tract in (h_1) is visible. Local loss of neurons with microglial and astroglial reaction in the area directly surrounding the electrode tract; otherwise intact neurons. Hemotoxylin-eosin; 50:1. Animal II-B.

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I. INTRODUCTION

10:00-10:10 Dr. Michael Cohen

Introduction

II. LIFE IN AN FFAC NURSERY

10:10-10:30 Christie Lievermann and Michael Marcus

Q & A Interview

10:40-11:00 Dr. Jack C. Redman

Discussion of life circumstances in the FFAC nurseries in Vietnam

III. THE CRASH

11:10-11:30 Mr. William Timm

Description of the C5A accident on April 4, 1975 near Saigon, Vietnam

11:40-12:00 Dr. Richard Snyder

Impact tolerance to the human brain

12:10-12:30 Dr. J. Kenneth Mason

Introduction to Aviation Pathology

Lunch 12:30-1:00

1:00-1:20 Dr. Y. King Liu

Film of head injury in monkeys

IV. CLINICAL FINDINGS

1:30-1:50 Dr. Itzhak Brook

Pediatrician's View

2:00-2:20 Dr. Eric Denhoff

General Introduction to the condition of MBD and related disorders

[illegible] C5A aircrash accident: A comparative study to Wendel's Monkey Model

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2:30-2:50 Dr. Steven Feldman

Elements of future rehabilitative efforts on behalf of the surviving children

Coffee Break 3:00-3:15

3:15-4:05 Dr. Bruce Copeland and Dr. C. Keith Connors

Psychological evaluation and diagnostic aspects

Psychiatric correlates of the psychological evaluation

4:15-4:35 Dr. Thomas Lustberg and Marcia Robinson

Psychiatric aspects

4:45-5:05 Dr. Marianne Schuelein

Pediatric Neurological and EEG aspects

V. CONCLUSION

5:10-5:30 Dr. Michael Malone

Summary and Conclusions

The Workshop will be held at the Hyatt Rosslyn, Saturday, March 8, 1980, Room 411, 1325 Wilson Boulevard, Arlington, Virginia, 703-841-9595.

Coffee will be provided beginning at 9:30 a.m.

V. Nauchildren Vs Lockheed & U.S. 3/8/80

(13–18 months old at time of accident)

1) Decompression injury – Caisson disease
2) Deceleration – 300 mph to 0 in short time (?)
3) Fire – CO in environment
4) Cyanide poisoning as a result of fire
5) Hypoxia (hypoxic) pulmonary insufficiency

Fractures (on 5 kids)

1) Knee stiffness
2) femoral neck
3) lower extremities injury
4) Mult. scars

Factors adv. to kids

1) hemoglobin (fetal) different from adult better O_2 exchange
2) Cooling hypothermia
3) Regeneration of trauma easier
4) lack of arteriosclerotic changes
5) " ?? lipid formation
6)

Factors adverse to kids

1) Surface area is larger / body wt
2) less restraint not made for children
3) inability to prevent trauma

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4) Anemia
5) Head larger w.r.t. to body.

No need for exaggeration nor have any need for secondary gain in this children.

Hypotonia (Acute symptoms) — De Hoff

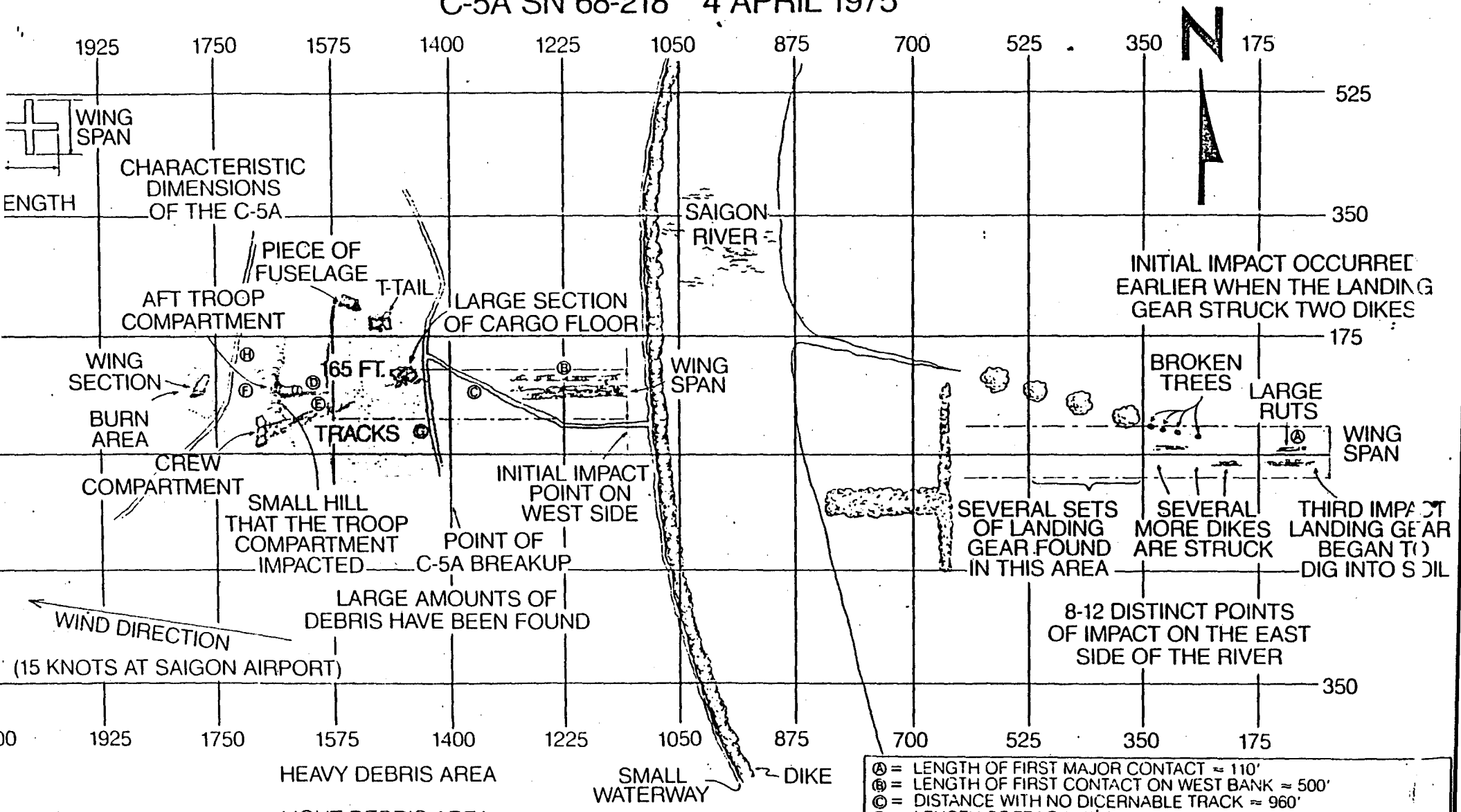
	Aircrash	Non-air crash	
Learning	49	5	Delayed symptom.
Communication			
Attention	31	0	
Behavioral			

8 min. of hypoxia produces permanent behavioral + M.B.D. using a monkey model (fetal monkeys) — Busby

Why does traumatized tissue hurt more during lowering of the atmospheric pressure?

Twin (one on crash plane & one came later) visual-motor deficit appears to be the dominant one.

WRECKAGE DIAGRAM
C-5A SN 68-218 4 APRIL 1975



Ⓐ = LENGTH OF FIRST MAJOR CONTACT ≈ 110'
Ⓑ = LENGTH OF FIRST CONTACT ON WEST BANK ≈ 500'
Ⓒ = DISTANCE WITH NO DICERNABLE TRACK ≈ 960'
Ⓓ = LENGTH OF TRACK LEADING TO TROOP COMPARTMENT ≈ 260'
Ⓔ = LENGTH OF TRACK LEADING TO CREW COMPARTMENT ≈ 390'
Ⓕ = DISTANCE FROM TROOP COMPARTMENT TO WING DEBRIS ≈ 360'
Ⓖ = DISTANCE FROM RIVER BANK TO CANAL WALKWAY ≈ 1150' (CONFIRMED ON MARY TOPOGRAPHIC MAP)
Ⓗ = DISTANCE FROM RIVER BANK TO CANAL WALKWAY ≈ 1990' (CONFIRMED ON ARMY TOPOGRAPHIC MAP)

T-TAIL IS ≈ 1310' FROM WEST BANK
TROOP COMPARTMENT IS ≈ 1715' FROM WEST BANK

DEFT. EX. ~~DD~~ Lw 8
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Crash victim

Vietnamese orphan awarded $500,000

WASHINGTON (AP) — A Vietnamese orphan who lived through an Air Force plane crash in Vietnam has won a $500,000 award from a federal jury that now will turn its attention to a similar suit filed by a second survivor.

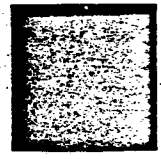
The jury award Monday was to Michael Moses Schneider, whose name was Hguyen Phi Khan when he boarded a C-5A mercy flight carrying homeless youngsters from Saigon to adoptive homes around the world. The plane crashed in a Saigon rice paddy April 4, 1975.

The damages will be paid by Lockheed Aircraft Corp., which manufactured the plane, and the federal government, whose Air Force crew flew the plane. The amount to be paid by each was not made public.

On Wednesday, the same jury will hear the case of James Matthew Zimmerly of St. Louis, who like Schneider was an infant Vietnamese orphan on the plane.

The Air Force said 135 children and adults of the 330 on board were killed. Of the 226 orphans on board, 76 perished. C-5A's are the world's largest aircraft.

Schneider, 6, living with adoptive parents in Denver, suffered permanent brain damage when the plane lost oxygen in flight and then crashed near Saigon's airport, the boy's attorney said.



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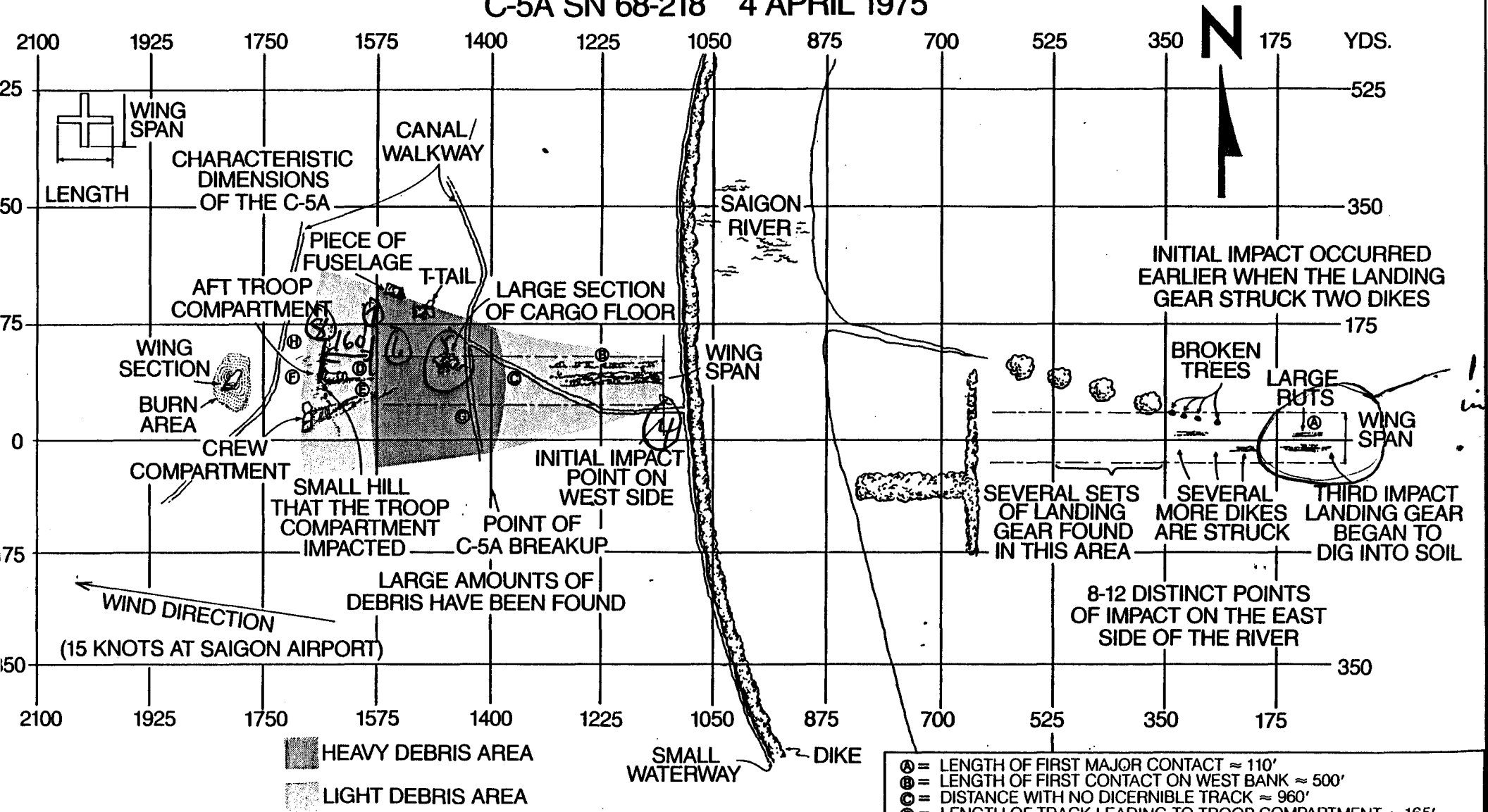
WRECKAGE DIAGRAM
C-5A SN 68-218 4 APRIL 1975



Ⓐ = LENGTH OF FIRST MAJOR CONTACT ≈ 110′
Ⓑ = LENGTH OF FIRST CONTACT ON WEST BANK ≈ 500′
Ⓒ = DISTANCE WITH NO DICERNIBLE TRACK ≈ 960′
Ⓓ = LENGTH OF TRACK LEADING TO TROOP COMPARTMENT ≈ 165′
Ⓔ = LENGTH OF TRACK LEADING TO CREW COMPARTMENT ≈ 450′ (AVG)
Ⓕ = DISTANCE FROM TROOP COMPARTMENT TO WING DEBRIS ≈ 360′
Ⓖ = DISTANCE FROM RIVER BANK TO CANAL/WALKWAY ≈ 1150′ (CONFIRMED ON ARMY TOPOGRAPHIC MAP)
Ⓗ = DISTANCE FROM RIVER BANK TO CANAL/WALKWAY ≈ 1990′ (CONFIRMED ON ARMY TOPOGRAPHIC MAP)

T-TAIL IS ≈ 1310′ FROM WEST BANK

DEFT. EX. ~~DD~~ LIU #10
DATE: 11/30/81
REPORTER: A. L. CASPAR

SUBCORTICAL EEG CHANGES IN RHESUS MONKEYS FOLLOWING EXPERIMENTAL HYPEREXTENSION-HYPERFLEXION (WHIPLASH)†

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†The work reported herein was performed while all authors were members of the faculty of Tulane University School of Medicine, New Orleans, LA 70112. The first author was a NIH Career Development Awardee (Grant No. GM 40723-05) during part of this investigation. The support provided by the National Institutes of Neurological and Communicative Diseases and Stroke (Grant No. NS GM 10517) is gratefully acknowledged.

Running Title:

SUBCORTICAL EEG CHANGES IN POSTWHIPLASHED RHESUS

Key Words:

Whiplash, Rhesus, Electroencephalography, Electrocorticogram, Hippocampus, Spiking

Abbreviations:

EEG - electroencephalogram; ECoG - electrocorticogram, EMG - electromyogram; RETIC - reticular formation; A SEP - anterior septum; CBL FAS - cerebellum fastigus nucleus; HIP - hippocampus; L - left and R - right.

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ABSTRACT

A controlled study, involving EEG recordings from the scalp and chronically implanted electrodes in the cortex (ECoG) as well as from selected subcortical nuclei, was undertaken to investigate the neurophysiologic effects on rhesus monkeys following experimental whiplash (hyperextension of the head and neck). Sixteen animals, equally divided into four groups, were studied through the following protocol: two animals within each group were whiplashed and then the brain of one was implanted with deep electrodes, while the second two animals were implanted with deep electrodes and then one was whiplashed after healing. Weekly EEG follow-ups showed hippocampal spiking in 3 of the 4 whiplashed and then electrode-implanted animals and in 1 of the 4 electrode-implanted and then whiplashed animals.

Several results deserve attention. (1) Prior to the onset of spiking, i.e., 6 to 8 weeks post-whiplash, all scalp, cortical and subcortical EEG recordings were normal. (2) When hippocampal EEG spiking did take place, only normal and mildly abnormal changes were seen in either the ECoG or scalp EEG. (3) The growth and development of this trauma-induced hippocampal spiking followed the classic sequence for the spread of an epileptogenic focus. This subclinical form of post-traumatic epilepsy may be a threshold phenomenon, i.e., the whiplash plus the subcortical electrode placements further decrease the already well-known low-spiking threshold of the hippocampi.

INTRODUCTION

After a front-to-rear automobile collision, the occupants of the automobile hit from behind may suffer a "whiplash injury", a term attributed to Crowe (1) to describe an injury to the neck in such accidents. Phenomenologically, the term comes from a description of the violent motion of head and neck in which the head is suddenly thrown backward resulting in a hyperextension of the neck (the whip phase) and immediately afterwards is recoiled in the opposite direction forcing the neck into hyperflexion (the lash phase). The use of the term "whiplash injury" by Braaf and Rosner (2) and States et al. (3) have been critized by other authors, e.g., Hamel and James (4) and Farbman (5), because it described the motion rather than the pathology. In spite of the criticism, the term is entrenched in the lay, medical and legal literature. Whiplash will be the term used hereafter in this paper to describe this special type of head and neck injury instead of the more cumbersome "hyperextension-hyperflexion".

The clinical problem associated with whiplash injury has been well stated by McNab (6):

> Everyone is well acquainted with the common story associated with whiplash injuries. Despite the history of pain the the neck, pain between the shoulder blades, pain the back of the head, pain down the arm, dysphagia, tinnitus, vertigo, and intermittent blurring of vision, there is remarkably little to find on examination. The usual x-ray films do not commonly show any gross abnormalities, even though there may be an alteration in the normal cervical curve. Despite the frustratingly negative examination, the patient's aches and pains fail to respond to the Brownian movements of routine medical therapy. As the months roll by, it becomes increasingly apparent that the patient is grossly emotionally disturbed.
>
> There is general agreement on the type of symptoms to be expected, but widely divergent views are held on the significance of these symptoms. These divergent viewpoints, rigidly held and hotly contested, are firmly based on impressions only. In an endeavor to make these impressions more factually significant, the progress of 575 patients has been carefully followed. Many physicians believe

that the 'whiplash syndrome' is demonstrated only by a group of hysterical, neurotic, if not frankly dishonest, people. However, there are certain disturbing features apparent from an analysis of the case histories available that make it difficult to accept the belief that litigation neurosis is the sole explanation of the long drawn out disability in every instance.

He proceeded to show, quite conclusively, that two or more years after settlement of the court action, 121 out of 266 or 45% continued to have symptoms.

In a recent paper using insurance claim data, O'Neill et al. (7) have stated, that despite some reduction in head and neck injuries due to the requirement of head restraints, the number of whiplash injuries in the U.S. may be "considerably in excess of one million." The reason given for the limited success of what appears to be a rational solution for the whiplash problem is that the adjustable head restraints were usually improperly positioned. The authors warned:

> an adjustable head restraint if left in its lowest position actually may increase the chances of severe whiplash injuries by acting as a fulcrum for the head, especially for taller people.

Torres and Shapiro (8) have investigated the EEG of post-whiplashed patients. In a population of 45 patients, 46% had electrical abnormalities of moderate or marked degree while the rest had either mildly abnormal or normal EEGs. Those patients who had more than one EEG showed progressive deterioration instead of improvement in their follow-up records. The authors concluded that:

> It is highly improbable that the association of these abnormalities, of higher degree and much higher incidence than any series of normals, with the history and clinical findings of whiplash injury might be exclusively due to chance factors. We conclude, therefore, that the electrical abnormalities were probably caused by the injury.

Gibbs (9) has reassessed the use of clinical EEG as an objective indicator of brain disorder in whiplash injury. He compared the percentage incidence of abnormal findings among 178 medicolegal cases of whiplash with

the percentage incidence of these same EEG findings among asymptomatic control subjects. He found that 11% had EEGs with focal slowing and 5% had paroxysmal slowing, two abnormalities almost never found in asymptomatic control subjects. Though age related, practically all the other abnormalities are found in whiplashed patients as well as asymptomatic controls. It should be emphasized, however, that the incidence of such abnormalities in the asymptomatic controls are found less frequently than in the whiplashed patients. In the adult group between ages of 20 to 60, which accounts for 148 of the 178 cases studied, approximately 65% of these patients with symptoms show normal EEG. Gibbs (9) has further contended:

> A patient who has symptoms and whose EEG shows mild abnormalities might deserve consideration, even though his complaints and abnormalities are the same as those of many other persons.

Scalp EEG work based on animal studies has been reported by Wickstrom et al. (10). Thirty-six EEGs were recorded from rhesus monkeys before and after controlled whiplash. The recorded EEGs were interpreted by an electroencephalographer. Of the 36, 7 had abnormal EEGs. Of the 7 with abnormal EEGs, 6 also had abnormal behavioral symptoms under clinical examination, yet only 4 had identifiable histopathology after necropsy. Of the remaining 29 with "normal" EEGs, examination subsequent to sacrifice revealed that 3 had spinal cord lesions, 2 had brain injuries and 2 others had both brain and spinal cord pathology. In short, approximately 24% of the monkeys with definite neurohistological lesions had no EEG abnormalities by the simulated clinical procedure. Thus, the abnormal EEG is a rather poor indicator of nervous tissue injury. Conversely, the presence or absence of neurohistological lesions cannot be depended upon as the indicator of EEG abnormality either.

Koshino et al. (12) have utilized megimide activation of the EEG to study whiplash and head injury patients with stubborn subjective symptoms, but who showed neither objective abnormalities of the neck nor abnormal clinical EEG. In a clinical study involving 35 whiplash and 45 head injury patients, it was found that 57% of the whiplash group and 73.3% of the head injury group had abnormal activated EEGs after injection of less than 100 mg of megimide. The mean doses of megimide required for the activation of irregular spike and wave complex in the whiplash and head injury groups were 80 mg and 72 mg respectively. Wada (13) has shown that the threshold of megimide activation of EEG in the normal person is more than 150 mg, and generally an abnormality is indicated if a threshold of less than 100 mg of megimide activated the EEG. Furthermore, the activated abnormal EEG's were provoked at almost the same rate irrespective of the duration from the time of injury to that of examination.

As far as we are aware, the only previous study of subcortical EEG changes following experimental whiplash was the work of Tsuchiya et al. (11). Rigid electrodes were implanted into the following areas of the rabbit brain: neocortex, hippocampus, premammillary area, internal capsule, preoptic area, lateral hypothalamus, ventromedial hypothalamus, central gray matter, and the reticular formation. The results can be summarized as follows:

1) In the neocortex, low amplitude fast waves which were observed immediately after the impact returned almost to the control pattern within 2 to 6 days,
2) In the hypothalamus, normal EEG pattern was observed soon after trauma. However, abnormal EEG, such as the high amplitude slow waves or spikes, were observed 7 to 45 days after the

impact and continued for some period. The regions from which the abnormal waves were recorded were: the medial preoptic area, ventromedial hypothalamic nucleus, posterior hypothalamus, premammillary nucleus, lateral hypothalamic nucleus and lateral preoptic area.

3) In the midbrain reticular formation, sporadic sharp spikes were observed within 2 to 3 days after the whiplash injury, with the EEG apparently returning to normal thereafter.

Besides the obvious objection to the use of rigid electrodes, the experiments of Tsuchiya et al. (11) were incompletely controlled. The combination of rigid electrodes implanted in the subcortex together with a high rotational acceleration during whiplash obviously contributed to the injury and the subsequent EEG changes. This illuminates the need for improved controls in spite of this study's importance and originality. This study by Tsuchiya, et al. (11) has laid the difficult foundations upon which our research is based.

1. Experimental Design

The experimental protocol called for the use of four matched groups of rhesus monkeys of the same sex, each weighing about 4 k. The study began with at least two baseline scalp EEG recordings per animal. In each series, after ascertaining the scalp EEG to be normal, two of the four animals were whiplashed in the acceleration facility. After the post-whiplash baseline scalp EEG was recorded, one (Animal A) of the above two experimental animals was randomly chosen to act as the scalp control, while the other was implanted with flexible deep electrodes (Animal B). The third (Animal C) was implanted with electrodes and then whiplashed after the wounds were allowed to heal. The fourth (Animal D) was implanted with electrodes to act as the electrode control for the series. The protocol is summarized as follows: Animal (A), whiplashed and scalp EEG followed; Animal (B), whiplashed and then implanted with deep electrodes; Animal (C), implanted with deep electrodes and then whiplashed; and Animal (D), implanted with deep electrodes only.

One of the main concerns in the above experimental protocol was: how can one be reasonably sure that, for Animal (C), the whiplash head injury was not the result of presence of deep flexible electrodes in the brain interacting with the high angular acceleration during the whiplash? To insure that the flexible electrode used did not excessively damage the brain during the acceleration pulse, we simulated our experiment in a gel-filled transparent half skull. Electrodes were inserted into the gel as during in vivo implantation into the brain. Indian ink was then injected into the

electrode track until it was filled. A small plastic wedge was used to fix and seal the electrode at the skull, just as used in an actual implantation procedure. The ensemble was whiplashed in a similar fashion as in a test, although the angular acceleration levels achieved were much lower. The procedure was recorded on high speed film and then analyzed. No ink left the tracks.

2. Flexible Electrode Implantation

The stereotaxic implantation was done in the manner of Heath (14), using flexible bipolar electrodes with silver-balled tips of approximatly 0.5 mm diameter and located 1 mm apart. The electrode implantation was performed under approximately 120 mg of phenobarbitol administered i.v. Because patients with whiplash symptoms very often become emotionally disturbed with time, we postulated the probable involvement of the limbic system, which has been theorized by Papez (15), Maclean (16), Nauta (17), and Zanchetti (18), as the regulator of emotional expression. Heath (19) has reported on data collected over a 20-year period which showed functional connections between certain sensory relay nuclei (ventro-postero-lateral nucleus of the thalamus and the fastigius nucleus of the cerebellum) and brain sites (primarily the septal region and hippocampus), where physiologic activity has been shown to be correlated with emotional expression. Thus, the deep sites chosen for chronic electrode implanation were: the left and right hippocampi (L HIP and R HIP), right reticular formation (R RETIC), right anterior septum (RA SEP) and right cerebellum fastigius nucleus (R CBL FAS). Epidural cortical leads were placed over the frontal, temporal and occipital cortex, to obtain the electrocorticogram (ECoG). The scalp leads used were F7, T3, T5, T8, T4, and T6, i.e., the midtemporal chain in the international

10-20 system. The animals received antibiotics upon completion of their electrode implantation to minimize infection at the surgical sites. Ten days to two weeks were allowed for wound healing.

3. Hyperextension-hyperflexion Trauma

On the day of the whiplash, the animal was tranquilized with phencyclidine hydrochloride (Sernylan® 2 mg/kg) intramuscularly. The torso of the rhesus was then restrained with a jacket and placed sitting on a miniature cart with its head and neck unrestrained. Briefly, the experimental setup consisted of a compressed air pump and reservoir connected to a 152 mm diameter circular cylinder with an associated piston having a 914 mm stroke length. The magnitude of the acceleration exerted on the cart was controlled by the pressure of the compressed air in the reservoir. The duration of acceleration of the vehicle can be varied somewhat by the placement of polyurethane pads of different thickness onto the impacting piston. During the acceleration phase, the cart triggers three pulsed x-ray units (Fexitron No. 730 Hewlett-Packard Corp.) through microswitches to record the changing relationship during the various stages of the hyperextension of the head and neck. A typical radiograph of an animal implanted with deep electrodes being whiplashed is shown in Figure 1. The event is also recorded on a Hycam (Redlake Corp.) high speed camera at 1,000 frames/sec against a preset grid. The acceleration procedure outlined above was previously reported in Domer et al. (20).

4. Data Acquisition

Whenever possible, at least one EEG per week per animal was monitored and tape recorded. The EEG technician kept a continuous log of each animal

during the recording session. Information, such as the beginning and end of sleep, wakefulness, and any abnormalities during these recording sessions was carefully noted and correlated to a time-code generator signal in order to facilitate the later signal analysis.

RESULTS

Typical cart and linear accelerations of the skull as well as the computed angular accelerations have been illustrated previously in Domer et al. (20). In the interest of continuity, the numerical results are reiterated: (1) The maximum linear acceleration of the cart was about 35 g's, occurring at about 30 msec after the onset of the impact. (2) The maximal calculated angular acceleration, achieved during hyperextension, was approximately 40×10^3 rad/sec^2 which occurred 50 msec after impact.

None of the monkeys lost consciousness following the whiplash as evidenced by the lack of eye closure and the maintenance of muscular tone. Furthermore, no blood issued from any orifice.

A synopsis of the electroencephalographic changes, if any, for all of the animals in the 4 series is given in Table I.

The animals with hippocampal spiking are: I-B, II-B, IV-B and IV-C. The time course of development of the hippocampal spiking activity in all 4 animals warrant individual attention and are summarized below:

Follow-up on Animal I-B

Two weeks post-whiplash, this animal developed occasional paroxysmal focal slowing during light sleep in F4-C4 and F3-C3. Since focal slowing is a form of scalp EEG abnormality that is almost never seen in unwhiplashed rhesus monkeys, we chose this animal for depth-electrode implantation on the notion that the depth correlates of the surface phenomena could be found. The electrode implantation took place 3 weeks post-whiplash. Two weeks were allowed for the surgical trauma to heal before the first

post-implant (5th week post-whiplash) recording was taken and shown in Fig. 2. Note the absence of abnormalities in the scalp, cortical and depth leads. Figures 3 through 6 recorded the serial events as the weeks passed. Figure 3 illustrates the incipient spiking activity in the right hippocampus 6 weeks post-whiplash during light sleep. The frontal cortical lead, which showed no abnormality, was dropped to make room for the right hippocampus in the first 6 channels. This was done to ease the signal analysis problem anticipated, i.e., the problem of detecting the subcortical spikes at the scalp. By the 8th week, shown in Fig. 4, the left hippocampus had developed a mirror focus. Figure 5 shows that during the 11th week, the left hippocampal activity during sleep was less dependent on the original right hippocampal focus, indicating the near completion of the epileptiform mirror focus. The temporal cortical lead was dropped to make room for the R HIP lead. By the 13th week, the right hippocampus of the animal was in complete "seizure-like" activity but only during sleep. By the 16th week, both hippocampi were continuously spiking practically at all times. Figure 6 is taken from the record of the 18th week showing continuous spiking acitivity in the hippocampi. Weekly tape-recordings continued until the 27th week when the animal knocked out its plugs by accident. The animal was sacrificed at this time. The last recording made showed a slight diminution of the spiking activity.

Follow-up Details on Animal II-B

This animal was whiplashed and then its subcortex implanted with electrodes two days later. Its first EEG record was taken 2 weeks after trauma. The 7th week post-whiplash record showed, during sleep, an occasional single spike in

the left hippocampus. When compared to the corresponding record (8th week post-whiplash) in the previous series shown in Fig. 4, the amplitude of the spikes are of equal magnitude but the frequency of occurrence is much lower. The frequency increased in the next two weekly recordings and reached a maximum of about 3 — 5 spikes for each ten seconds of sleep record as shown in Fig. 7. No mirror focus developed in the right hippocampus nor did the spiking reach the "seizure" level found in Series I.

Follow-up Details of Animal IV-B

This animal was whiplashed and its subcortex implanted with electrodes 10 days later. The first post-implant record was taken 10 days following electrode implantation. The 8th week post-whiplash record showed sharp waves and an occasional spike in the right hippocampus. The activity increased in the right hippocampus as the weeks passed and by the 11th week, a mirror focus had developed in the left hippocampus. By the 18th week, both hippocampi were showing continuous spiking activity. The recordings were continued for 52 weeks with only a slight attenuation in activity. The results were very similar to animal I-B.

Follow-up Details of Animal IV-C

This animal was implanted with electrodes and then monitored for 16 weeks and no abnormalities developed in the EEG. The animal was whiplashed at this point. The post-whiplash recordings were started 4 days later. Seven weeks post-whiplash, the record showed random sharp waves and occasional spikes in the left hippocampus with delta and theta wave activity in the right hippocampus. By the 10th week, the left hippocampus had

developed random bursts of sharp waves and spikes and the right hippocampus had developed a mirror focus. By the 14th week, both sides were in complete "seizure-like" activity with the left hippocampus slightly more active. Continuous spiking activity occurred in both hippocampi thereafter. This was the first C type animal which developed spiking activity. The results were again very similar to animal I-B.

Histologic Controls and Pathological Findings

A standard intracardiac perfusion-fixation technique was used at the time of sacrifice. A laminectomy was performed on the cervical spine to expose the cervical spinal cord. A craniotomy was next performed and the brain and spinal cord removed *in toto*. Any gross pathology was described and photographed. Following coronal section, the blocks were embedded in celloidin. The sections were stained with either hematoxylin-eosin or cresyl-violet or both techniques. The histological evaluation was done in the manner of Unterharnscheidt and Higgins (21). In our evaluation, special attention was given to the exact locations of the electrode tracts and their endpoints described. The neurohistology has been completed in Series I and II.

All animals showed traumatic lesions associated with the electrode implantation. In the interest of brevity, only the microphotographs associated with the spiking nuclei are displayed. Figures 8 and 9 show the right and left Ammon's horn formation in animals I-B and II-B respectively. In Fig. 8, the endpoint of the electrode tract in Sommer's sector (h_1) is labelled a. Only a few neurons are visible in the region surrounded by the arrows. Figure 9 shows typically the marked local glial and mesenchymal reaction surrounding the electrode tract labelled a. All the neuro-

histology completed thus far confirms the electrode placement for all 8 animals. There were no primary or secondary traumatic lesions elsewhere in the brain or spinal cord.

DISCUSSION

Numerous means have been found to induce epileptiform activity in the brain. These include a range of different agents such as cobalt, penicillin, metrazol, ethylene chloride, dry ice and others. As far as we are aware, this is the first time that subcortical epileptiform activity has been exhibited in a controlled whiplash primate experiment. Post-traumatic epilepsy occurs in 10% of all severely closed head-injured patients with a clinical diagnosis of contusion.

In the present experiment, the physical event initiating trauma was a whiplash movement of the head and neck and not a direct-contact impact of the scalp and skull. If one accepts cerebral concussion as the dramatic, sudden loss of consciousness as a result of trauma, then none of the whiplashed animals in this study were concussed.

Torres and Shapiro (8) have compared the clinical EEG of 45 head injury patients with 45 patients suffering from whiplash injuries. In their patient selection procedure, they showed that while both groups had the same subjective complaints, there was one major difference: 23 of the patients with contact head injuries suffered from cerebral concussion, or amnesia but this was totally absent in the whiplashed group. Thus, the fact that our animals were not concussed is, therefore, not surprising. Traumatological investigators differ widely on the question of whether traumatic unconsciousness and whiplash injuries are accompanied by histologic changes. Our neurohistological findings for Series I and II show no definite primary or secondary traumatic lesions as a result of our measured whiplash input although previous results on a much larger

population of rhesus done by Wickstrom et al. (10) did show a few such lesions. Thus, the hippocampal spiking exhibited by whiplashed primates might be considered as a subclinical form of post-traumatic-epilepsy.

Walker et al. (22) have described experimental results to support the concept that the traumatic unconscious state is very similar to the aftermath of a generalized epileptic convulsion. Subcortical spiking at localized nuclear sites of the brain, however, seldom leads to a clinical seizure. In the rhesus monkey brain, with our particular depth leads, the point has been made by Heath (19):

> In some experiments (with cobalt induced epileptiform activity), when these epileptiform discharges spread to encompass the entire brain, the monkey had a clinical epileptic seizure. Behavioral changes were minimal or absent when the epileptiform discharges were limited to one or more nuclear sites without affecting the septal region. But when the septal region was involved, either alone or in concert with epileptiform activity at other deep nuclear sites, the monkeys invariably displayed gross behavioral aberrations in the form of impaired emotionality and reduced awareness which progressed to catatonia.

No gross motor behavioral changes, such as clinical convulsions, were observed in any of the 4 animals even when the spiking discharges in the hippocampi were continuous.

The mirror focus phenomenon, seen in animals I-B, IV-B, and IV-C have been seen in all experimentally induced epilepsy using different agents on the cortex. Morrel (23) has characterized the growth and development of the mirror-focus as being similar to the learning curve. The growth and development of our trammatically induced hippocampal focus closely parallels an artificially induced local epileptiform activity. The altered metabolism of the nervous tissue appears to be the unifying factor connecting all the experimental models, including our own. This

altered metabolism can be in either the neurons themselves or in the autoregulatory system of the neuronal networks, or both.

The hippocampal spiking activity is most probably a threshold phenomenon, i.e., the combination of the traumatic presence of the electrode plus the whiplash is enough to drive the sensitive neuronal system and/or the neurons themselves, beyond its spiking threshold; whereas, each by itself is insufficient to accomplish the same. The low spiking threshold of the hippocampus is well-known and as such it is not surprising that it is the only site in our experimental study to exhibit such a phenomenon.

When subcortical spiking does occur, why is it confined mainly to the animal which was whiplashed and then implanted with electrodes, i.e., animal B in each series? We sought the answer to the above question through its control, animal C, which was implanted with electrodes and then whiplashed. The only difference between B and C, other than the normal variations of the repeatability of the experiment, is the presence of the deep electrodes during whiplash. Our tentative conjecture is that the presence of the electrodes tether the brain, so that the relative motion between the skull and brain is less than would be the case if the electrodes were absent. The brain material is almost incompressible with a bulk modulus K = 2.07 giga pascals (300,000 psi) and a Young's modulus E = 68,900 pascals (10 psi). On the other hand, its shear modulus is 1,378 pascals (G = 0.2 psi). In whiplash, the mechanical input is primarily a rotational acceleration and secondarily a translational acceleration of the head and neck. Both inputs induce a graded set of shear strains, maximum at the subdural cortex and decreasing towards the diencephalomesencephalic core. The flexible electrodes, which are

anchored by plastic wedges on the skull, have an order of magnitude higher shear modulus. The brain, being a viscoelastic material, begins to compress the implanted electrode soon after insertion. The frictional forces generated are such that no discernable movement of the electrodes were detected from either the flash x-rays or from the neurohistology subsequent to sacrifice.

When the spiking activity was continuous within the hippocampi, the EEG activity of the remaining scalp leads, which simulated the clinical EEG, was only mildly abnormal or normal. However, if the scalp EEG records from Figs. 2 through 6 were read serially, it is possible to discern a progressive deterioration. This mild abnormality is in agreement with the results of Torres and Shapiro (8) on human whiplash patients.

In spite of the relatively small experimental sample size, some conclusions can be drawn from the above results. The most important point to be made concerns the number of electrode controls, i.e. D animals, which on the basis of the experimental protocol given above is only four. In fact, however, Heath (14) has implanted deep electrodes into the various nuclei of the limbic system of the rhesus over the past 20 years. In over 200 animals in which our chosen sites were implanted as electrode controls, none have developed subcortical spiking during long-term follow-up using identical electrodes and procedures as in the present study. If in 200 normally implanted animals no spiking occurred, but that as a result of the given whiplash, 3 out of 4 of the B animals and 1 out of 4 C animals developed spiking, then heuristically, one would assume that the whiplash episode was a significant event.

It is premature to come to any definite conclusions concerning the interesting lesions observed. Many more extensive and detailed experiments are needed before one can decide whether these lesions are caused by the whiplash or electrode trauma or one synergistically acting on the other. The neurohistology will constitute the content of a future paper.

CONCLUSION

1) The immediate post-whiplash scalp EEG can be considered as a normal baseline recording if free from any previous abnormalities. So far, when the whiplash animals do develop abnormalities, they do so at least 6 weeks post-whiplash.

2) When subcortical EEG changes did take place, only normal or mild abnormal tracings were observable in either the ECoG or the scalp EEG during the same time period in this series of experiments.

3) The spiking activities in the hippocampi occurred mainly in animals which were whiplashed and then implanted with electrodes i.e., B animals. The presence of spiking activity was observed in only one animal which was implanted with electrodes and then whiplashed. These results suggest that not only is there minimal interaction between the brain matter and the flexible electrodes due to the whiplash but that these electrodes may have a tethering effect in reducing the rotation displacement of the brain.

4) The hippocampal spiking activities is most likely a threshold phenomenon, i.e., the spiking is the result of the synergistic combination of the invasive presence of the recording electrodes and the hypersensitivity of the hippocampi to whiplash. Previous electrode implantation studies in more than 200 rhesus monkeys over a 20 year period have shown that the invasive placements of these flexible electrodes in these limbic system sites by themselves did not produce spiking activity even in long term (up to 26 weeks) EEG follow-ups. Hence, the hippocampal spiking seen in our small sample constitutes a statistically significant event.

Our experimental results suggest the following alternative management of potential medicolegal whiplash patients:

(a) Should the patient exhibit a reduction of lordosis of the cervical spine together with severe muscle spasm during the acute phase of the syndrome then at least one, preferably two, clinical EEGs should be ordered.

(b) If subjective complaints persist after 6 weeks, e.g. radicular symptoms, further serial EEGs, in addition to the traditional EMG, should be ordered. Based on the data of Torres and Shapiro (8) and Gibbs (9) in 40% of these patients, EEG abnormalities as compared to the baseline recording, should appear, thus establishing a prima facie clinical diagnosis.

(c) For all patients with a reduction of cervical lordosis, anticonvulsant drugs such as phenytoin and/or phenobarbital may be simultaneously prescribed on an experimental basis as a prophylactic measure even though the definitive experimental and clinical studies are still lacking. These drugs have already been approved for use in direct-impact closed head injuries and therefore, should be suitable for a clinical trial in whiplash patients.

Acknowledgment

The authors wish to express appreciation to Mr. Herbert Diagle for the technical assistance provided in electrode implantation and EEG monitoring. The generous bioinstrumentation assistance provided by Dr. K. W. Krieger during the second-half of this study is hereby acknowledged. The advice and encouragement of Dr. J. K. Wickstrom, immediate past Chairman of Orthopedic Surgery, Tulane University School of Medicine, throughout the course of this research is very much appreciated.

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TABLE I

ANIMAL	WEIGHT and SEX	EEG CHANGES
I-A	38 N F	No post-whiplash abnormalities in scalp EEG for 27 weeks.
I-B	45 N F	Two weeks post-whiplash occasional paroxysmal focal slowing appeared in F4-C4 and F3-C3 during light sleep. Hippocampal spiking developed 6-weeks post whiplash on L HIP which spread to the right side as a mirror-focus. Lost electrode plug on 27th week.
I-C	38 N F	No post-whiplash abnormalities in scalp and depth EEG in 27 weeks.
I-D	38 N F	No post-whiplash abnormalities in scalp and depth EEG in 27 weeks.
II-A	36 N M	No post-whiplash abnormalities in scalp and depth EEG in 11 weeks.
II-B	35 N M	No scalp EEG abnormalities post whiplash. Seven weeks post-whiplash, occasional left hippocampal spiking occurred. Spiking continued to 11th week. Lost electrode plug in 11th week. No mirror focus phenomenon.
II-C	37 N M	No EEG abnormalities post-whiplash.
II-D	38 N M	No scalp EEG abnormalities post whiplash.
III-A	38 N M	No scalp EEG changes for 10 weeks. Recording interrupted for 6 weeks. Resumed recording for 2 more weeks. No scalp EEG changes
III-B	29 N M	Post whiplash EEG recording for 10 weeks with no changes. No recording for 6 weeks for technical reasons. Resumed recording for 2 more weeks. No discernable changes.

TABLE I (cont.)

III-C	36 N M	Post whiplash EEG recording for 10 weeks with no changes. No recording for 6 weeks for technical reasons. Resumed recording for 2 more weeks. No discernable changes.
III-D	36 N M	No EEG changes in either scalp or depth leads for 10 weeks. Recording interrupted for 6 weeks. Resumed recording for 2 more weeks with no discerning changes.
IV-A	37 N M	No scalp EEG changes in 22 weeks.
IV-B	40 N M	Left hippocampal spiking 8 weeks post-whiplash. Mirror-focus fully developed on right hippocampus by the 10th week. Continuous spiking in both hippocampi from 12th week on. Sacrificed on 52nd week.
IV-C	44 N M	Right hippocampal spiking 7 weeks post-whiplash. Mirror-focus fully developed on left hippocampus by 11th week. Continuous spiking thereafter. Given phenytoin on the 28th week. By the 31st week, the spiking activity markedly decreased on the left hippocampus but less so on the right hippocampus. Lost plug at 42nd week.
IV-D	36 N M	No scalp or depth EEG changes for 22 weeks.

Caption for Table I

Synopsis of EEG follow up on all animals in the experimental protocol. Animal weight given in newtons (N).

LIST OF FIGURES

Fig. 9(b) The glial-mesenchymal scar surrounding the electrode tract in (h_1) is visible. Local loss of neurons with microglial and astroglial reaction in the area directly surrounding the electrode tract; otherwise intact neurons. Hemotoxylin-eosin; 50:1. Animal II-B.

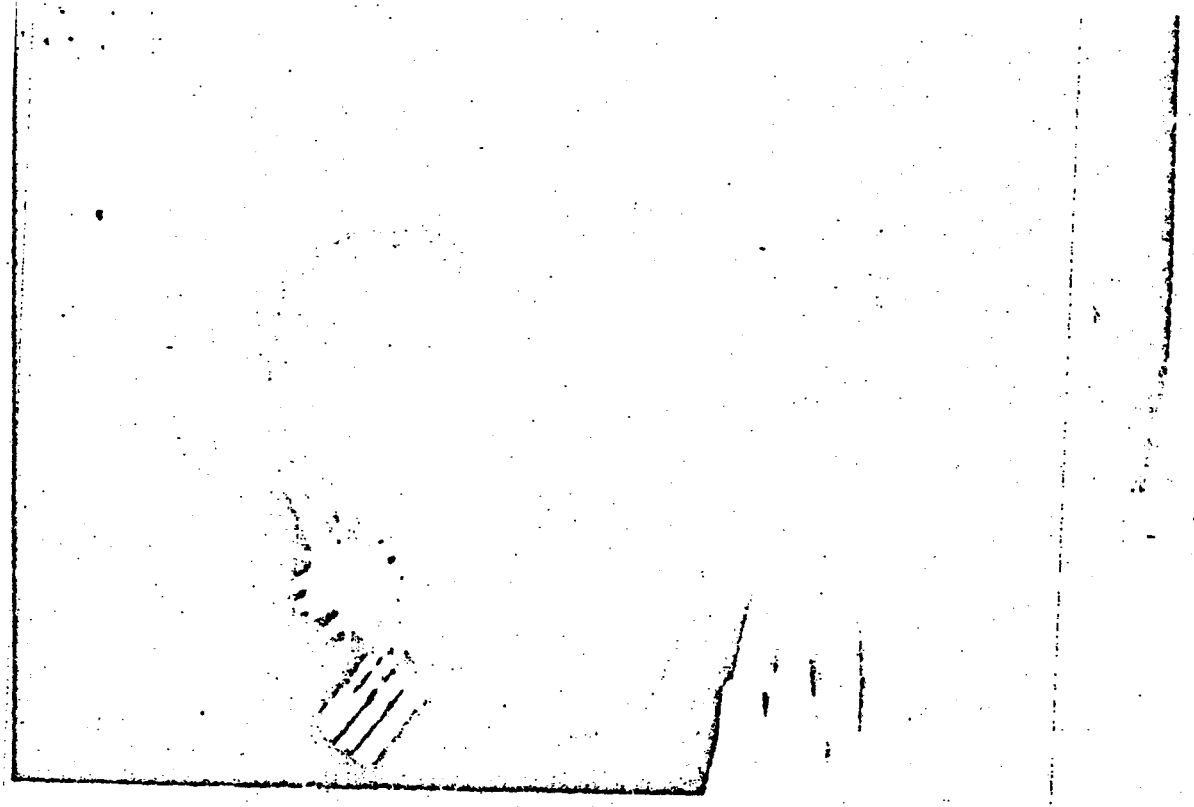


Fig. 1 Flash X-ray of a rhesus monkey head and neck implanted with electrodes and then whiplashed (animal C) during the hyperextension phase.

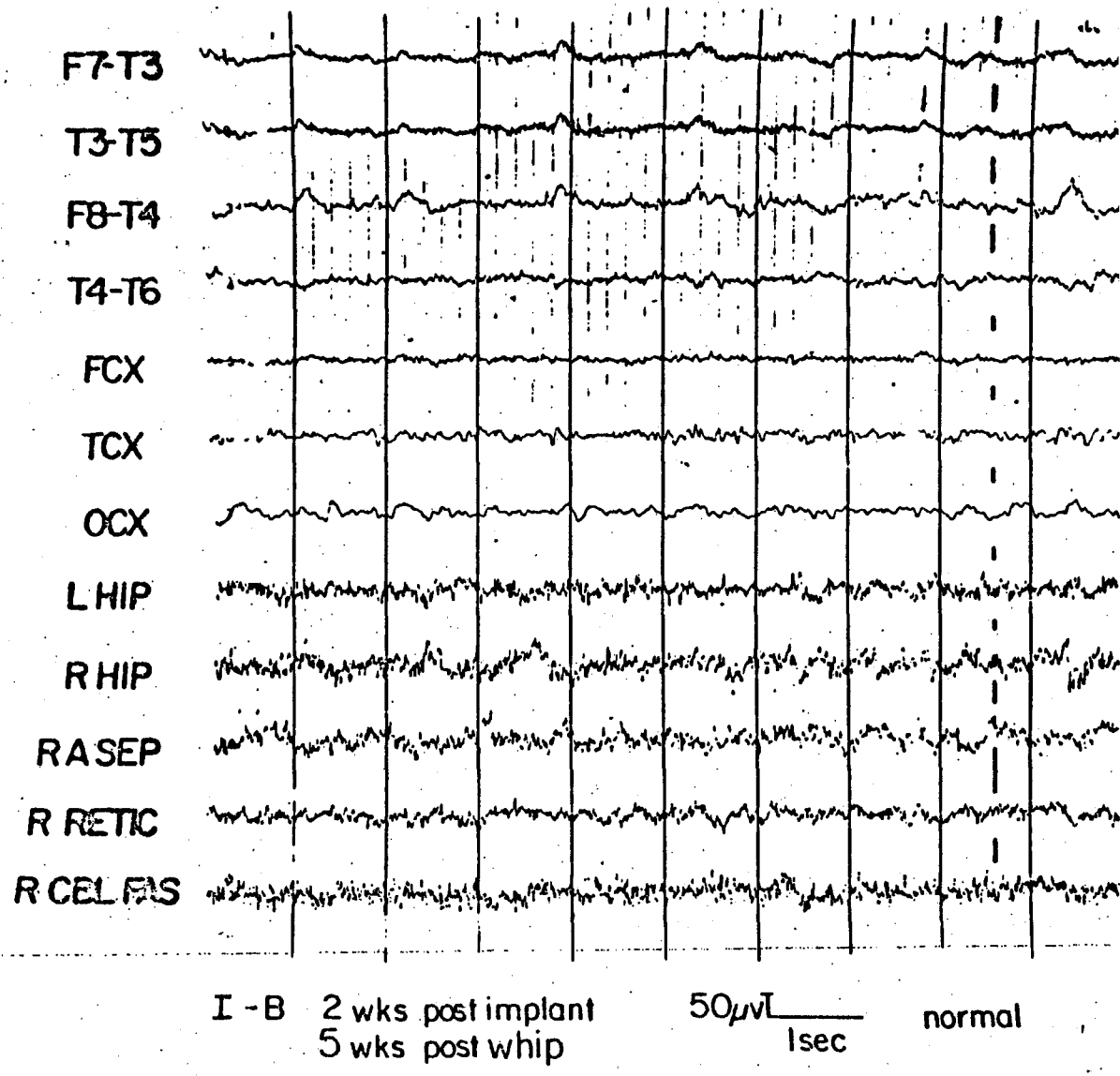


Fig. 2 The EEG of animal I-B 5 weeks post-whiplash and 2 weeks post electrode implantation. This record is considered normal.

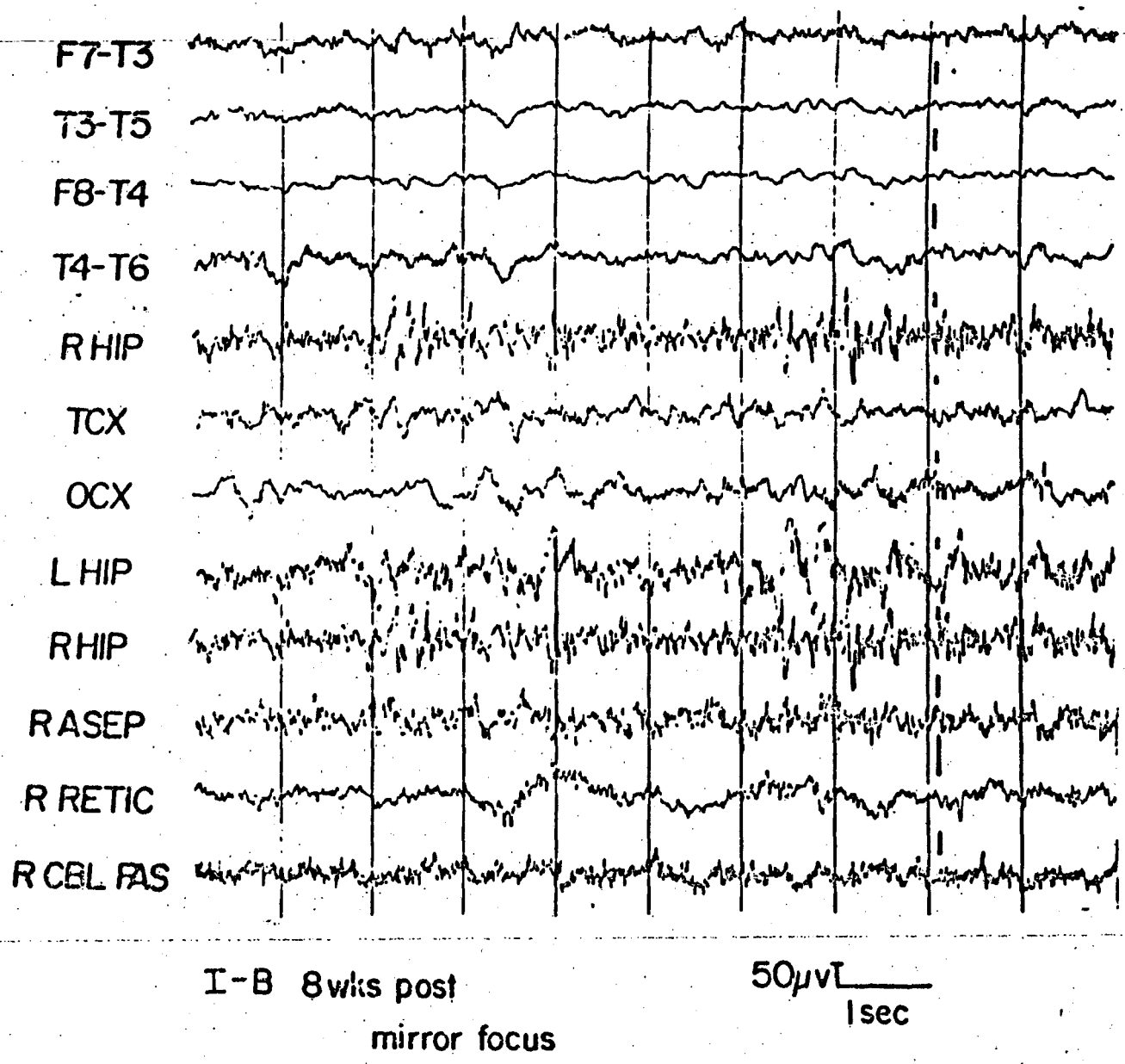


Fig. 4 The EEG of animal I-B 8 weeks post-whiplash illustrating the development of a mirror or secondary focus in the left hippocampus (L HIP) during sleep.

Fig. 5 The EEG of animal I-B 11 weeks post-whiplash showing the increas independence of the mirror focus (L HIP) during sleep. The temporal cortical lead was dropped to accommodate the L HIP lead.

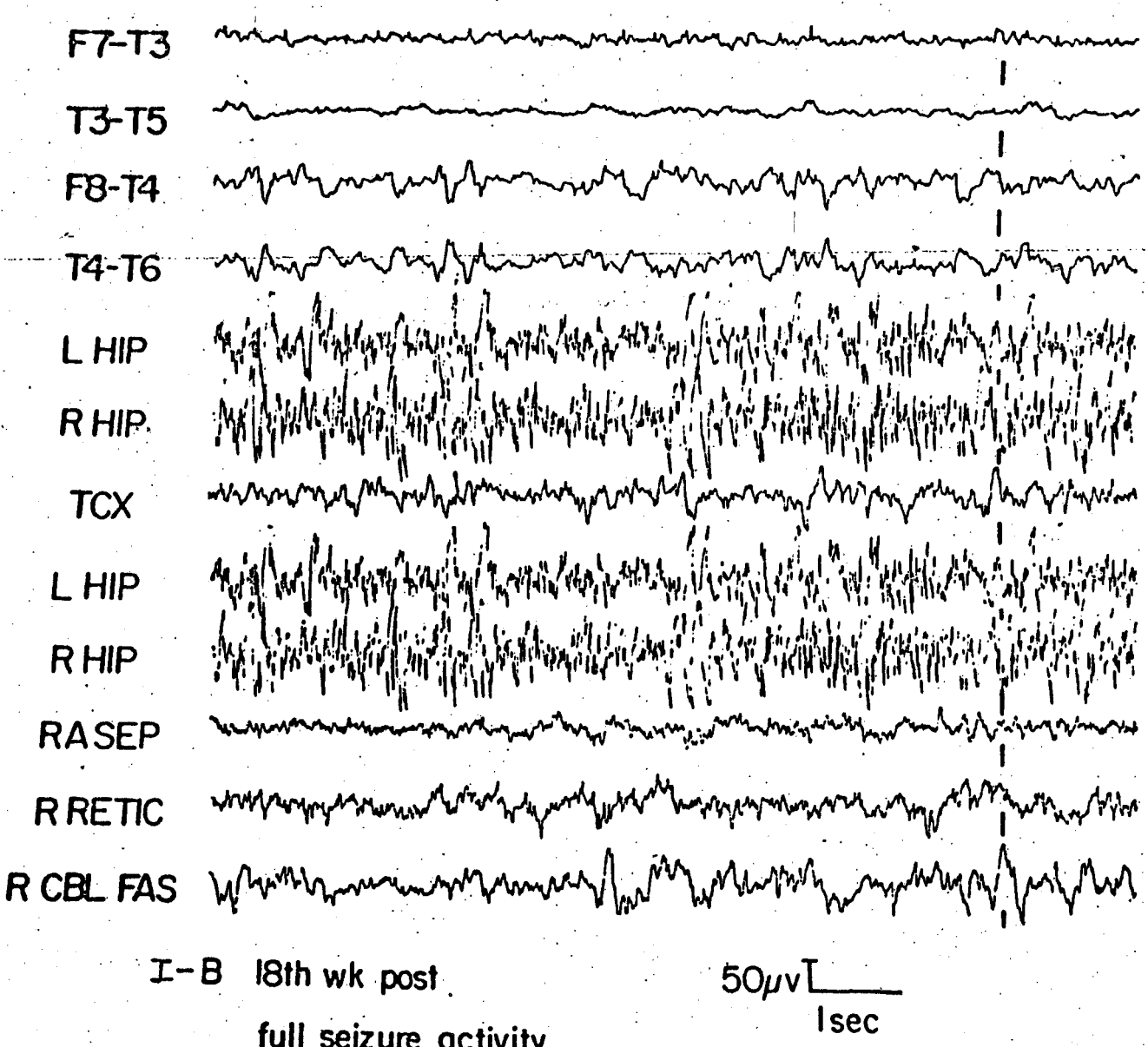


Fig. 6 The EEG of animal I-B 18 weeks post-whiplash illustrating the complete independence of the spiking foci (R HIP and L HIP). Furthermore, the "seizure" activity was practically continuous.

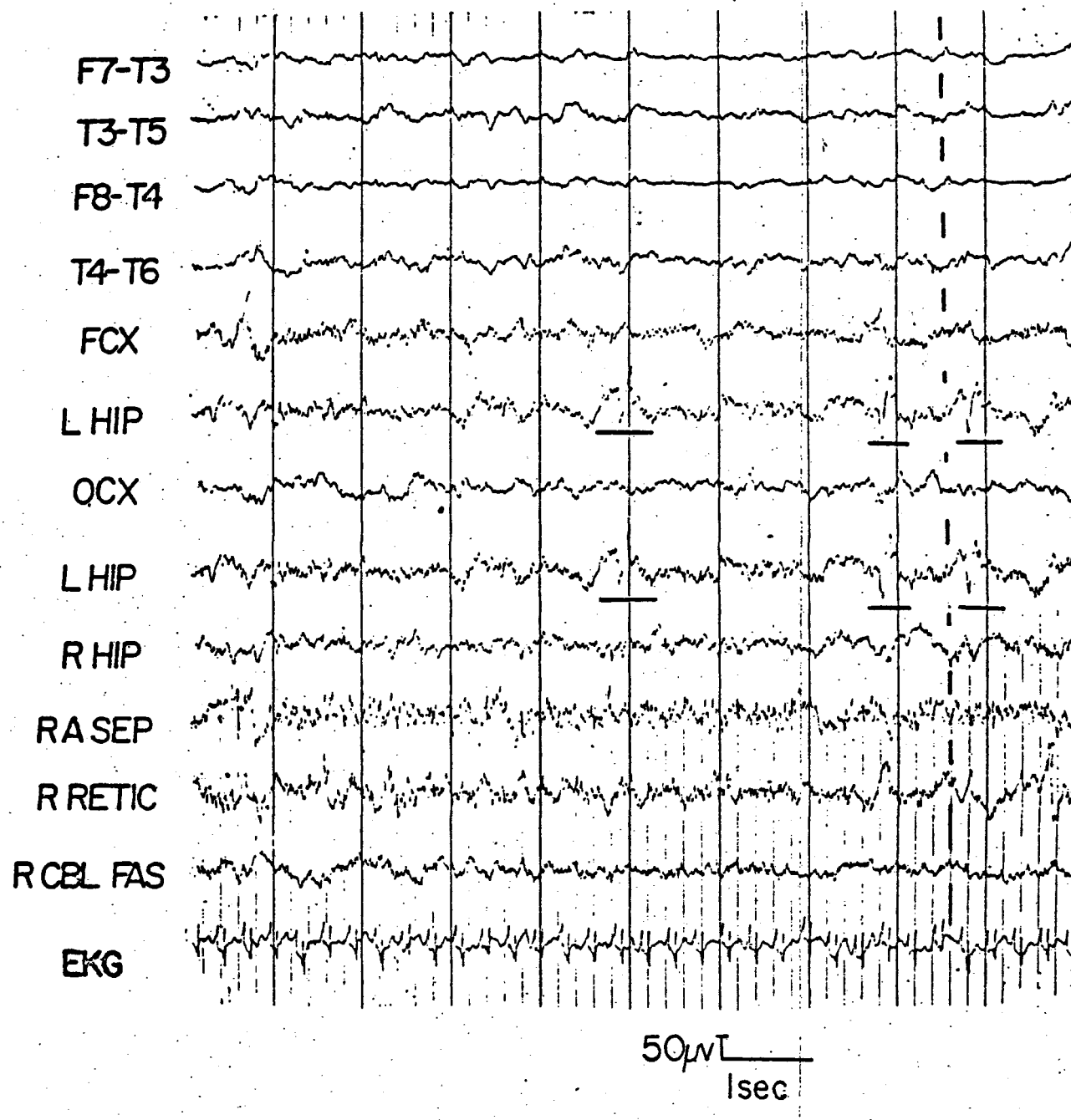
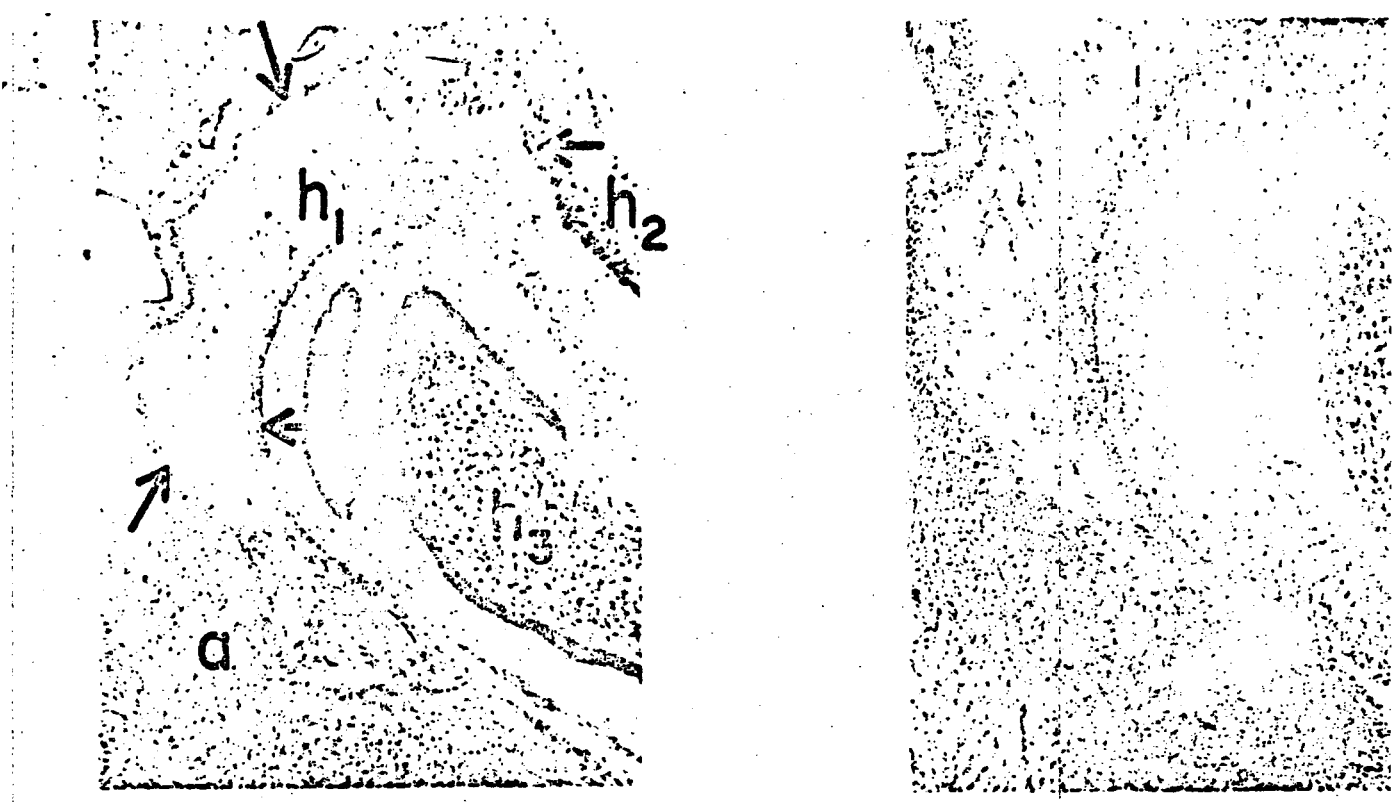


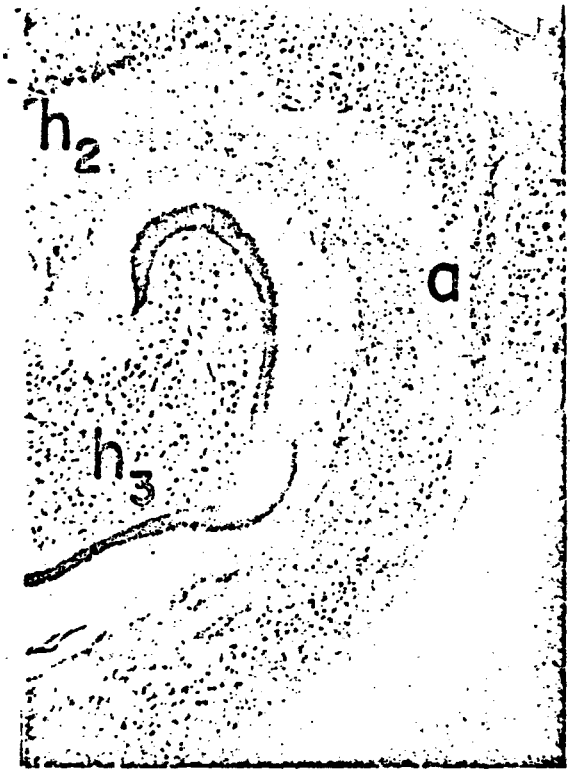
Fig. 7 The EEG of animal II-B 9 weeks post-whiplash during sleep. Note the number of spikes is typically 3 spikes per 10 seconds of sleep record. No mirror focus phenomenon.



(a) (b)

Fig. 8(a) Ammon's horn formation. Endpoint of electrode tract is Sommer's sector (h_1) at a. Traumatic necrosis of the entire area of Sommer's sector (h_1). Only a few neurons are visible in the region surrounded by the arrows. Spielmeyer's sector (h_2), and Bratz' sector (h_3) are intact. Cresyl-violet; 30:1. Animal I-B.

Fig. 8(b) The electrode tract shows hemosiderin laden macrophages and a marked glial-mesenchymal scar. The area of the traumatic necrosis contain debris laden macrophages and gitter cells. A few remaining neurons of the Sommer's sector (h_1) are shrunken and hyperchromatic. Cresyl-violet; 50:1. Animal I-B.



a

b

Fig. 9a Ammon's horn formation. Electrode tract extending into Sommer's sector (h_1). Marked local glial and mesenchymal reaction surrounding the tract at a. Neurons in Bratz sector (h_3), Spielmeyer's sector (h_2) and Sommer's sector (h_1) intact with the exception of a minimal loss of neurons with glial reaction in the immediate surrounding of the electrode tract in (h_1). Hematoxylin-eosin; 30:1. Animal II-B.

Fig. 9b The glial-mesenchymal scar surrounding the electrode tract in (h_1) is visible. Local loss of neurons with microglial and astroglial reaction in the area directly surrounding the electrode tract; otherwise intact neurons. Hemotoxylin-eosin; 50:1. Animal II-B.

Accident Report

On April 4, 1975 a Lockheed C-5A aft ramp together with the attached pressure door failed at approximately 23,000 ft. The structural failure resulted in a sudden decompression of the aircraft, and partial loss of control by the cutting of the number 1 and 2 hydraulic lines, the control cables to the tail, and the alternate electric trim and rudder yaw, Even though number three hydraulic system was not damaged, primary pitch and yaw control were lost due to damage to the control cables to the tail. Using the remaining controls available to them, right aileron, spoilers, and engine thrust; the pilots were able to maintain control of the aircraft by a combination of banking aircraft and thrusting the engines in order to keep a quasi-level descent for an emergency landing. A quasi-level descent being a series of dives and pull-ups until the aircraft was at landing altitide. The aircraft approached its first touch down point on the east side of the Saigon River. Just before touch down the engines were at full throttle to reduce the descent rate and the aircraft was at a slight roll angle. As the aircraft was touching down the engines were retarded to idle. The velocity of the aircraft as recorded by the MADAR DATA was approximately 270 knots (456 ft./sec.) approximately 3-4 seconds. The average wind velocity recorded at Saigon Airport at the time of the crash was 15 knots. The direction of the wind was approximately to the west, therefore the ground airspeed of the C-5A was about 283 knots (478 ft./sec.). The velocity is about <u>2-1/2</u> times the aircraft's normal landing velocity. It will be demonstrated later that the C-5A came to a complete stop in a <u>shorter</u> distance (~1900 ft.).

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than it does when it lands at its normal landing velocity (~2300 ft.). Therefore the aircraft impacted at 283 knots, and stopped in approximately 1900 ft.. A normal landing would be at approximately 110 knots. (190 ft./sec.) and would stop in about 2300 ft.

Impacts on the East Side of the Saigon River

As the C-5A approached its first impact point it had a velocity of 283 knots and the pilots had limited control of the aircraft. There was no record of the descent speed. The initial impact occurred when the aft landing gear struck a dike. The aircraft still lofting above the ground struck another dike this time more severely than the first. The landing gear dug into the soil for a short distance. The aircraft bounced up again and then settled back down hitting a third dike. The landing gear again dug into the soil. It is probable that two complete sets of landing gear were lost during or shortly after this impact. Photographs show wheels and pieces of the landing gear spread throughout this area. The C-5A bounced up again and made several small ruts with its engines or wing tips. The aircraft then hit another dike. Finally the C-5A became airborne again slicing several treetops off with its starboard wing. From the films it appears that there were at least eight or more distinct impact points east of the Saigon River. These multiple impacts all occurred in a distance of about 350 yards. The impacts were of sufficient magnitude (snapped off several pieces of landing gear) to have weakened part or all of the C-5A structure.

Impacts on the West Side of the Saigon River

The C-5A crossed the Saigon River at a probable velocity of 283 knots. This velocity may not be precisely relied upon and is believed to somewhat less because of the series of impacts encountered on the east side. The angle of attack of the aircraft (nose up or down) also cannot be estimated because of the lack of in-flight data. It is to be emphasized that the pilots had no control of the C-5A during any of the impacts.

The aircraft impacted on the west side of the Saigon River breaking the remainder of its landing gear off. The C-5A went into a sliding skid for about 175 yards. After this point the skid marks disappeared indicating that the aircraft may have lifted off the ground. The C-5A traveled about 150 yards and broke into four separate sections: the T-tail, the aft troop compartment, the flight deck and the complete wing structure. At this point of impact, large amounts of debris were found and a large section of the cargo floor was located. This is also the area where almost all of the dead were located. Northwest and about 100 yards away from the last impact point, the T-tail was found. The T-tail had a clean fracture indicating a sudden separation from the fuselage. It appears the tail was thrown over to its location as a result of the impact. The flight deck moved in a south-west direction and traveled approximately 400 yards from the impact. It appears that the flight deck traveled about 150 yards in the air and skidded to a stop in the remaining 250 yards. The wing structure also detached during the impact and through a combination of inertial (96,000 lbs. fuel) and lift forces was propelled approximately

525 yards from the point of last impact. The aft troop compartment became detached from between the wing section and the T-tail, and was propelled from the impact primarily by inertial forces and possibly some lift force. The troop compartment began digging into the ground approximately 175 yds. from the point of last impact. The aft troop compartment then came to a sudden stop after hitting an elevation. The total distance the troop compartment dug into the ground was approximately 2 lengths of the structure or about 40 yards.

The velocity of the four major sections were equal at the point of break-up. The velocity at this point has been estimated (see Appendix I) as 200 knots, (338 ft./sec.). The estimated "G" forces for the aft troop compartment, flight deck and the T-tail are summarized below (see Appendix I for details):

1. T-tail: After close examination of the photographs, it was concluded that the T-tail had been sheared off during the last impact. The minimum "G" force range required to break the tail off according to our engineering analysis with data from Lockheed reports is 11 to 15.

2. Flight Deck: Given the initial velocity as 200 knots, and the measured slide path of the flight deck, an average "G" force range of 6-8 has been estimated.

3. Aft Troop Compartment: The aft troop compartment had an initial height at the point of break-up. The height combined with a velocity of 200 knots turned the aft troop compartment into a projectile. The troop compartment was airborne as indicated by the photographs for approximately 175 yards, and smashed down onto the ground at the end of its trajectory. The average vertical "G" force range was estimated to be 10g-30g. The aft troop compartment then started digging into the ground and came to a sudden stop by hitting a small hill. The average estimated horizontal "G" force range during the deceleration was 7-13. At the point of impact with the hill, the estimated horizontal "G" force range was 220 to 480. It is obvious from the engineering analysis that the "G" force environment in the aft troop compartment was extremely complex and severe.

Summary

In conclusion the C-5A had an approach speed of 2-1/2 times its normal landing speed. The pilot only had limited control of the aircraft before the crash and no control during the crash landing. The C-5A structure experienced a series of 8-12 impacts, some sufficiently severe to break off landing gear, on the east side of the Saigon River. Approaching the west side of the river the aircraft had a velocity of approximately 270 knots. The C-5A impacted and slid for a short time and

bounced into the air again and impacted again about 400 feet away, breaking up into four major sections, each moving at a velocity of approximately 200 knots. The aft troop compartment experienced a severe and extremely complex "G" force environment. The engineering analysis of Appendix I demonstrates the complexity involved in attempting to calculate the "G" force environment. The assumption of constant deceleration over the complete crash site cannot be used. The average "G" force ranges in Appendix I are all based on conservative assumptions. Peak "G" forces, greater than what is calculated, undoubtly exist; but cannot be calculated.

Appendix I

Estimation of Deceleration Levels

In order for some of the passengers and crew to survive the crash, the airframe and ground had to absorb the energy of the airframe/passengers/crew in a manner that made their survival possible while removing the danger of a post landing fire. This energy absorption was accomplished over several definable periods of time/distances. One method of characterizing the events that occurred during the time from aircraft touchdown to points where the various parts came to rest would be to develop the deceleration time history. As indicated by the location of the major parts in the photographs of the crash site, the aircraft was subjected to complex set aerodynamic, inertia, and frictional forces. These complex forces thus would yield an equally complex deceleration time history. Since only the final position and an estimation of the initial conditions are known, it is not possible to evaluate but the simplest assumptions (constant deceleration) without some additional data. Even though not valid, the assumption of a constant deceleration for both the flight deck and aft troop compartment may be made after the aircraft breaks up. This assumption will yield a lower bound on the estimated maximum "G" load. Since only the initial conditions are known, additional data (structural failure) is needed to determine the intermediate conditions. Use of structural failures will only yield a lower bound on the applied loads/maximum "G's" since the rate of failure is not known.

The first step is to determine the form of the deceleration while the aircraft is intact, thus developing lift. The deceleration force is given by:

$$F = \mu (W-L) + D - T - D_p$$

Where:

μ = Coefficient friction

W = Weight

L = Lift = $1/2\rho V^2 C_L S$

D = Drag = $1/2\rho V^2 C_d S$

T = Thrust

D_p = Drag of landing gear post

ρ = Density of air

$\underline{v}$ = Velocity

C_L = Coefficient of lift

C_d = Coefficient of drag

S = Wing area.

The deceleration is given by:

$$a = 1/m\{F\} = 1/m\{\mu(W-L) + D - T - D_p\}$$

where : m = mass of aircraft

The deceleration can be written as:

$$\frac{dv}{dt} = a$$

or :

$$\frac{m \cdot dv}{\mu (W-L) + D - T - D_p} = dt$$

since L, D and D_p will be a function of velocity. The aircraft may be pitching, therefore both C_L and C_d may be a function of time, but they are assumed to change much slower than the velocity. Intergrating the above will allow an estimation of the form of deceleration versus time. A cubic variation of deceleration with time would be a good approximation for the above equation using the stated assumptions:

$$\frac{dv}{dt} = a = ct^3$$

where c = constant

yielding:

$$V_F - V_I = \frac{ct^4}{4}$$

where V_F = final velocity

V_I = Initial

and,

$$S_F - S_I = \frac{ct^5}{20} + V_I t.$$

where S_F = final position

S_I = Initial position

Next, the equations can be developed for the region in which a constant deceleration is to be assumed, as with the structural failure which is to be used with the above equation, this assumption will yield a lower bound on the maximum "G" estimate. For this assumption:

$$\frac{dv}{dt} = a$$

yielding:

$$V_F - V_I = at$$

and,

$$S_F - S_I = \frac{at^2}{2} + V_I t.$$

For the above formulations the distances are obtained from the referenced reports, velocities from referenced reports and calculations. The calculation of an intermediate velocity is made by assuming a minimum deceleration to fail parts of the structure. This must be done since there is one more unknown than equations available. The tail failure was selected because the normal flight loads on the tail are seen as bending movements/axial loads on the fuselage. The deceleration loads are also seen as bending movements/axial loads on the fuselage, thus, the loads needed to fail the fuselage are known (Figure I and Lockheed Reports). Since the axial force is now higher, several calculations on the magnitude of the failure load were made. The calculated "G's" for the failure load is 13 plus or minus 2. For the estimate "G" an intermediate velocity of 360 ft./sec. plus or minus 20 ft./sec. is obtained (Figure II). The aft troop compartment will require about one and a half seconds to travel from the break up point to its final touch down point, about the same time to travel from its break up height to the ground. At the point of final impact the aft troop compartment will experience 10-30 vertical maximum "G's". The horizontal maximum "G's" will depend on the method used. If it is assumed that the aft troop compartment would have gone as far as the forward flight deck if the hill had not been present, then the average "G's" would have been about 7 (Figure III) with a much higher "G" level when the hill is impacted, (Figure IV). If the average "G" is calculated then the "G" is about 13. Since some of the seats containing children failed, it is possible to estimate a level of horizontal "G" loading in the aft troop compartment. Depending on the weight of the children, the horizontal loading would range from 60-85 "G's", (Figure V).

Summary

Within the assumptions made, the following maximum "G" loadings have been calculated, these would be mimimum values since the time rate of failure is not known.

Horizontal

11 - 15 before break-up (use of structural failure)

7 - 13 after break-up (integration)

60 - 80 after break-up (use of structural failure)

Within the assumptions made, the following "G" loadings has been estimated for various impacts.

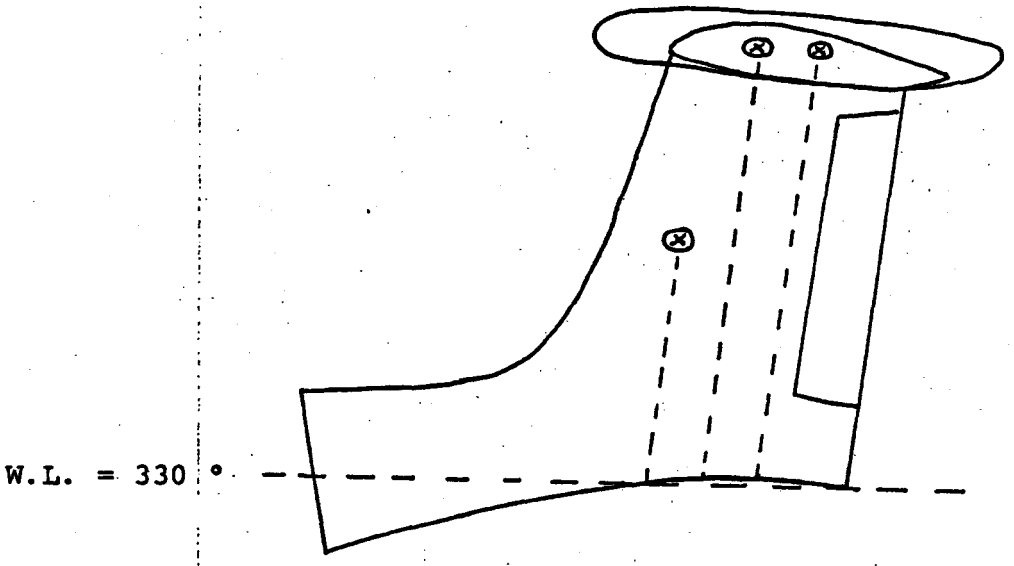
Horizontal

60 - 80 after break-up (use of structural failure)

220 - 480 after break-up (integration)

Vertical

10 - 30 after break-up (integration)



Item	Weight	$\bar{X}$	$\bar{Y}$	$\bar{Z}$	Mass
Bullet	769.4	2895.6	0.0	786.6	23.9
H.S.	3275.4	2921.3	140.1	787.5	101.7
V.S.	6151.0	2786.6	-0.1	633.1	191.02

$F = m(a) = [\ 23.9 + 203.4 + 196]\ a = 428.3(a)$ 15.

$M = 23.9\ (456.6)\ a + (2)\ (101.7)\ (457.5)\ a + (191.02)\ (303.1)a$

$\bar{M} = 1.619 \times 10^5\ (a)$

Figure I - Sample Calculation

Deceleration given by

$$a = ct^3$$

where $a = -13$ "G's" $= -416$ ft./sec^2

$$V_F - V_I = \frac{ct^4}{4} = \frac{-416t}{4} = -104t$$

$$V_F = V_I + \frac{ct^4}{4}$$

$$S_F - S_I = V_I t + \frac{ct^5}{20} = V_I t - \frac{416t^2}{20}$$

$$-20.8t^2 + V_I t - (S_F - S_I) = 0$$

$$t = \frac{-V_I \pm \sqrt{(V_I)^2 - (4)(20.8)(S_F - S_I)}}{(-20.8)(2)}$$

, where $S_I = 0$

$S_F \approx 500$ ft.

$V_I \approx 463$ ft./sec

$$t = \frac{V_I \pm \sqrt{(V_I)^2 - (4)(20.8)(S_F)}}{41.6}$$

$t \approx 1.2$ sec. $V_F \approx 360$ ft./sec.

Figure II - Sample Collections

$$V_F - V_I = at$$, where $V_F = 0$

$V_I = 360$

$$S_F - S_I = \frac{at^2}{2} + V_I t$$

$S_F \approx 750$ ft.

$S_I = 0$

$$S_F - S_I = \frac{a}{2} \frac{V_I^2}{a^2} - \frac{V_I^2}{a}$$

$$a = \frac{-(V_I)^2}{(2)(S_F - S_I)}$$

Deceleration would be $7.0 \pm .5$

$$t = \frac{V_I \pm \sqrt{(V_I)^2 + 2a(S_F - S_I)}}{a}$$

$t \approx 0.5$

Velocity at Impact : $V_F = V_I + at$

$V_F = 250$ ft./sec. $\pm$ 10 ft./sec.

Figure III – Sample Calculations

$$"G" = \frac{(V_I)^2}{64.4(\Delta X)}$$

ΔX = penetration into hill

ΔX = 2, 3, 4 ft.

then $"G_2" = 460 \pm 20$

$"G_3" = 320 \pm 20$

$"G_4" = 240 \pm 20$

$$t_2 = \frac{V_I}{(32.2)\ "G_2"} = \frac{250\ ft/s}{32.2\ ft/s^2(460)} \simeq .0169 \text{ sec or } 16.9 \text{ millisec}$$

$$t_3 = \frac{V_I}{(32.2)\ "G_3"} = \frac{250\ ft/s}{32.2(320)} \simeq .0243 \text{ sec or } 24.3 \text{ millisec}$$

$$t_4 = \frac{V_I}{(32.2)\ "G_4"} = \frac{250}{32.2(240)} \simeq .032\$ \text{ sec } 32.4 \text{ millis}$$

Figure IV - Sample Calculations

Seat is designed to transfer:

2500 lb horizontal

1250 lb vertical

375 lb lateral

to the floor beam without failing. For seat to fail in the horizontal direction with children, the "G" would be given by

$$G = \frac{2500}{(N)W_c}$$

N = number of children

W_c = Weight per child.

$$G = 60 - 85,$$

actual value would depend on the weight of the children. Note: the moment arm has not been adjusted for the children so the above estimates would be less than the actual values.

Figure V - Sample Calculations

Appendix II

Review of Reports by J.W. Turnbow and John W. Edwards

- Turnbow indicated that all seats remained attached and were facing rearward. Not all the seats in the aft troop compartment were facing rearward, and evidence presented indicated that some seats failed, but remained partially attached.

- The density of the wreckage and its location does not agree with the concept of many successive failures that Turnbow and Edwards hold to. In fact the wreckage diagram indicates that the aircraft parts were not always in contact with the ground.

- Structural failures in the aft troop compartment and the T-tail are inconsistent with the "G" forces they calculate.

- Photographs of the crash site and their description of the site are inconsistent. Example: rise the aft troop compartment is resting against.

- The amusement ride comparison with the C-5A crash analogy given by R.D. Jablonsky, Inc. cannot be compared. An amusement ride is a controlled uni-directional recreational vehicle. It produces constant accelerations and decelerations and is designed for safety. The C-5A crash was an uncontrollable large mass moving at 270 knots on inconsistent terrain. Multiple directional "G" forces were experienced with extremely high and uneven peaks.

Material Review

- USAF Collateral Report, Vol. I, II, III.
- Photographic documentation (still and motion picture) of the accident site and wreckage.

- John Edwards
  - * Trial Testimony - 5-21-80
  - * Trial Testimony - 3-11-80
  - * Deposition
  - * Report - Crash of AF68-218 C-5A on 4 April 1975

- James Turnbow
  - * Deposition - 8 October 1981
  - * Report - Analysis of "G" Levels Associated with the C-5A accident near Saigon - April 4, 1975.

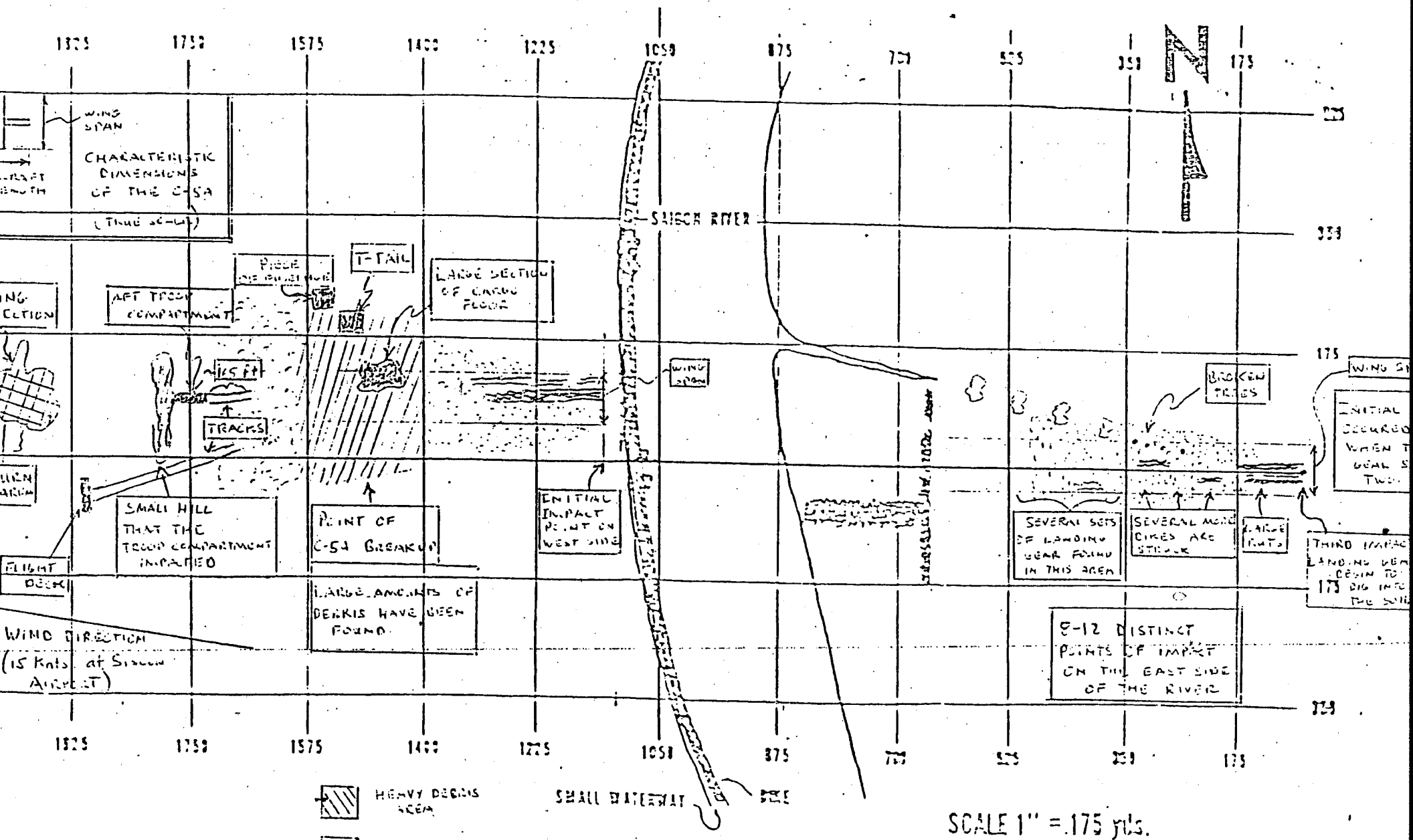
- William Timm
  - * Trial Testimony 3-18-80
  - * Trial Testimony 5-12-80

  Personnel Location Defendant's Exhibit D1210

- Lockheed Reports
  - Report LG1US-46-2-2
  - Report LG1US-54-12-1
  - Report LG1US-44-1-2
- Article by Jane's - C-5A aircraft
- Photogrammetric Measurements and Soil/Vegetation Interpretations Related to the C-5A Incident by Dr. Stanley A. Morain.
- Failure of the C-5A Aft Pressure Closure by Joseph F. Tilson.

WRECKAGE DIAGRAM

C-5A SN 68-218 4 APRIL 1975



PHOTOGRAMMETRIC MEASUREMENTS AND SOIL/VEGETATION INTERPRETATIONS RELATED TO THE C-5A INCIDENT

Prepared by

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for

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DEFT. EX. [redacted]-110 #12
DATE: 11/3/81
REPORTER: A. J. GASDOR

October 16 to November 22
1981

Table of Contents

INTRODUCTION

This report addresses three questions regarding the 1975 crash of a Lockheed C-5A aircraft near Saigon, South Vietnam. Said aircraft apparently developed mechanical problems shortly after takeoff from Saigon on the afternoon of April 4 and while attempting a return landing crashed a few miles northeast of the airport in an area devoted to rice paddy culture. After making a series of very brief contacts with the ground on the east side of the Saigon River (heading approximately 275°), the aircraft became airborne for a distance sufficient to overfly the river (see Plaintiff's Exhibit 3h reproduced here as Figure 1). It made a second major contact immediately west of the river and later lost its structural integrity and separated into several units, each of which came to a final rest at some distance from the others. One of the units, called the "troop compartment," but also described as "compartment E" on Lockheed diagrams of the C-5A configuration, is the focus of this report (see Defendant's Exhibit D1217 reproduced here as Figure 2).

By analysis of low altitude aerial oblique photographs taken shortly after the accident, (within 24-48 hours), the first question involves measurement of the lengths and depths of tracks produced by the aircraft's landing gear at its main points of impact on both the east and west side of the Saigon River. Measurement of the length and depth of what appear to be "skid marks" made by the troop compartment after

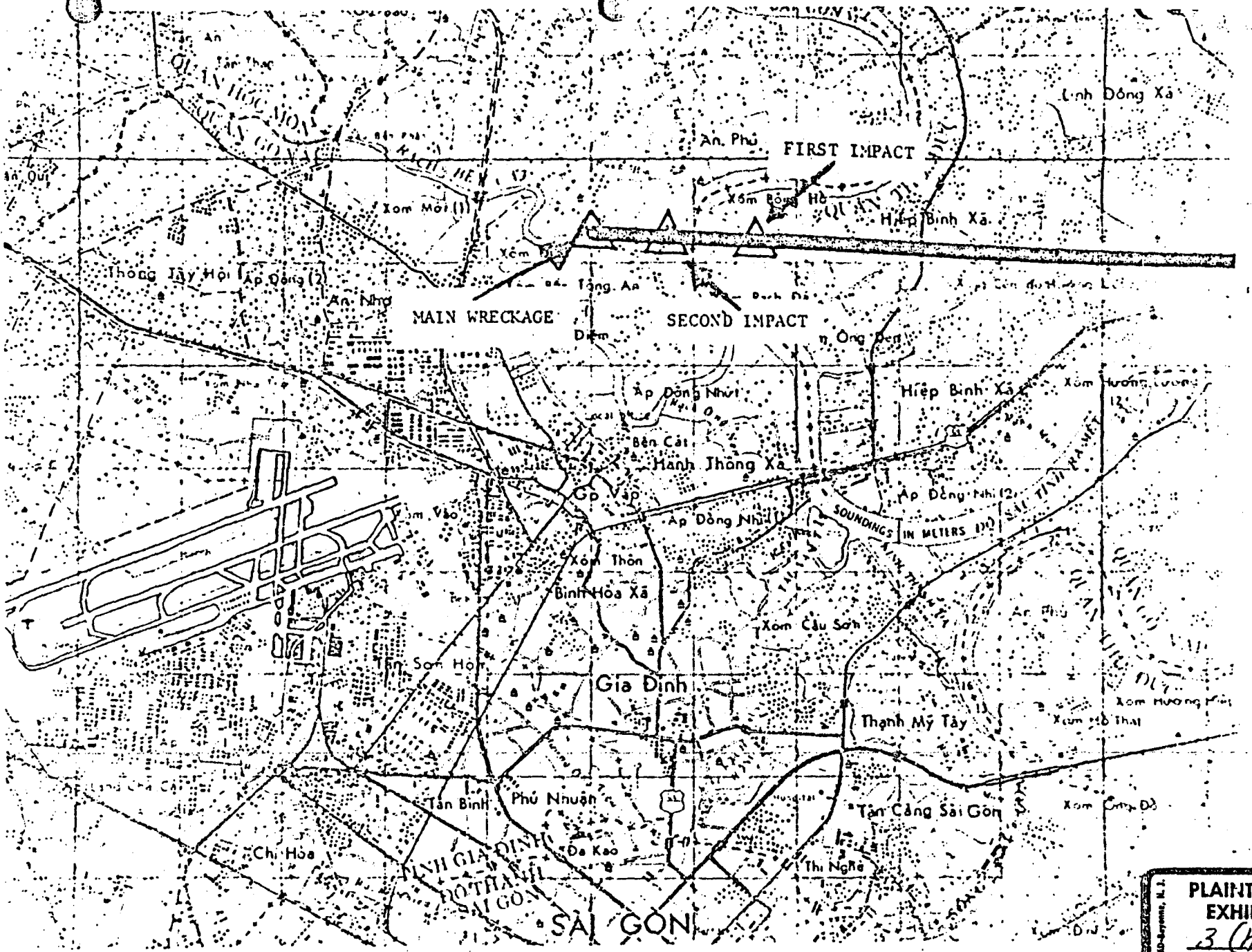


Figure 1: Heading and Location of C-5A Impact

PLAINTIFF'S EXHIBIT 3(h)

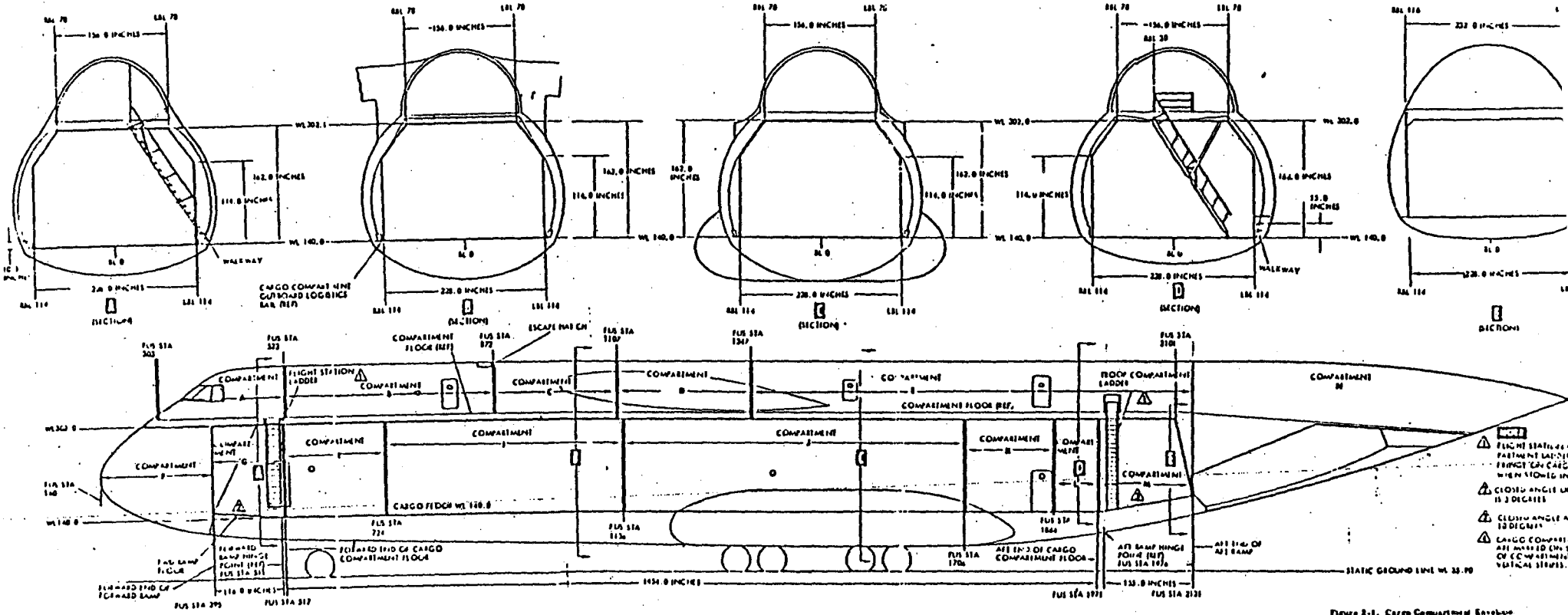


Figure 3-1. Cargo Compartment Envelope

it separated from the main frame are also reported. The second question relates to soil/water relations along the ground track of the aircraft, and an explanation of the topographic conditions in the vicinity of the troop compartment. Lastly, the third question involves an assessment of vegetational conditions with specific regard to patterns and discolorations observed on the photographs.

All of the measurements reported in Section I of this analysis were made directly from 5" x 7" black-and-white prints provided to the author by the firm of Lewis, Wilson, Lewis and Jones. As a convenience to the reader, the annotated prints included in the following pages are 8" x 10" enlargements of those same photographs, which, for purposes of inventory control, have been given the following numerical designations:

Walker Proof Set #	Morain #
748	14
257	15
269	16
736	18
321	19
735(?)	17
767	7
335	8
338	9
262	2
340	1
762	3
761	4
757	4A
187	5
83	6
300	6A

Measurements were made using a Bausch and Lomb 7-power hand magnifier having a 0.1 millimeter (mm) etched scale. Photo lengths longer than 2 centimeters (cm) were made with a National Bureau of Standards 15 centimeter scale. Calculations were performed with a Canon Card LC-61T hand calculator.

SECTION 1: PHOTOGRAMMETRIC MEASUREMENTS

I. Point of First Touch Down - East of Saigon River

A. Length of Main Tire Tracks (calculated to be 100-110 feet long)

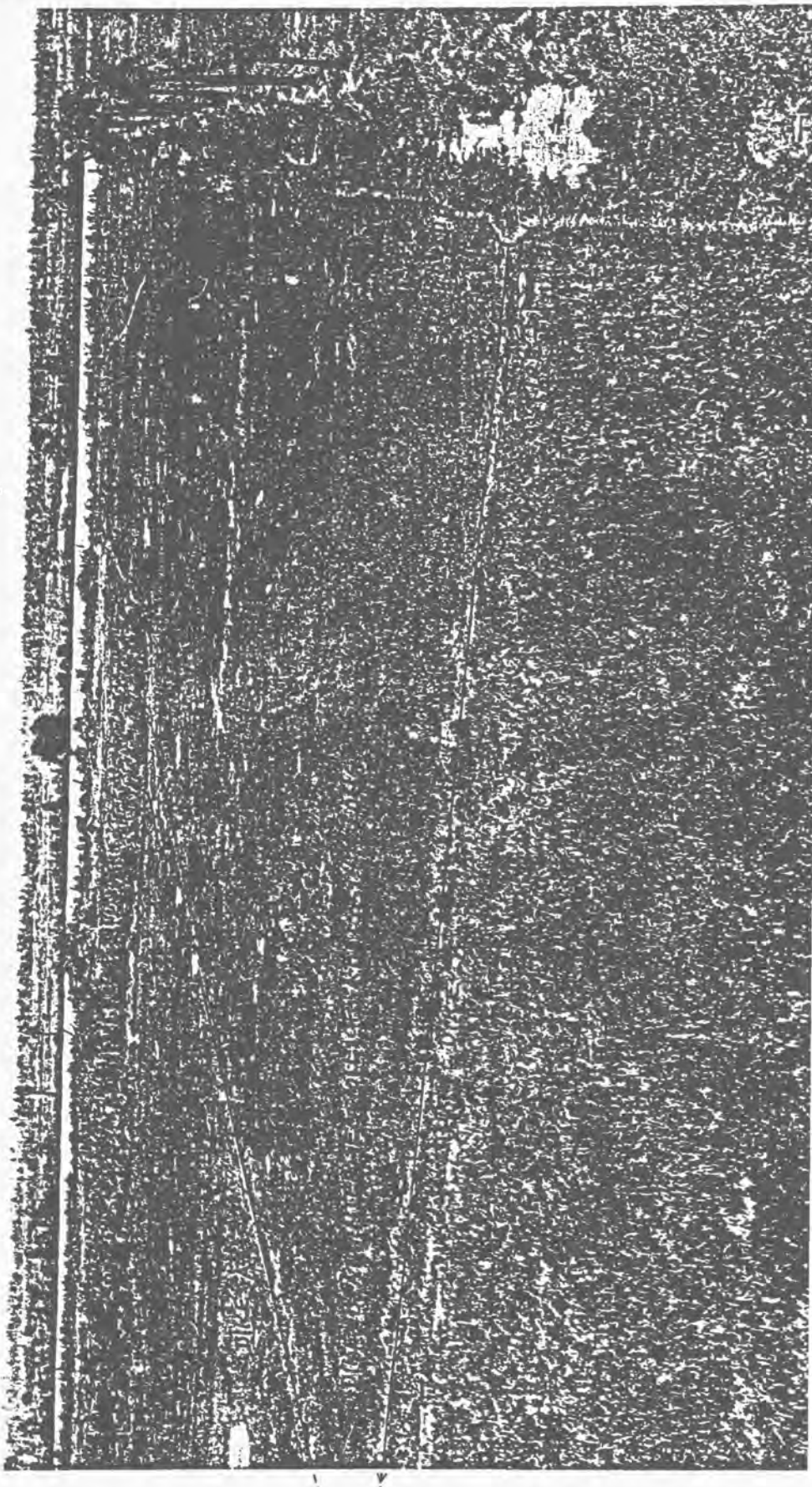
On the photo marked Morain #14 (Walker 748) the tire located in corner of field at right middle foreground was used to measure the field boundary running roughly orthogonal to the aircraft's flight path (Figure 3). On Defendant's Exhibit D1216 (Figure 4), tire height (diameter) is given as 3.75'. This is equal to .4cm on Morain #14. By intersection of the paddy borders to the left of the photograph a border length of 15.7cm is obtained; therefore, by proportional measurement

0.4cm:3.75' as 15.7cm:length of paddy dike = 147'

Since this border is not parallel with the bottom edge of the photo, a cosine correction can be applied. The angle is so small, however, ($< 10^{o}$) that for these purposes the cosine approaches unity and the border can be assumed to be somewhere between 145' and 150' long.

On the photo marked Morain #15 (Walker 257) and shown here as Figure 5, the field border measured on photo Morain #14 is

Figure 3: Morain #14 (Walker 748)



NOTE

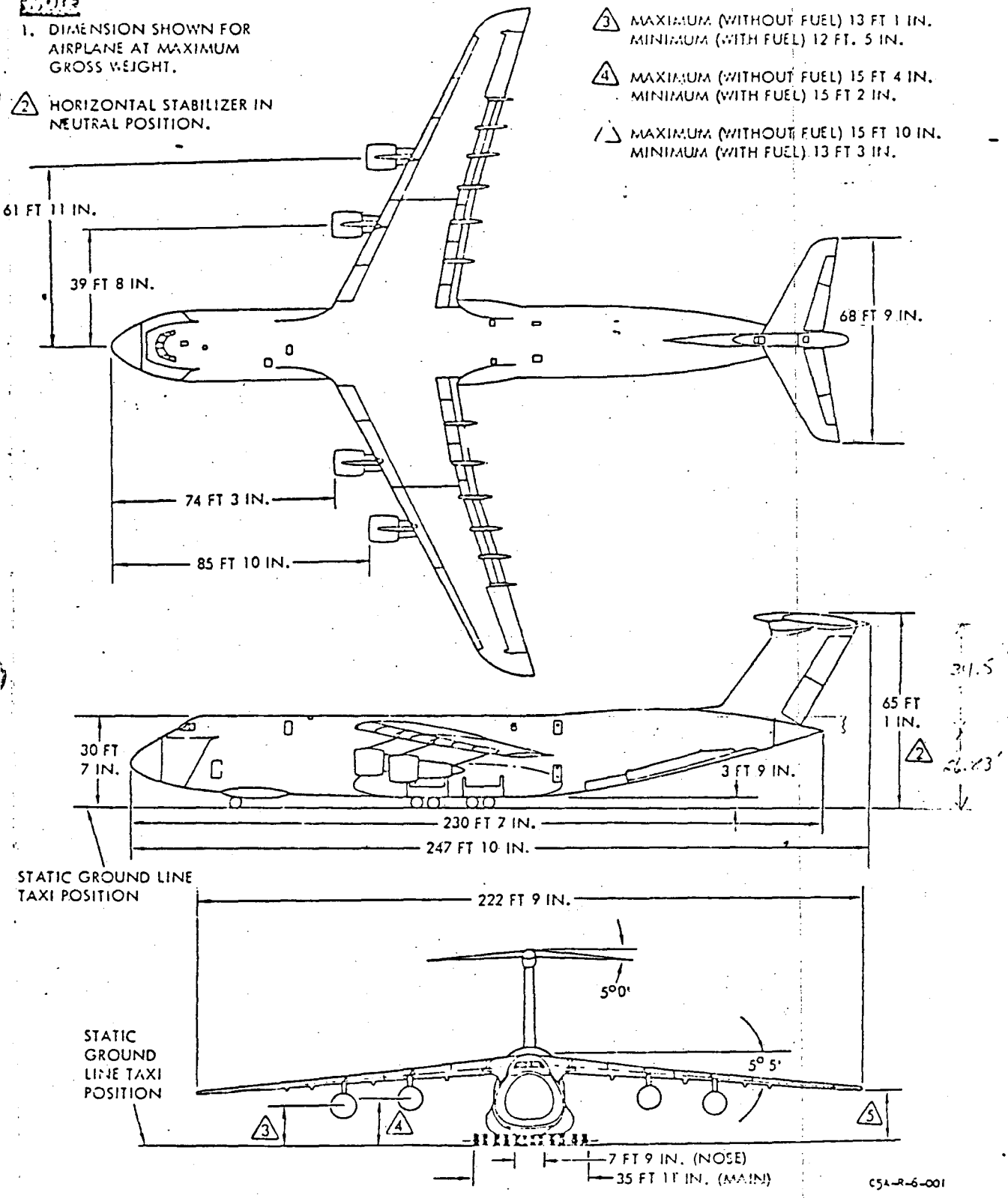
1. DIMENSION SHOWN FOR AIRPLANE AT MAXIMUM GROSS WEIGHT.

2. HORIZONTAL STABILIZER IN NEUTRAL POSITION.

3. MAXIMUM (WITHOUT FUEL) 13 FT 1 IN. MINIMUM (WITH FUEL) 12 FT. 5 IN.

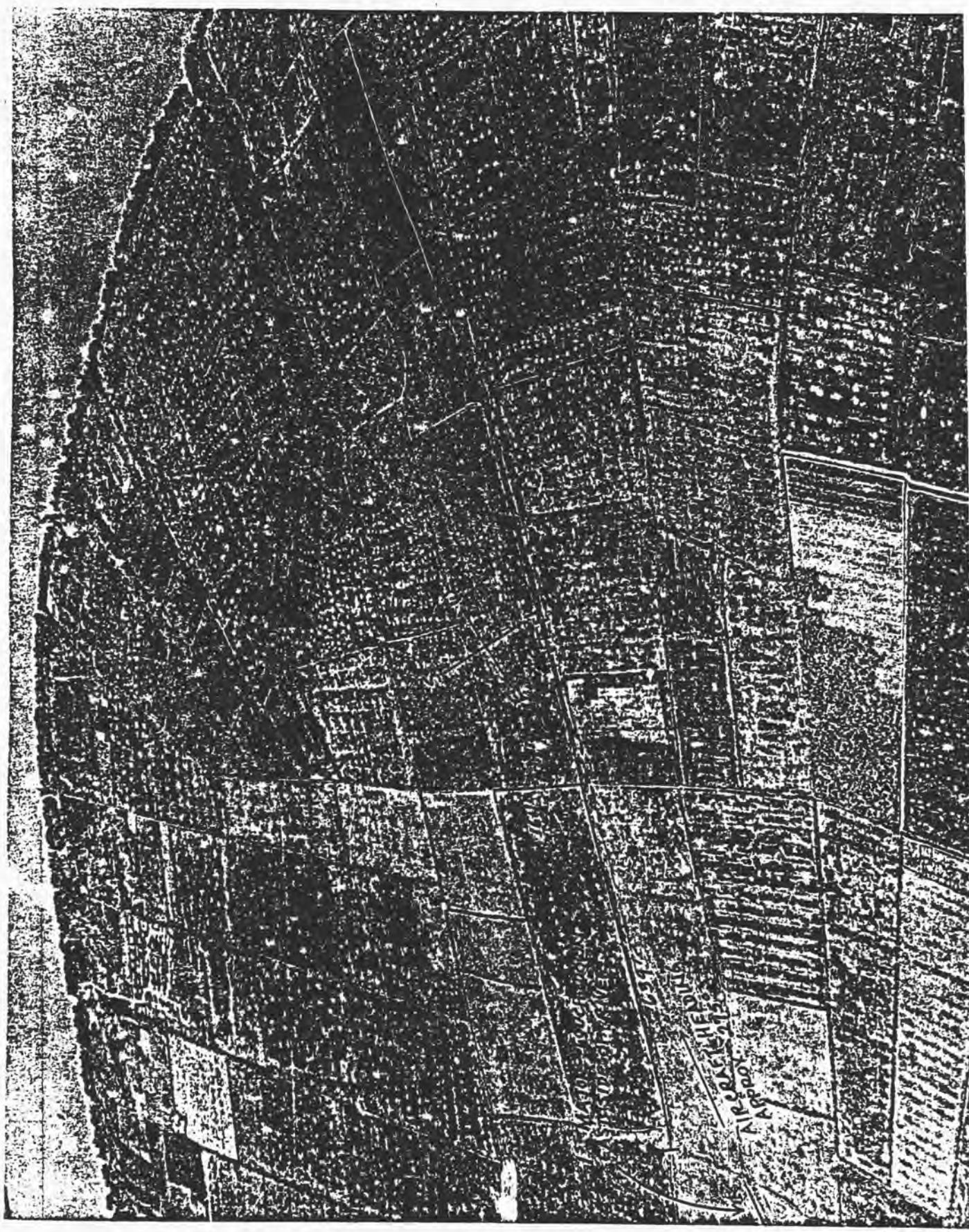
4. MAXIMUM (WITHOUT FUEL) 15 FT 4 IN. MINIMUM (WITH FUEL) 15 FT 2 IN.

5. MAXIMUM (WITHOUT FUEL) 15 FT 10 IN. MINIMUM (WITH FUEL) 13 FT 3 IN.



C5A-R-6-001

Figure 5: Morain #15 (Walker 257)



.94cm long. The aerial view on #15 indicates that its extended length is 1.05cm. Therefore, the true length of the border is:

0.94:147' as 1.05cm:x = 164'

The field extending eastward from the above measured border measures 4.35cm.

1.05:164' as 4.35cm:x = 679' x cos. 20°(.94) = 639'

For confirmation of the above measurement the photo marked Morain #16 (Walker 269) can be used (Figure 6). The field border previsouly measured as 147' measures .82cm. The extended length is .91cm, therefore:

.82cm:147' as .91cm:x = 163', and....

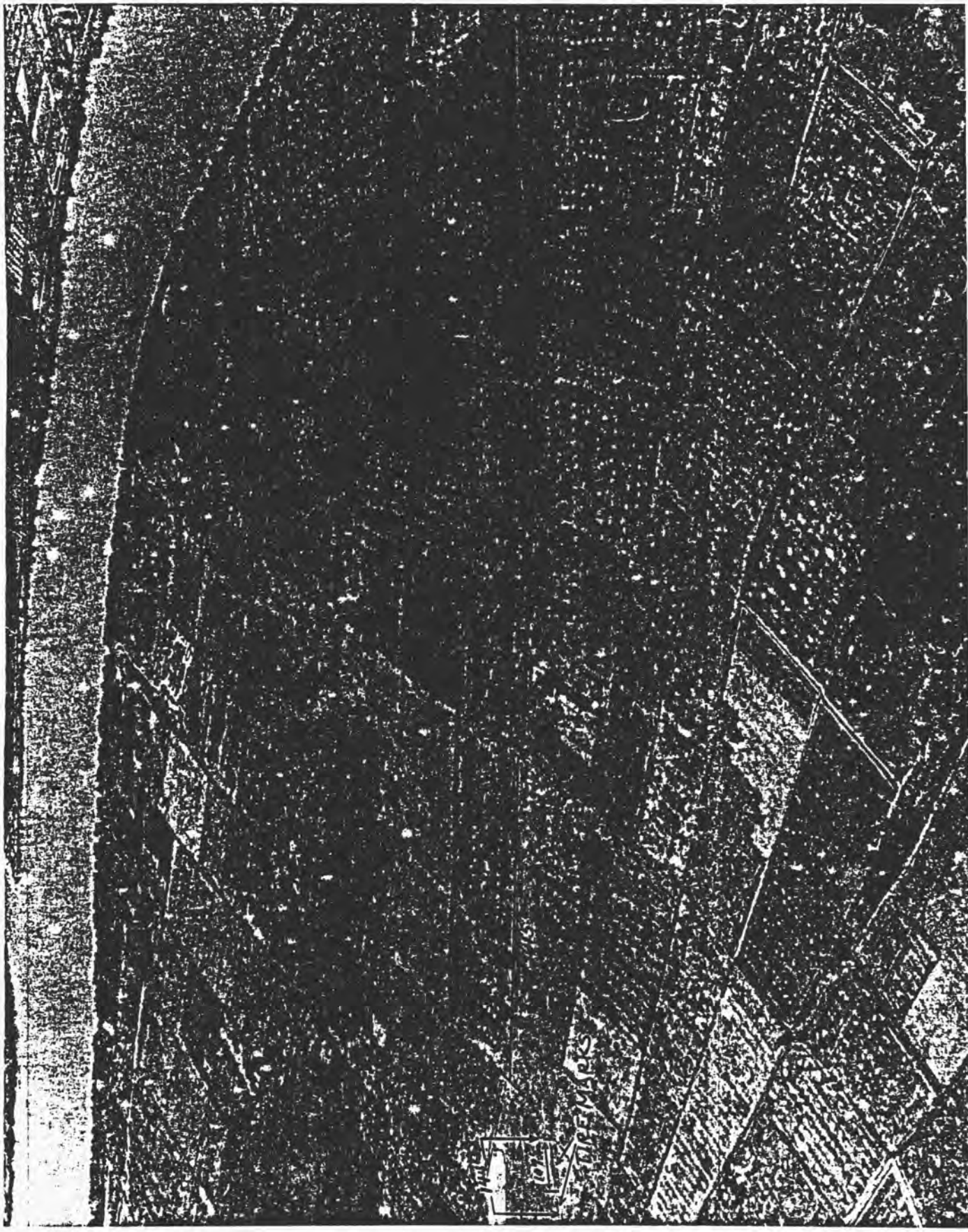
.91cm:163' as 3.65cm:x = 654'x cos.(.94) = 615'

From this same photograph the length of the main track on the east side of the river measures .60mm. This would make it approximately:

.91cm:163' as .60cm:x = 107' long

The shorter segment of the track, continuing eastward along the main track, is .20mm or about 36' long. Since these measurements all lie in nearly the same range of the photo marked Morain #16, no cosine correction needs to be applied.

Figure 6: Morain #16 (Walker 269)



B. Depth of Tire Tracks (calculated to be 25-30 inches at deepest point)

On Morain #14 (Walker 748), see Figure 3, the tire in the right foreground is known to be 17" (1.4') high (data taken from published statistics). The tire measures 0.1cm on the photograph and the dike immediately behind the tire is .12cm. Therefore, the height of the paddy dike is calculated to be

0.1cm:1.4' as .12cm:x = 1.68' or 20 inches

On the photo marked Morain #18 (Walker 736) and presented here as Figure 7, it is reasonable to assume that paddy dikes in the vicinity of the C-5A tracks average 15-20" in height. In some places, depending upon minor variations in relief they may only be 12" (1 foot). Morain #19 (which is Walker 321), Figure 8, is a ground photograph of tire marks shown in Morain #18 and Morain #17 (which is Walker 735 - Figure 9). The perspective of this ground based photo shows the relative height of the paddy dike in the immediate foreground in front of the inspecting officer, as well as behind him in the mid-distance. Tire marks in the field in which the two military personnel are standing appear to be merely scrapes having a depth of probably no more than 6 or 8". The tire marks go through the dike, as clearly indicated in Figure 9, and begin to penetrate the soil more deeply in the adjacent field. The absence of water in these grooves indicates that the depth of penetration was insufficient to initiate lateral seepage from the perched water table that characterizes these soils.

Figure 7: Moraln #16 (Walker 736)

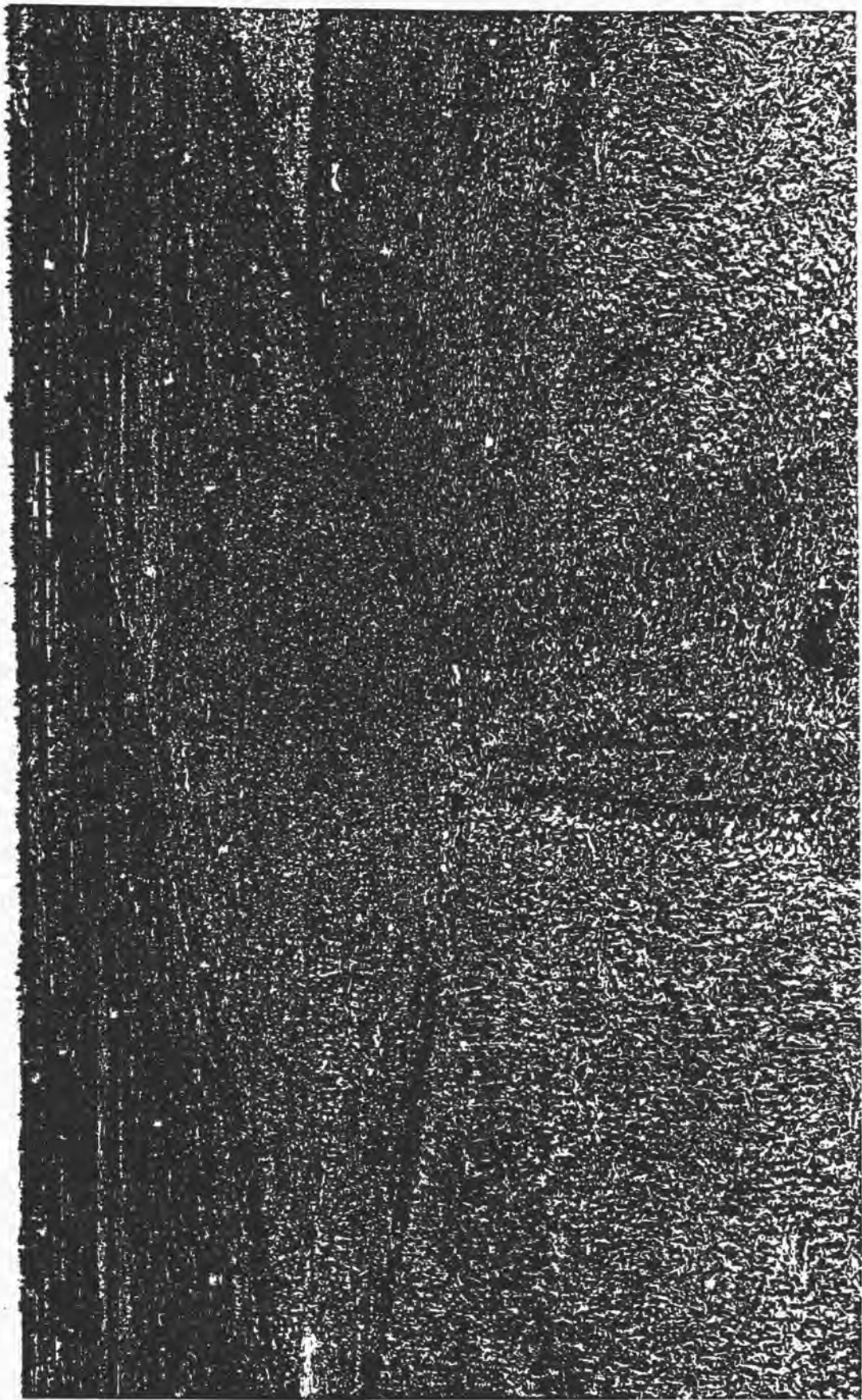
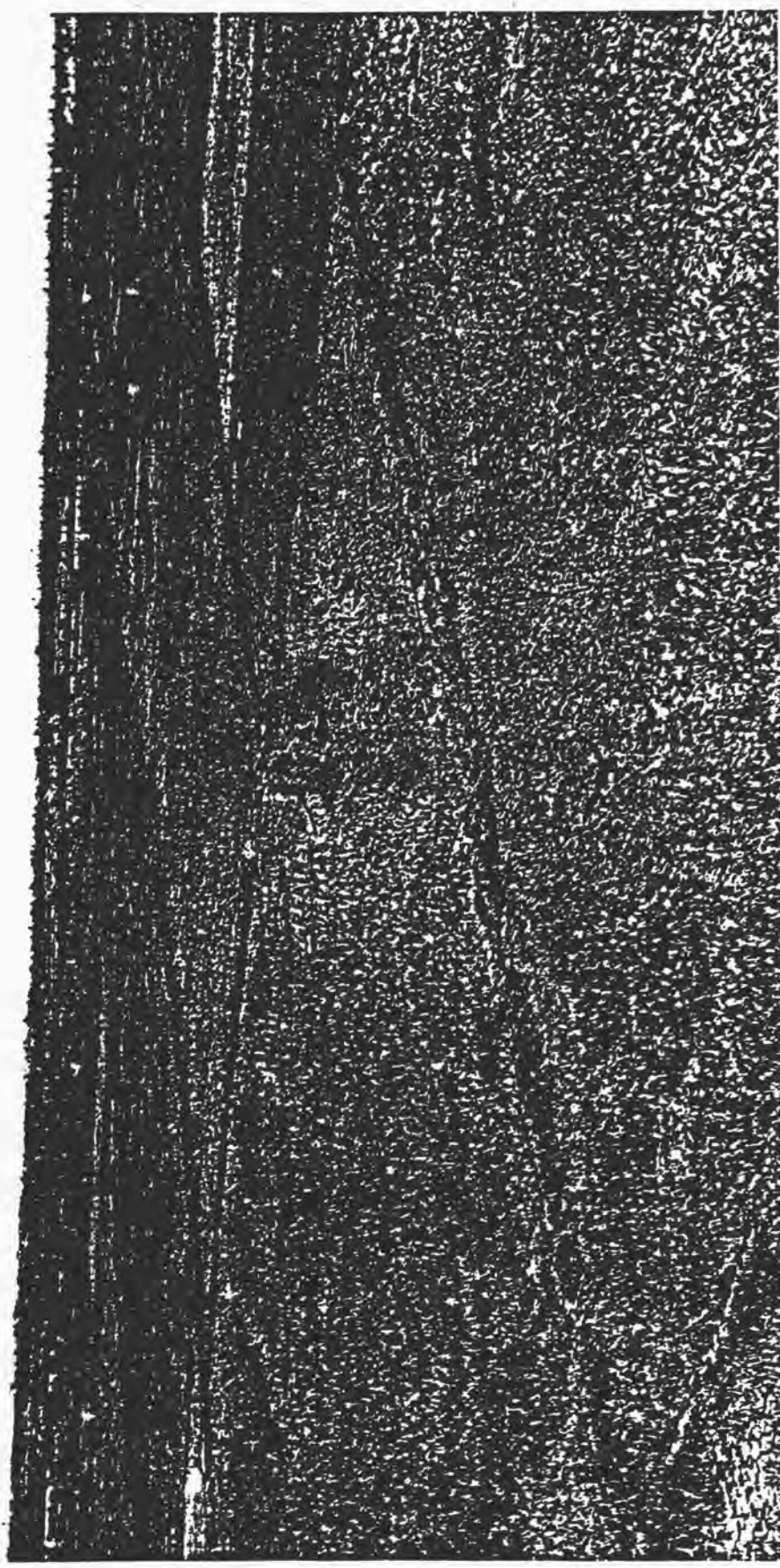


Figure 8: Morain #19 (Walker 321)

Figure 9: Morain #17 (Walker 735)



Measurements from the left track on Figure 7 indicate a depth of approximately: (assuming the dike in the foreground is 20" high)

0.15cm:20" as .2cm:x' = 27"

II. Point of First Touch Down - West Side of Saigon River

A. Length of Tracks (Procedure #1 - maximum 460')
(Procedure #2 - minimum 442'; maximum 576')

Procedure #1

Based on an estimated height of the Vietnamese person located at point A in Figure 10, which is Morain #7 and Walker 767, and by intersecting the field boundaries to right of said picture, the length of the paddy dike is estimated to be 100 feet.* This is calculated as follows:

- Vietnamese person is estimated to be 5.33' tall. He measures 0.49cm on photo. Therefore....

  .49cm:5.3' as x:1' = .09cm. Therefore: 1' = .09cm

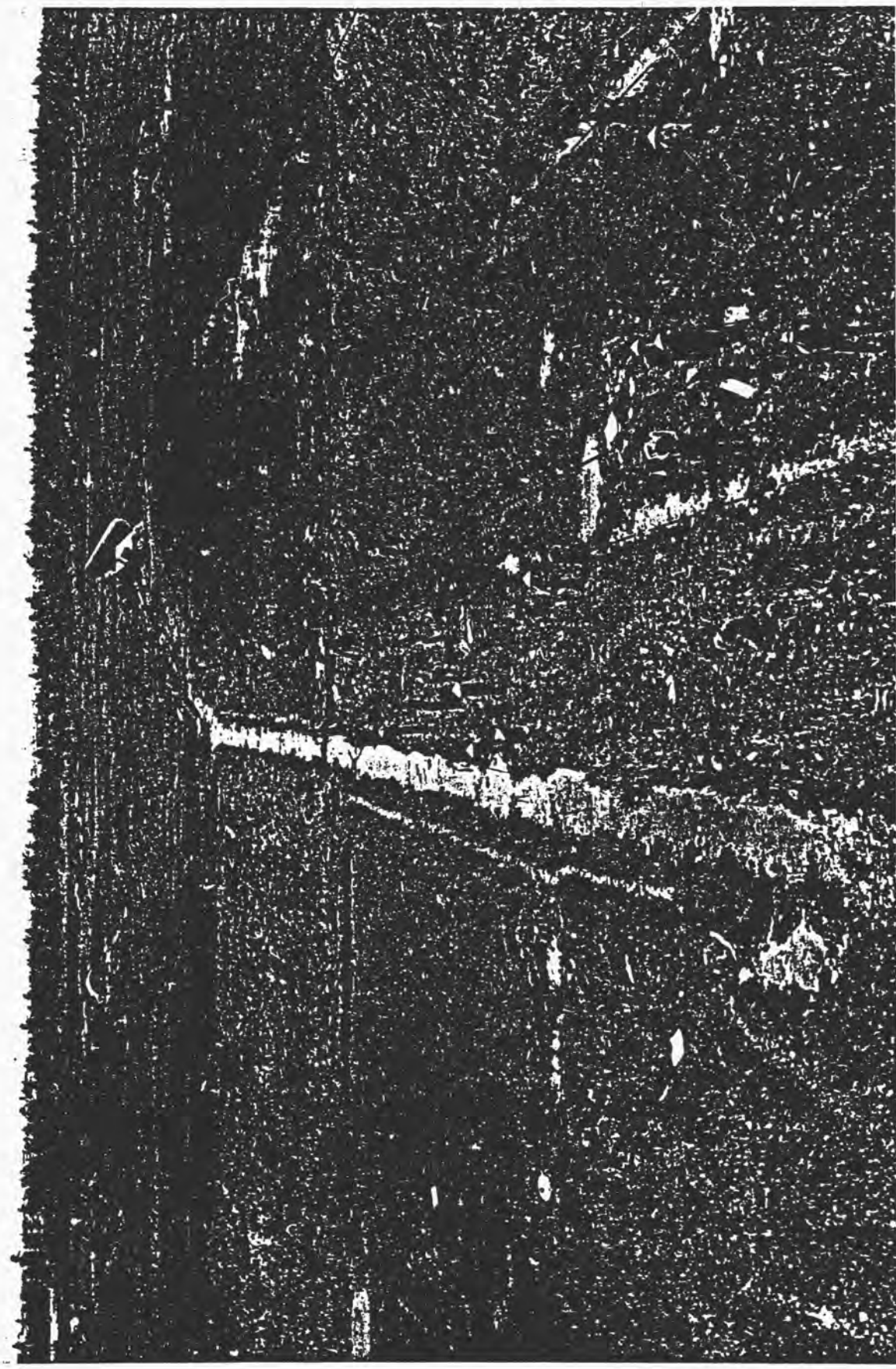
- If the field border is now measured from left (flooded channel) to right (intersection of field borders to right of picture), it measures 9cm. Therefore....

  .09cm:1' as 9cm:x' = 100 feet

---

* Figure 10 is cropped slightly differently from the photograph actually employed in the measurement phase. Extension of the boundaries to the right is not necessary in Figure 10 since the boundary intersection can be viewed directly on the photo. If the same procedures are applied to Figure 10, the length of the paddy dike is calculated to be 106 feet.

Figure 10: Morain #7 (Walker 767)



On photo marked Morain #8 (also Walker 335) and shown here as Figure 11, it is possible to extend the east-west running field border eastward to its intersection with the north-south border of the adjacent field (dashed line in Figure 11). If it is assumed that this intersection is 100 feet from the canal, as measured earlier, then it is possible to measure the length of the north-south border of the field as:

3cm:100' as 3.7cm:x = 123.3'

Also in Figure 11 there is a person (assumed to be Vietnamese) walking toward the C-5A track along the north-south trending drainage ditch in middle foreground (marked as point A on the photo). This person measures 0.29cm. Assuming the person is 5.3' tall....

.29cm:5.3' as x:1' = .55cm = 1'

If the field border along which the person is walking measures 6.5cm, the border is 118' long.

By visual inspection of the photo marked Morain #9 (also Walker 338) and shown here as Figure 12, the field border closest to the west bank of the river is estimated to be between 120 and 140 feet. From this combined evidence the track can be reconstructed as shown in Figure 13 and calculated to be 460 feet long.

Procedure #2

One can use the Army Topographic Command Map at 1:50,000 scale for verification of the above calculation. On this map

Figure 11: Morain #8 (Walker 335)

Figure 12: Morain #9 (Walker 338)

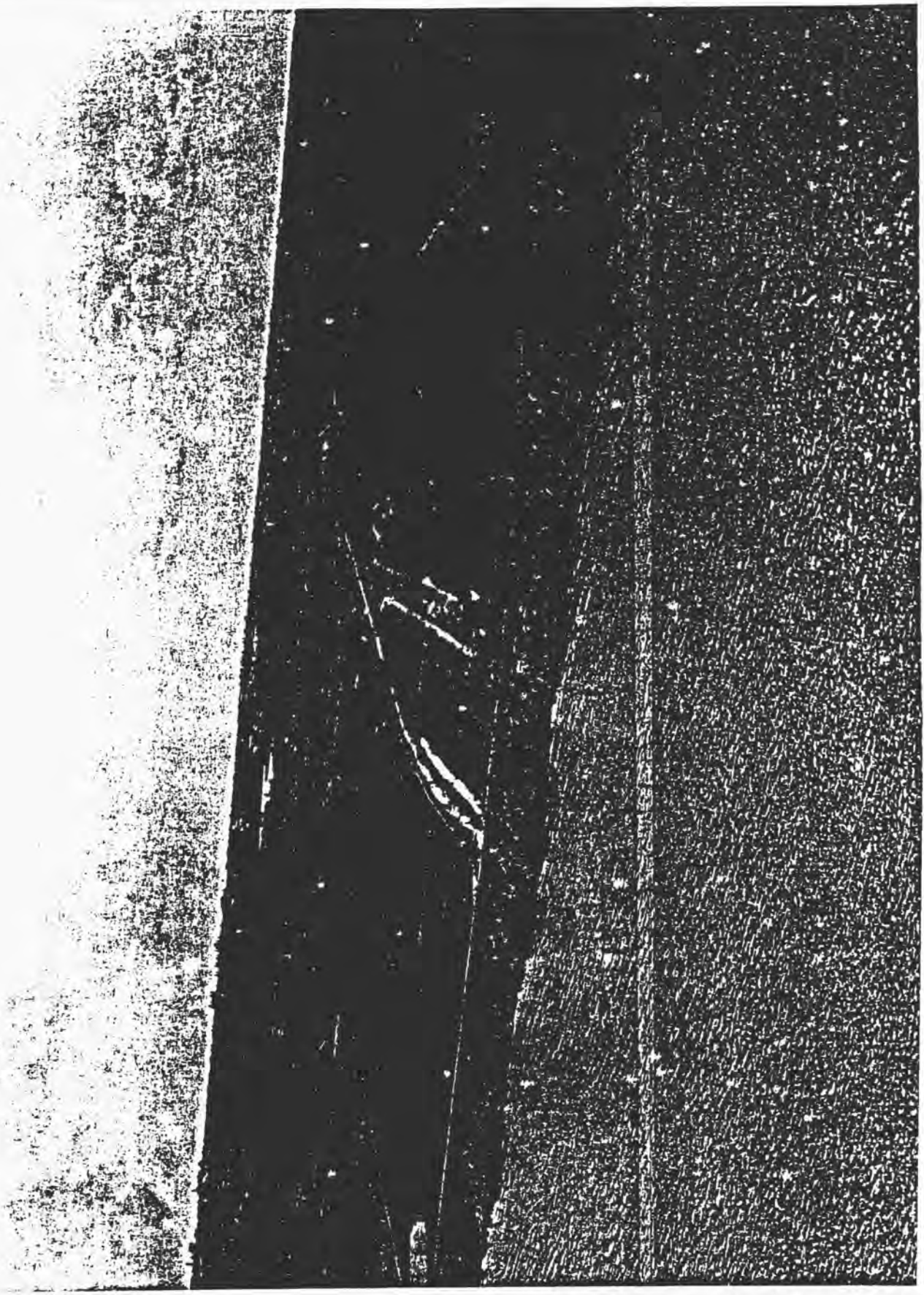
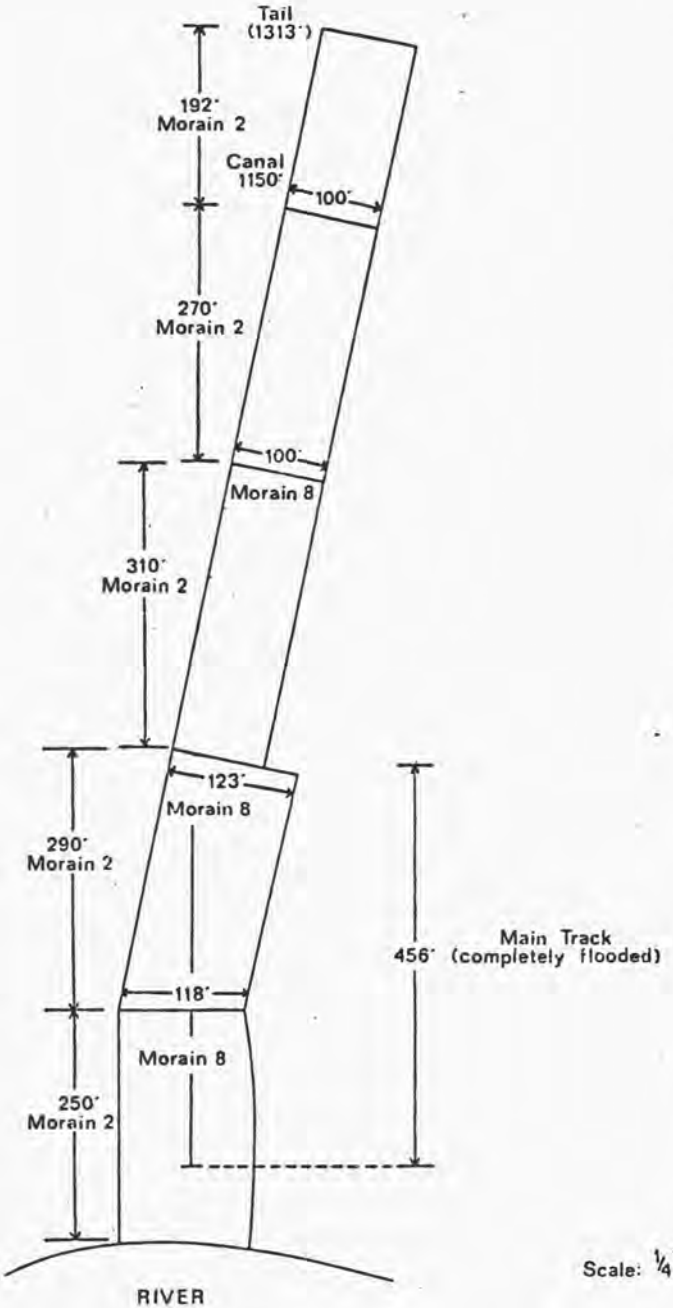


Figure 13


Tail
(1313')
192'
Morain 2
Canal
1150'
100'
270'
Morain 2
100'
Morain 8
310'
Morain 2
123'
Morain 8
290'
Morain 2
456'
Main Track
(completely flooded)
118'
Morain 8
250'
Morain 2
RIVER
Scale: ¼" = 50'

(Figure 14 - line segment A), the distance from the west bank of the Saigon River to the north-south trending drainage canal is 0.7cm or 1155'. The same distance as shown on the photo marked Morain #2 (which is Walker 262) and shown here as Figure 15, using measurements from the T-tail section* to establish scale is calculated to be 1150'.

.18cm:34.5' as 6cm:x = 1150', and 1cm = 192'

I conclude from this that Figure 15 can be used to give accurate measurements of distances so long as they are on a horizontal line equidistant from the camera lens.

The remaining measurements in this section were made from Figure 15 but use supporting data from the photos marked Morain #7, 8, 9 (which are Walker 767, 335 and 338, respectively). The tail section is .85cm west of the north-south trending drainage canal. This is equivalent to 163', or a total of 1313' from the west bank of the Saigon River (1150 + 163).

.18cm:34.5' as .85cm:x = 163'

The aft end of the troop compartment is 2.95cm from the north-south trending canal, which is equivalent to 565'. The aft end of troop compartment therefore is 565' + 1150' from west bank of river, or 1715'.

.18cm:34.5' as 2.95cm:x = 565'

---

* See page 27 for method for calculating dimensions of T-tail.

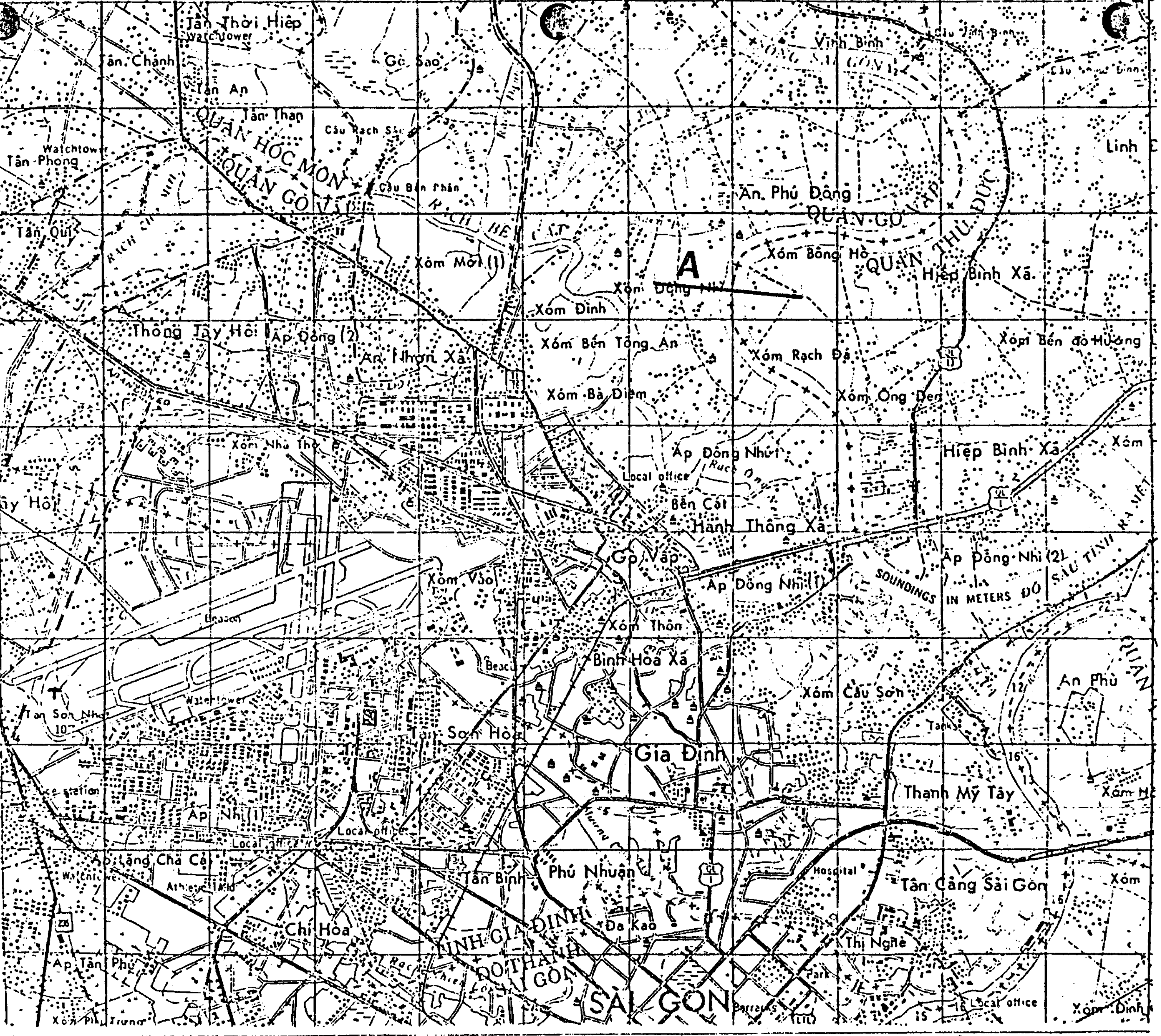


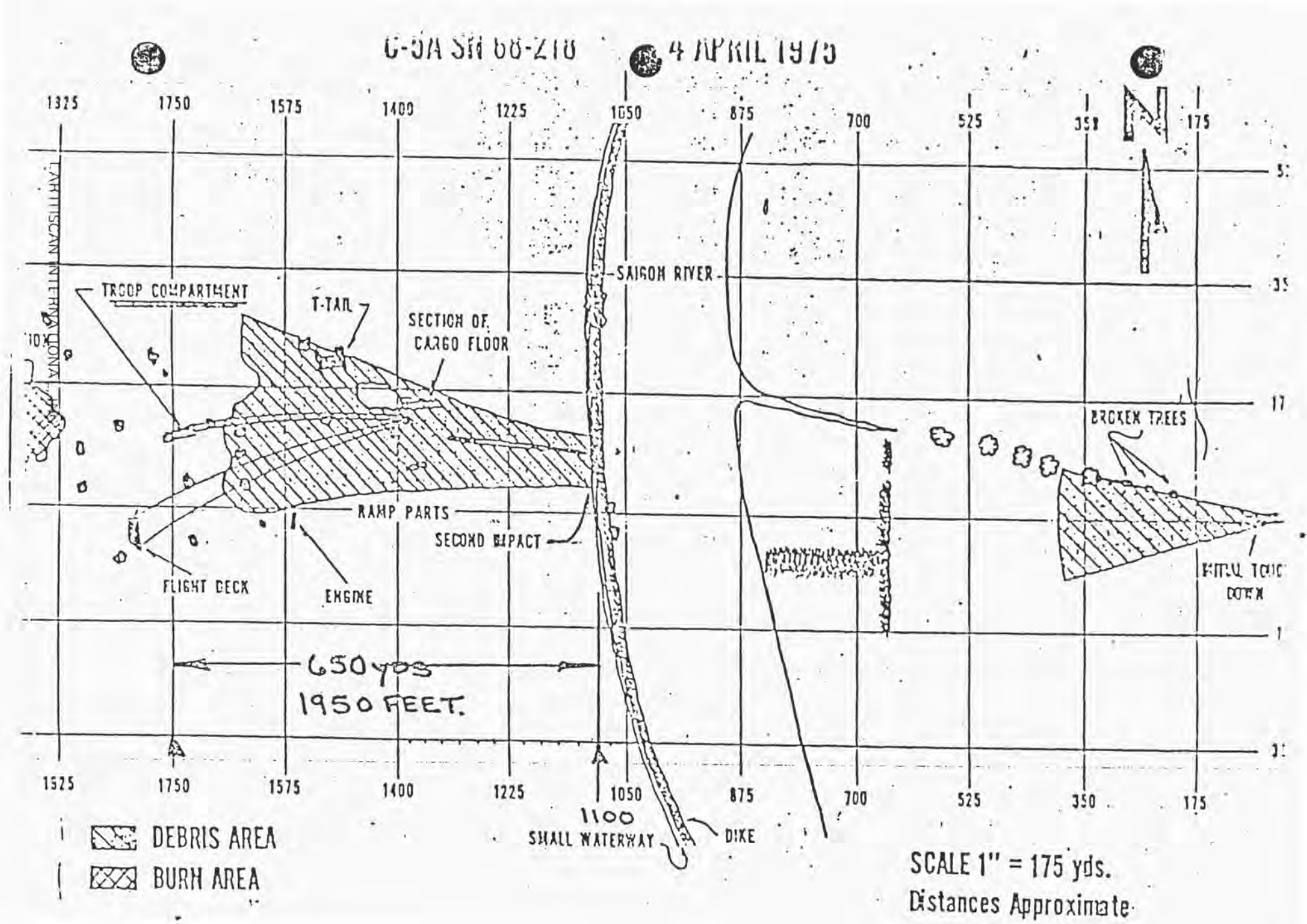
Figure 14: Portion of U.S. Army Topographic Command Map of Saigon, 1:50,000 scale

Figure 15: Morain #2 (Walker 262)

For comparison the debris map (doc C-5A SN 68-218 - 4 April 1975), and shown here as Figure 16, shows approximately 1315' and 2106', respectively. If there had been continuous contact with the ground after touch down on the west bank, the maximum length of the deceleration tracks for the troop compartment would be somewhere between 1715' and 1740'. The evidence in Figures 10, 11 and 12, as well as those figures presented in Part III indicate that there was not continuous contact and that, therefore, the deceleration tracks are shorter than 1715'.

From Figure 12 it is clear that the main flooded track begins approximately one-third of the way into the field adjacent to the west bank of the river and ends essentially at the field line extending north-south from the brushfield a few hundred feet south of the track. As measured on Figure 15 this would make the main track a <u>minimum</u> of 2.3cm long. Therefore, if 1cm = 192' (as has already been shown), then 2.3cm = 442'.

The end of this skid mark is identifiable on the photograph (Figure 12) as a short left (southward) trending hook of water. Evident, but less prominent, marks that are shallow and only partially flooded extend westward for a short distance (approx. another 0.7cm from the terminus of the main track). Therefore, <u>maximum</u> length of track would be 3cm, or 576'. The extension of 0.7cm for the less prominent track is estimated from relationships illustrated in Figures 10, 11 and 12, where it is clearly shown that the tracks disappear completely near the west field border of the next adjacent paddy. Water is evident in this track for only 538', the last 38', or so, being essentially dry.


C-5A SN 68-218
4 APRIL 1975
1325
1750
1575
1400
1225
1050
875
700
525
350
175
TROOP COMPARTMENT
T-TAIL
SECTION OF CARGO FLOOR
SAIGON RIVER
BROKEN TREES
RAMP PARTS
SECOND IMPACT
FLIGHT DECK
ENGINE
650 yds
1950 FEET.
1100
SMALL WATERWAY
DIKE
DEBRIS AREA
BURN AREA
SCALE 1" = 175 yds.
Distances Approximate

B. Depth of Tracks (Refer to Figures 10, 11)

Throughout most of their length the tracks are flooded by seepage water. The prominence of these gouges and the general absence of soil material or grass stems along them suggests that the water standing in them is at least several inches deep. At their western end the tracks become less deep and less water-logged until they finally disappear altogether. The remaining paragraphs in this part refer only to the depths of depressions from the general soil surface to the top of the water surface. From the point of first touch down on the west side of the river to their disappearance toward the west, it appears that the tracks gradually diminish in depth. The most pronounced soil depression occurs at the very beginning and is teardrop-shaped (center foreground Figure 11). Water does not fill the depression, which indicates along with several other similar occurrences to the left center of the photo that water did not enter the depressions via the drainage canals fringing the field. Since water seeks its own level, it is reasonable that all of the water in the scene represents the same topographic level. For water to have entered the teardrop depression, the only reasonable explanation is that there is a shallow subsurface (perched) water table in the area, and that water has seeped laterally into it (see Section II - Soil/Water Relations for more discussion on this point).

Because of the oblique nature of the photo and the absence of nearby features to aid in measurement, it is not possible to

calculate the depth of the teardrop depression. However, there is a person standing on the dike in the left foreground (point B on Figure 11) who can be used for scale. If this person is between 5' and 6' tall, it is reasonably clear that the depressions can be no more than 6" to 12" deep. As they progress westward (Figure 10), they become shallower...probably no more than 4" to 8" deep.

The features presumed to be strut and engine gouges on Figure 10 give the clearest notions of depth along this part of the C-5A's ground trajectory. By comparison to other measurable features in the scene, their depth does not exceed 6 to 8 inches.

III. Tracks Leading to Troop Compartment

A. Basis of Measurements (Refer to Figure 4, Defendant's Exhibit D1216)

The aft tip of fuselage to the aft tip of tail calculated to be about 35' by the following reasoning:

- Vertical height from base of wheels to top of fuselage = 30.58'
- Vertical height of wheels = 3.75'
- Vertical height from aft tip of fuselage to horizontal extension along top of fuselage = 3.75'
- Height from base of tires to top of tail = 65.08'
- Height from horizontal extension along top of tail to aft tip of tail = 3.75'
- Height from aft tip of tail to aft tip of fuselage therefore is (65.08 - 3.75) - (30.58 - 3.75), or 34.5 feet.

By an alternate method using proportional measurements taken directly from Figure 4, the dimension is....

1.65cm:30.6' as 1.95cm x = 36'

The length of the troop compartment (compartment E) derived directly from Figure 2 (Defendant's Exhibit D1217) using the proportional measurement method is calculated to be ....

1.55cm:155" as 7.9cm:x = 790" or 65'

B. Length of Tracks (calculated to be a maximum of 260')

Derived by the proportional measurement method on photograph marked Morain #1 (also Walker 340) and shown here as Figure 17. Length visible on the right side of the compartment (containing water) is 4.2 cm. Troop compartment is 1.05cm. Therefore....

1.05cm:65' as 4.2cm:x = 260'

Conditions on the surface show that the soil only bore a very heavy weight for a distance of two or three times the length of the troop compartment, or a total of 160-175 feet. Although there are minor tracks extending for another 100 feet to the east, they are so shallow that I conclude they were made either by an object only modestly in contact with the surface, or one skimming lightly over it.

C. Depth of Tracks

The main track leading to compartment E is shown in photos marked Morain #3 (Walker 762) and #4 (Walker 761), Figures 18 and 19, respectively. These show the extent of lateral seepage (drainage) referred to above. They also show that the marks begin almost exactly at the point where the vegetation looks taller in the middle foreground. There are faint indications of tracks eastward from this point but they appear to be displaced laterally to the right and may therefore be unrelated. Figure 19, in particular, refutes the idea that the tracks extend eastward onto the drier terrain.

Figure 17: Morain #1 (Walker 340)

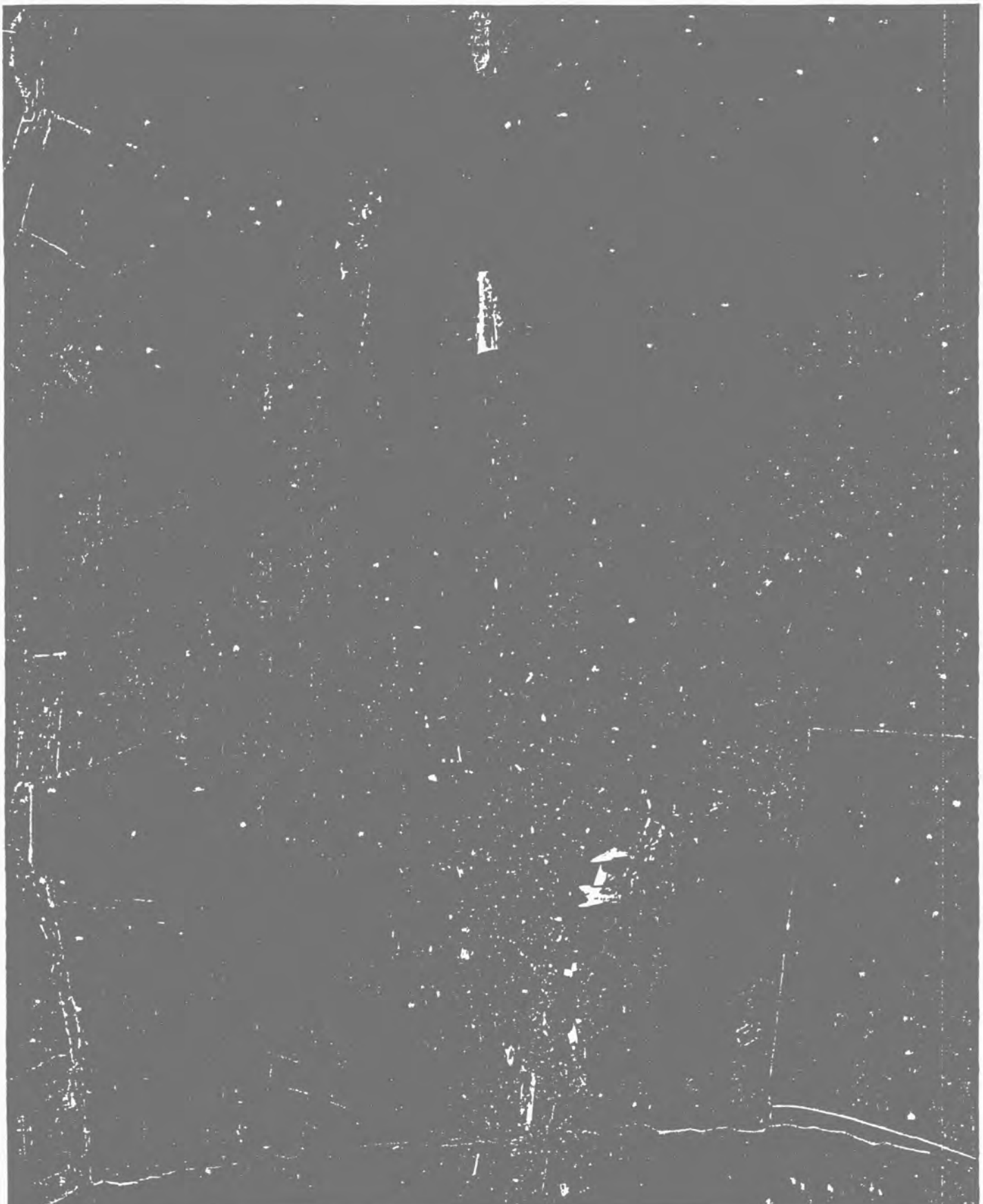


Figure 18: Morain #3 (Walker 762)

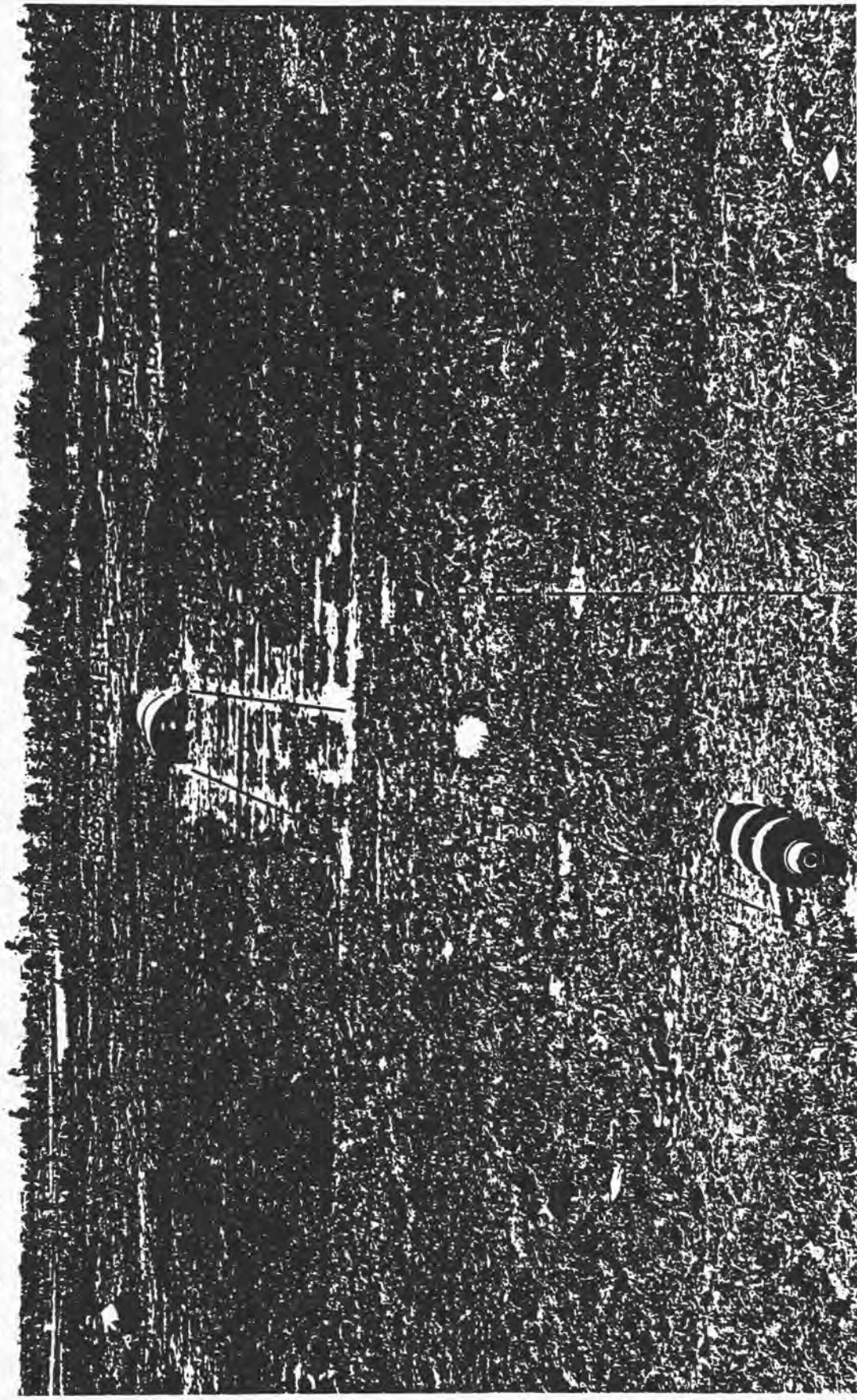
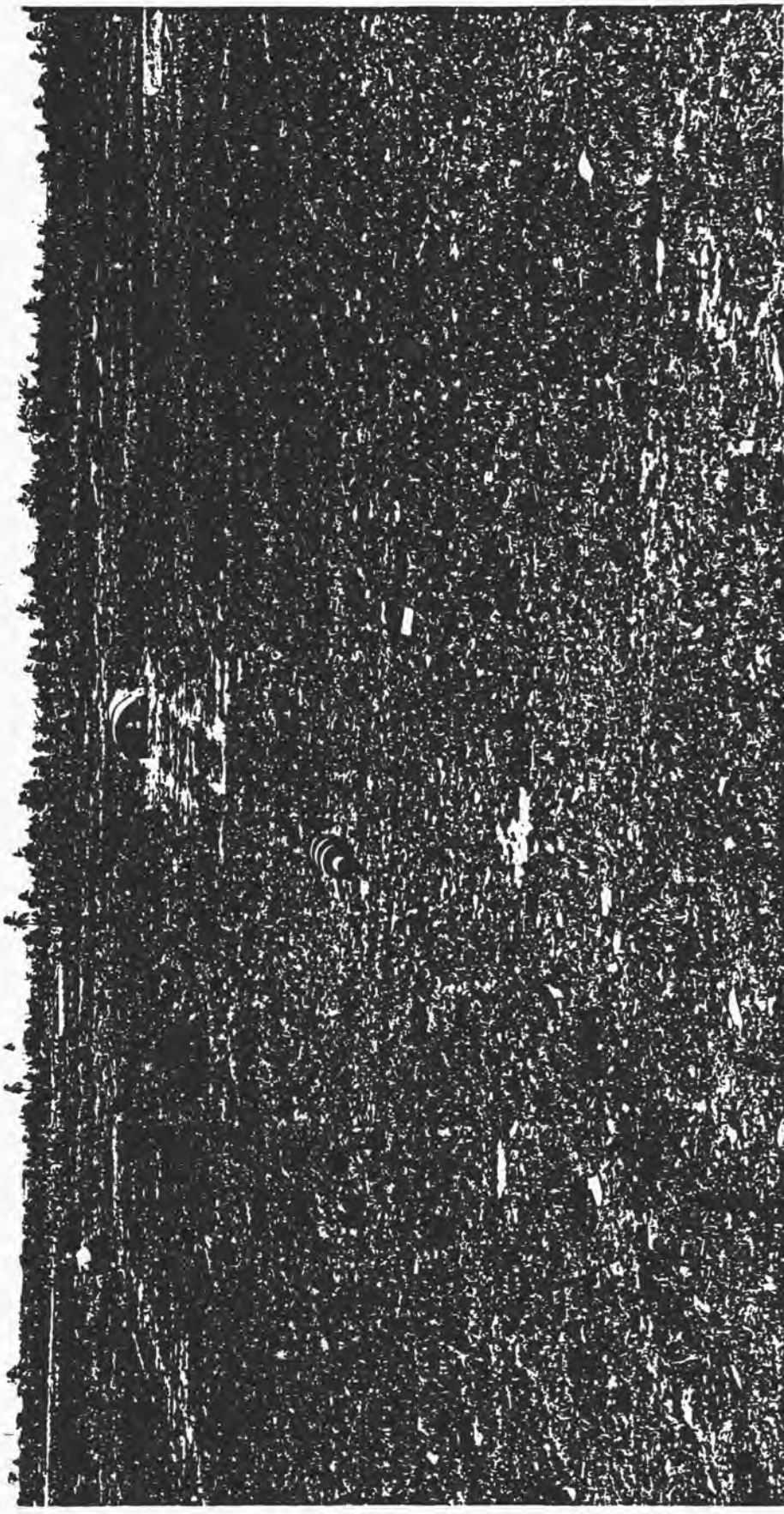


Figure 19: Morain #4 (Walker 761)



Evidence from the photo marked Morain #5 (Walker 187) and shown here as Figure 20, shows tracks flooded by water leading into the aft end of compartment E. The surface appears to be relatively flat, so it is not possible to calculate the depth of tracks. The track on the left side of the compartment cannot be too deep because grass stems and even some soil material along the track are above the seepage line. Ground between the tracks is saturated with water but more than half of the area is above the water (seepage) line. In the photo marked Morain #6 (Walker 83) and presented here as Figure 21, it appears that the skid pathway is depressed in relation to the ground level on either side of the pathway. From measurements taken from Figure 20, and compared to aircraft design specifications in Defendant's Exhibit D1217 (Figure 2), the depression is no more than 11" deep.

Method of Calculation: design specification for the width of the aft end of compartment E at the inflection point = 232". On Figure 20 this dimension measures 5.2cm. The point of inflection on the right side of the compartment is .4cm above soil/water level. Therefore:

5.2cm:232" as .4cm:x = 17.8"

The same point on the left side of the compartment is .15cm above soil/water level. Therefore:

5.2cm:232" as .15cm:x = 6.7"

By subtraction the left side of the compartment is about one foot lower than the right side.

Figure 20: Morain #5 (Walker 187)

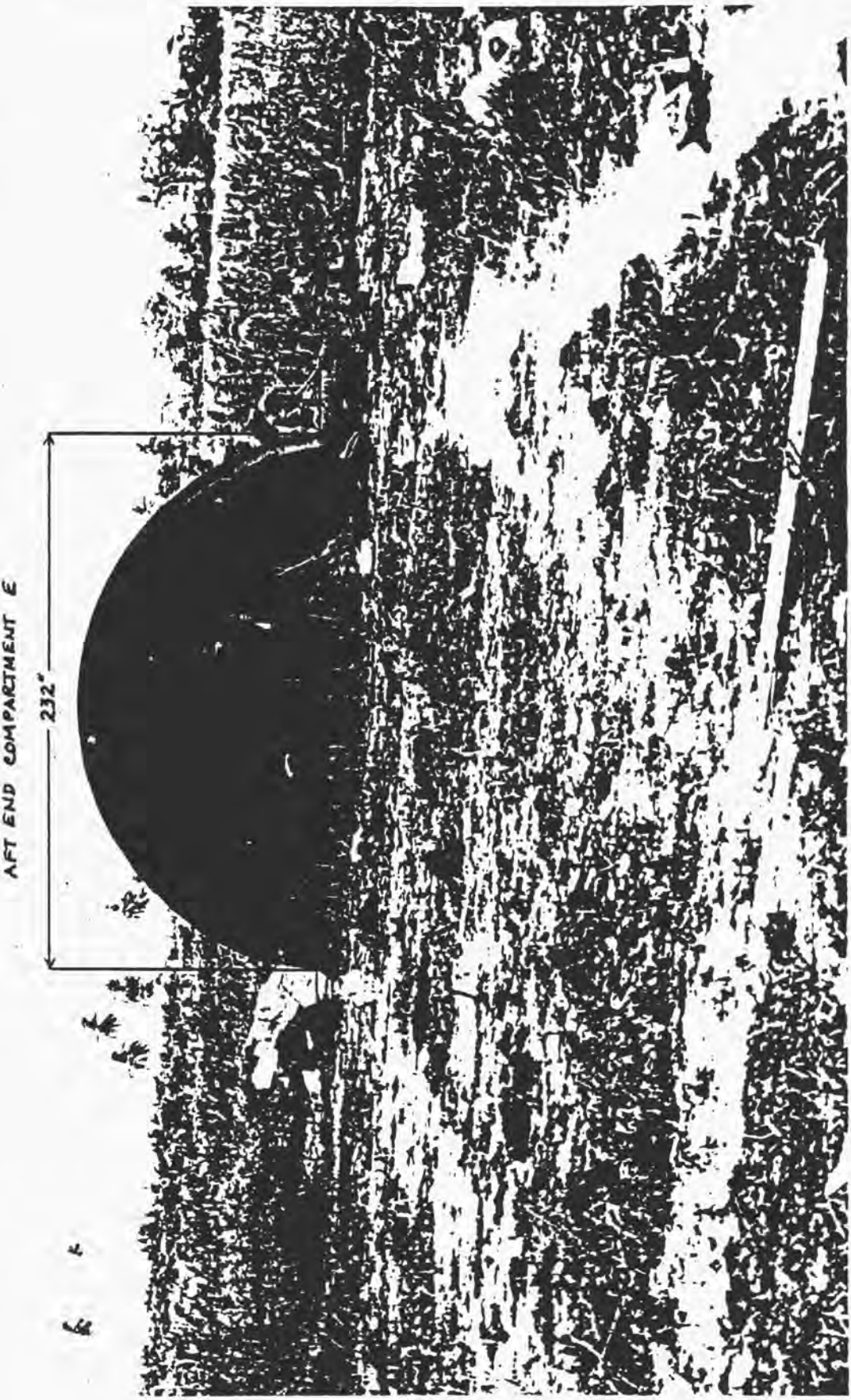


Figure 21: Morain #6 (Walker 83)

IV. Distance from Troop Compartment to Wing Debris Area

The distance from the troop compartment to the wing debris area is estimated to be around 350'. This is an average of three measurements and is a reasonable value based on pictorial evidence shown in Plaintiff's Exhibits 3E and 3210 (also labeled as Piper 12). In these photos the wing area and troop compartment are clearly visible, and the wing debris is shown to be located in the field immediately to the west of the location of the troop compartment. By previous calculations (Figure 13, page 20) these fields tend to have dimensions ranging from roughly 200' to 300'.

A second line of evidence confirms that 350' is a reasonable distance. By previous calculations, the troop compartment is located some 1715' west of the west bank of the Saigon River. An additional 350' would place the wing area some 2065' west of the river bank. Figure 14 (page 22) and Plaintiff's Exhibit 1000-57 show that there is a drainage canal located approximately 1990' west of the river bank (distance arrived at by proportional measurement of Figure 14), and that the wing has come to rest in the field just west of that north-south trending canal. Plaintiff's Exhibit 3210 also shows this canal. Document C-5A SN68-218 shows the wing area to be 2425' west of the river bank, which would place it some 440' west of the canal. This is not possible, as Exhibits 3210 and 3E clearly show.

V. Distance from Troop Compartment to Flight Deck

The distance from the foreward end of the troop compartment to the center of the flight deck area is calculated to be 150'.

This is based on four independent proportional measurements from Figures 17, 18 and 19 of this report and Plaintiff's Exhibit 3E. The value is reasonable when viewed on Plaintiff's Exhibit 1000-87 and compared to other distances already calculated. The troop compartment is approximately 400' from the T-tail (page 21-24), and 350' eastward of the wing debris. The aerial view in Exhibit 1000-87 clearly shows that the flight deck is closer to the troop compartment than either of the other units mentioned. Document C-5A SN 68-218 indicates a distance of approximately 325' which would place it at about the same distance as the wing debris. Neither Plaintiff's Exhibit 3210 nor 3216 suggest this. In both cases they show the flight deck to be much closer to the troop compartment than is the wing debris.

SECTION II: SOILS AND THE WATER ENVIRONMENT

I. Soil/Water Relations

Coordinates for the site of the C-5A incident are given as $10^{\circ}50'20"$ North latitude and $106^{\circ}42'40"$ East longitude. According to Defendant's Exhibit DD-2541-5 (Figure 22) this is an area of acid alluvial soils and acid sulphate soils developed on the flood plain of the Saigon River and its tributaries. Such soils are characterized by their fine particle textures dominated by silt and clay size particles (generally .002 to .005mm). The result is that they are generally heavy and intractable, particularly when wet.

Tropical occurrences of these soils, as is the case throughout the lower Mekong River Delta are generally poorly drained. Unlike rice growing regions in East Asia where irrigation is required for successful cropping, Southeast Asia is characterized by a need for drainage canals. Normal flood water and monsoon rainfall combine to make the region so wet that drainage, not irrigation, is the major problem. This is an important point with regard to the C-5A case because it means the subsoils are water-logged most of the year and that only for a few months during the season of lower rainfall would the soils be relatively dry at the surface. Climatic diagrams of the Saigon area indicate that this lower rainfall period occurs from January to March. By April the rainy season has generally begun.

Undifferentiated alluvial soils.
Sols alluviaux, non différenciés.

Saline alluvial soils.
Sols alluviaux salins.

Acid alluvial soils (acid sulphate soils).
Sols alluviaux acides (sols sulfatés acides).

Very acid alluvial soils (strongly acid sulphate soils).
Sols alluviaux très acides (sols sulfatés très acides).

Red and yellow podzolic soils on old alluvial sediments ; plane to undulating topography.
Sols podzoliques rouges et jaunes sur alluvions anciennes ; topographie plate à ondulée.

Gray podzolic soils on old alluvial sediments ; plane to undulating topography.
Sols podzoliques gris sur alluvions anciennes ; topographie plate à ondulée.

Low humic gley soils on old alluvial sediments ; plane topography.
"Low humic gley soils" sur alluvions anciennes ; topographie plate.

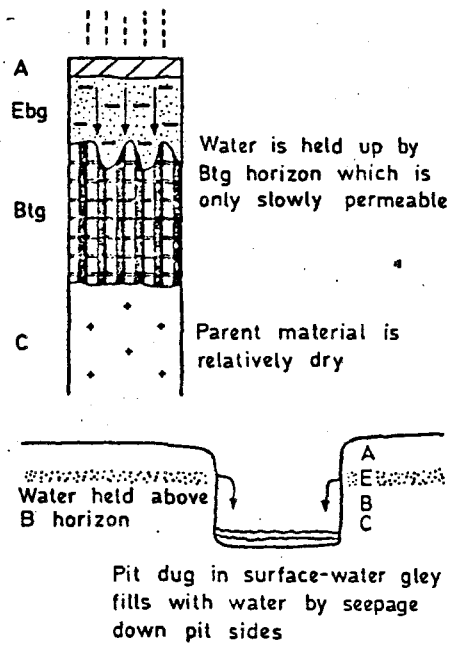
The persistence of water in the rice growing soils around Saigon leads to the development of subsoil layers that are generally very clay rich and which impede the downward percolation of natural rain water. Often the recognition that a natural area would be suitable for the development of paddy culture is based on the presence of these subsoil clay layers and the naturally boggy conditions that result. Current rice producing areas in the vicinity of Saigon have been under paddy since before the turn of the century, and, as a result, one can reasonably assume that the conditions favoring their initial development have been enhanced. An example of the formation of these clay layers is given in Figure 23 showing a typical paddy soil in the Bangkok Plain of Thailand (an area similar in most regards to that around Saigon). Figure 24 (from Defendant's Exhibit DD-2541-6) shows in diagrammatic form the mechanism by which water is impeded and seeps laterally into depressions that penetrate to any level below the level of perched water.

When one compares this general scenario with the appearance of tracks left by the C-5A on both the east and west sides of the Saigon River, the following points may be raised:

- On the east side of the river little or no standing water can be observed in the wheel marks. This would indicate that the soils are relatively dry and that the depth of penetration did not reach the level of perched water.
- The paddy fields in the vicinity of the initial touch down also appear to be relatively dry.

Figure 23

Figure 24: Extracted from Defendant's Exhibit DD-2541-6. Shows the relationship of soil layers and the mechanism by which water seeps laterally into depressions.



Only in rare instances can a pool of standing water be seen, and these are generally in the corners of fields. This follows with the idea that the incident occurred at the end of the dryest part of the year.

- On the west side of the river the perched water table must have been closer to the surface than on the east side. Evidence for this is given by the fact that the tracks are shallower than on the east side but are nevertheless filled with water.
- Toward the western terminus of the tracks shown in Figures 11, 12 and 13 there is less water visible at the surface, and eventually they become dry altogether. This suggests that the C-5A was gradually becoming airborne and placing less stress on what would otherwise have been a rather boggy and plastic situation.
- The tracks terminating at the aft end of the troop compartment are flooded but otherwise appear to be rather shallow. The compartment itself seems to be resting in a small swampy depression, westward of which there is a topographic rise.

II. Topography

Evidence for the existence of higher (slightly elevated) terrain forward of the troop compartment can be observed on

Plaintiff's Exhibit 10j; 3E; and Tarbell 4F, 4HH and 4II.

Exhibit 10j provides an indication of a gentle rise extending from the middle left side of the image along a line passing in front of the crew compartment, reaching a high point in the vicinity of the troop compartment and declining slightly toward the right center of the frame. It appears that the land slopes gently westward from this line toward the river tributary in the middle distance. Both the wing section and the crew compartment are westward of this slope inflection. Looking eastward from the troop compartment land appears to be sloping toward the Saigon River and the drainage canal fringing it. In this perspective the margin of discolored terrain seems to follow fairly closely the slope inflection.

Exhibit 3E shows an oblique aerial perspective the right side of which shows a distinct change in vegetation type along which it may be inferred there is a corresponding change in elevation and drainage conditions. A tracing has been made of the line distinguishing the two terrain conditions but, because of the pattern of discoloration in the vicinity of the troop compartment, its location can only be inferred.

Tarbell 4F shows a helicopter parked near the crew compartment. The photo was taken from a position somewhere between the troop and crew compartments in an area of discolored vegetation (facing approximately 225° or WSW). The helicopter is resting on the ground but is sloping such that the nose is higher than the aft section. This is evidence that the land surface is

sloping away from the viewer (a conclusion confirmed by interpretation of Plaintiff's Exhibit 10j and 3E). In the foreground of the photo, vegetation has clearly been burned as noted by the absence of leaves, dense mat of bare stems and absence of chlorophyll. Green matter appears to be present in the vicinity of the helicopter.

Tarbell 4HH shows the exact same helicopter looking eastward from a point a few yards away from the crew compartment back toward the Saigon River (facing approximately 135° ESE). In this perspective both the crew compartment and helicopter are resting on a land surface that is sloping away from the viewer. This slope is gentle but the vantage point of the photographer clearly shows that he is standing on higher ground which slopes away to the middle distance, then levels off on the paddy land immediately west of the Saigon River.

Tarbell 4II was taken by the photographer rotating his angle of view from the previous photo to approximately 70° east of north. The view shows the same helicopter and its location with regard to the troop compartment. Again it appears that the terrain slopes away from the photographer toward the troop compartment, beyond which the terrain levels off. The foreward end of the troop compartment is located at the base of the slope.

From this evidence I conclude that....

- There is a slight elevational rise (perhaps no more than a few feet) at the forward end of the troop compartment.
- The soils at the forward end are comprised of iron rich clays containing some pelletized iron concretions, all of which is highly resistent to deformation.

- Water infiltrating the slight rise percolated downward, then seeped laterally onto the terrain where the troop compartment finally came to rest. This accounts for the generally boggy conditions and presence of standing water around the troop compartment. This topographic situation is typical of natural bogs in low lying terrain and springs in more arid environments.

SECTION III: VEGETATION CONDITIONS

General vegetation in the vicinity of the C-5A flight path is rice paddies in various stages of growth, scattered shrubs and trees (mainly palms) and patches of natural grass (possibly regrowth). Along the immediate path rice paddies were dominant until the final few hundred feet of the trajectory of the troop compartment. At this point, which I have referred to as the area of tall grass in Figures 17 and 18, the vegetation appears to be natural and has much the same aspect of a small boggy depression. Westward of the troop compartment there appear to be small patches of palmetto-type regrowth (ie. dense stands of short palm scrub rather than stands of palm trees). These are interspersed with grass and low growing herbaceous vegetation. It can be stated without reservation that the vegetation around the troop compartment is not rice or rice stubble, but natural grass and palm. The best aerial photos I have seen in this connection are Plaintiff's Exhibit 3E and Tarbell 4E, 4G, 4I, 4CC, 4EE and 4HH.

The point of greatest interest in Plaintiff's Exhibit 3E is the bird's eye view of discolorations associated with the vegetation. An interpretation of these patterns has been made and prepared as an overlay to this exhibit. From visual inspection of the above listed photographs, I am confident that some areas show the effects of fire, others the effects of either a flash fire or of physiological damage caused by a fuel spill and still others appear to be "normal." The main evidence for burning consists of blackened surface, absence of chlorophyll and absence of leaves to reveal stems and twigs. Evidence for the flash fire or physiologic damage consists of an orange-brown surface, damage to growing tips (leading to a brown or orange surface) but retention of leafy matter. The damage is such as to remove chlorophyll but not to consume the vegetable matter. The blackened areas, on the other hand, reveal a consumption of vegetable matter as well as the loss of chlorophyll.

As a final conclusion, I have noted that several pictures show the C-5A wing on fire westward of the troop compartment. Plaintiff's Exhibit 3E shows not only the area of the troop compartment but also those of the crew compartment and wing. Areas known to have burned in the vicinity of the wing have essentially the same appearance as those around the other main units. Consequently, it seems reasonable to conclude that all of these areas experienced burning and fuel spill, even if momentary, during the final moments of the incident. It is reasonable to further conclude that burning around the troop compartment would have been quickly self-extinguishing because of all the water.

A. Materials Used

The revised wreckage diagram presented in this summary is based on calculations from the photographs illustrated in the report; additional photographs cited, but not illustrated; multiple viewings of a short color aerial film strip; and, pertinent maps prepared by the U.S. Army Topographic Command. Photographs from the Walker series, the Piper series and the Tarbell series are included in the analysis. It is my conclusion that the diagram represents a reasonable reconstruction based on these measurements. The points summarized in Part C of this section are based on personal field experience and research into the nature and properties of tropical soils; experience in rice-growing areas; library research regarding soil and vegetation conditions in the Mekong Delta, which includes the area around Saigon; and, interpretation of the photographs cited in the report.

B. Key Measurements

1. East Side of Saigon River

a. the longest and deepest ruts are about 110' long and 27" deep, and terminate approximately 2240' east of the river bank;

b. there are indications of multiple contacts with the ground, but very few of the marks are long enough, or deep enough, to have initiated lateral seepage from the perched water table that exists in this soil environment.

2. West Side of Saigon River

a. the first, most prominent and continuous track is approximately 500' long and of unknown depth due to standing water. The depth from water level to general ground level is estimated to be 6-12". There is no evidence that these tracks breach the main drainage canal at their western end;

b. evidence for less prominent tracks extending across the main drainage canal to the west is weak. The marks that are most observable trend in a direction parallel to this canal which leads to the conclusion that they are related to the drainage system in the field and not to the C-5A;

c. the T-tail, aft end of the troop compartment, flight deck and wing debris area are located 1310', 1715', 1840' and 2065' respectively from the river bank. These measurements are confirmed by the plotted locations of major north-south trending canals and walkways on the 1:50,000 scale Army Topographic Command Map;

d. the length of tracks aft of the troop compartment is calculated to be 260'. Only the last 165 feet supported a heavy weight as evidenced by the swampy condition of the site. In their eastward end (100 feet) the tracks are shallower. From this I conclude they were made by an object that was only skimming the surface.

e. the length of tracks leading into the remains of the flight deck is calculated to be 460 feet. The absence of water around this unit indicates that it came to rest on a relatively well-drained slope (see also point C.1c, below);

f. the distance over which no tracks can be reasonably discerned is 950 feet. It is in this area that most of the C-5A debris is found.

C. Other Soil/Water and Vegetational Conclusions

1. Evidence from published maps, combined with the geographic coordinates of the crash site, confirm the presence of so-called "acid alluvial" and "acid sulphate" soil. By nature, these soils are poorly drained and their general mode of genesis leads to the existence of a perched water table which fluctuates vertically throughout the year. In April, at the beginning of the wettest part of the annual cycle one finds this water table anywhere from a few inches to a foot or more below the soil surface.

a. On the east side of the river there is no indication of standing water in the multiple contacks made by the C-5A. Thus, it is reasonable to conclude that the aircraft was only "skimming" the surface and that, except for one or two instances, never impacted hard enough to penetrate the surface soil layer and initiate lateral seepage;

b. On the west side of the river, the initial impact was sufficiently deep that the resulting tracks are flooded. These tracks terminate rather abruptly and do not breach the drainage canal toward which they are headed. It is reasonable to conclude, therefore, that the aircraft was no longer in complete contact with the ground, although it might have been skimming the surface. There is evidence of a "skipping" action beyond the terminus of the main track, and almost all of the resulting depressions are filled with water. The absence of water-filled tracks for the ensuing 950 feet cause me to conclude that the aircraft was not rolling across the surface. The next indisputable evidence of tracks occurs 260' eastward of the troop compartment and these are flooded. I conclude that the last major impact of the troop compartment occurred 165 feet away from its final resting place. The intensity of its impact was sufficient to cause seepage to take place around it. The occurrence of water in this locale is enhanced by the presence of an elevational abuttment at the foreward end of the troop compartment (see below).

c. Evidence from several of the photographs clearly indicates a hummock-like feature having a slightly

higher elevation than the surrounding terrain (two or more feet). Drainage radiates away from the troop compartment to the northwest, west and southwest (which explains why the flight deck is located on relatively dry ground); while, to the east, the terrain is essentially flat. Water infiltrating this hummock percolates downward to the subsurface water table. The water level in the hummock fluctuates seasonally, just as it does in the surrounding terrain, but over a greater vertical range. The result is that over a long period of time, iron has been mobilized during the chemically reducing, wettest part of the annual cycle, and oxidized during the better drained, driest part of the year. This leads to the formation of resistent "iron stone" or pelletized, iron-rich, concretions surrounded by non-expanding clay species. In my experience this would be a wall-like abuttment against which the troop compartment came to rest.

1. Evidence has been presented to show that patterns of vegetation discoloration around the troop compartment and flight deck are similar to those in the vicinity of the wing debris. It is logical and reasonable to conclude that some burning took place around all of these units, but that it was obviously most intense

and prolonged around the wing. The fire around the troop compartment was most probably brief and self-extinguishing due to the swampy environment.

D. Revised Wreckage Diagram

A revised wreckage diagram has been prepared from all of the above evidence (Figure 25). For comparative purposes it has been designed along lines similar to Figure 16 (page 25), which was prepared by the U.S. Government. Figure 25 lists all of the key measurements and makes note of major terrain observations along the ground track of the aircraft. The most noticeable difference between the two figures is that photographic measurements place the troop compartment, wing section and crew compartment much closer to the west bank than originally shown. In addition, the debris area has been subdivided into "heavy" and "light" categories, and the length of tracks has been scaled to reflect their measured length from the photographs.

WRECKAGE DIAGRAM

C-5A SN 68-218 4 APRIL 1975