

AD-A035 700

MEDICAL COLL OF VIRGINIA RICHMOND DEPT OF SURGERY
PROTOCOL FOR MANAGEMENT OF PENETRATING CAROTID ARTERY INJURY. (U)
FEB 77 L J GREENFIELD, W G LIEKWEG

F/G 6/5

N00014-76-C-0422

UNCLASSIFIED

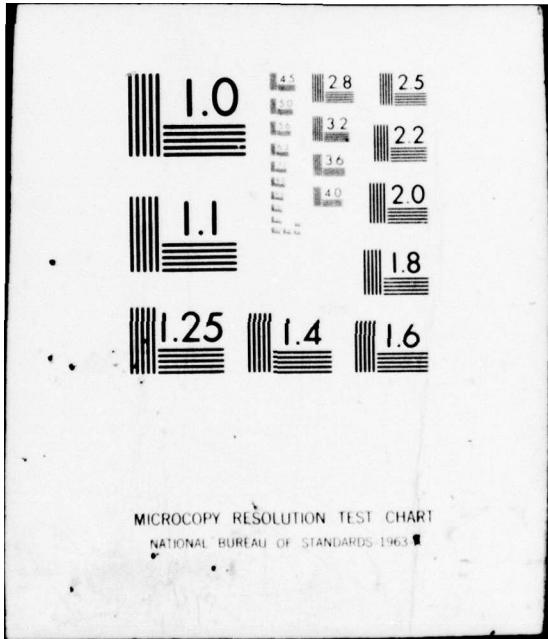
GR57-76-02

NL

1 of 1
AD
A035700

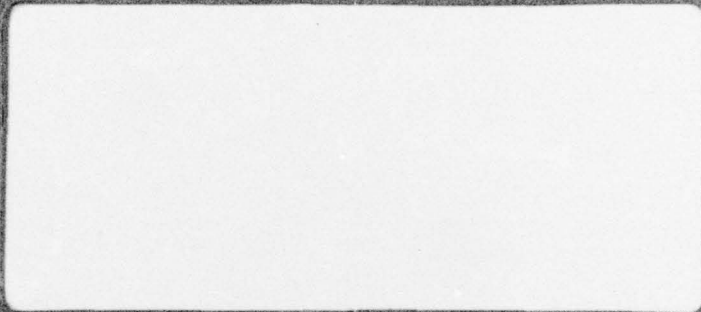


END
DATE
FILMED
3-77



MICROCOPY RESOLUTION TEST CHART
NATIONAL BUREAU OF STANDARDS-1963-A

ADA035700



200 1473

~~1~~
6
B.S.

ADG

OFFICE OF NAVAL RESEARCH

Contract N00014-76-C-0422 ✓

Project NR 105-821

TECHNICAL REPORT NO. II ✓

PROTOCOL FOR MANAGEMENT OF
PENETRATING CAROTID ARTERY INJURY

Lazar J. Greenfield, M.D. and William G. Liekweg, Jr., M.D.

Prepared for Publication

in

Surgery

Medical College of Virginia ✓
Department of Surgery
Richmond, Virginia

February 8, 1977

Reproduction in whole or in part is permitted for
any purpose of the United States Government

Distribution of this report is unlimited

ACCESSION for		
NTIS	White Section	<input checked="" type="checkbox"/>
DOC	Buff Section	<input type="checkbox"/>
UNANNOUNCED		<input type="checkbox"/>
JUSTIFICATION		
BY		
DISTRIBUTION/AVAILABILITY CODES		
Dist.	AVAIL.	AND/OR SPECIAL
A		

Following the conflict in Southeast Asia a number of civilian and military reports were published in an effort to establish guidelines for management of carotid arterial injuries.^{2,3,4,8,13,16,17} These articles focused on the pre-operative neurological state of the patient and attempted to establish guidelines for repair based on the presence or absence of neurologic deficits. The conclusions were vague, however, leaving the surgeon with confusing criteria on which to base his treatment. This report reviews a 10 year experience at the Medical College of Virginia of isolated common and internal carotid injuries in eighteen patients. Comparison with published reports of 215 cases has allowed us to develop specific clinical guidelines for management of carotid arterial injuries.

PATIENTS AND METHODS

From January 1966 to December 1975, 96 patients with penetrating injuries to the neck underwent surgical exploration at the Medical College of Virginia, Virginia Commonwealth University. Eighteen of them had isolated injuries to the common or internal carotid arteries. Those patients with associated injuries that might have influenced their clinical course were eliminated from review, as were those with isolated external carotid or venous injuries.

The delay from time of injury to surgical intervention could be determined accurately in 12 cases and the mean interval was three hours and fifteen minutes. Seventeen of the eighteen patients were male and the age range was from 19 to 70 years with a mean of 31 years. There was an equal distribution of right and left sided injuries. Sixteen wounds were inflicted by low caliber gunshot wounds and there were two penetrating injuries from knife wounds.

A thorough neurological evaluation was performed preoperatively in each patient. The comatose state was defined as unresponsiveness to verbal stimuli. Systemic hypotension defined as systolic blood pressure less than 90 mmHg was

present on admission in seven patients and was corrected preoperatively in each case. There was no correlation between hypotension, neurological deficit and the end result (Tables 1 and 2). Four patients had significant neurological deficit and associated hypotension. Correction of hypotension did not improve the neurological deficit.

There were four deaths (mortality rate 22%), all occurring in the immediate post-operative period (i.e., within 72 hours). All deaths were related to cerebral injury. Post-mortem examination was obtained in one of the patients and showed macroscopic evidence of hemorrhagic infarction. The second patient who died had a clinical course compatible with uncal herniation. The third death followed 12 hours of generalized seizure activity. Subsequent EEG's showed no evidence of cerebral activity. In the fourth patient, disseminated intravascular coagulation complicated the clinical picture, although the neurological status of the patient was unchanged following surgery.

ABSENCE OF PRE-OPERATIVE NEUROLOGIC DEFICIT:

On examining the preoperative neurological status of the patients, ten of them showed no deficit (Table 1). All had primary vascular repair of their arterial injury and nine of them are now alive and well without neurological sequelae. One patient (Case #5) had a transient monoparesis which cleared completely. Another patient (Case #4) developed a left hemiparesis post-operatively. This patient had a benign course until the fifth post-operative day when he developed a left hemiplegia and rapidly became comatose. He underwent emergency cranial decompression by right frontal lobectomy. The pathology report showed hemorrhagic infarction of the right frontal lobe. At two years follow-up, this patient is ambulatory with a brace and is under treatment for a seizure disorder.

PRE-OPERATIVE NEUROLOGICAL DEFICIT:

In eight cases (Table 2), the patient had a significant neurological deficit

after hypotension had been corrected. The deficits consisted of monoplegia, hemiplegia or quadriplegia, with or without an associated comatose state. All deaths occurred in this group.

Four patients had operative repair of their vascular injury. Patients #12 and #14 had localized deficits manifested as monoplegia and hemiplegia, respectively. Both of them cleared their deficit following revascularization. Patients #13 and #17 were comatose and patient #13 was also quadiplegic. Both of them died within 72 hours following revascularization.

An additional four patients were managed by ligation of the carotid system. All of these patients were comatose at the time of surgery and three of them also had localizing neurological signs. There were two deaths in the group, one of which occurred nine months following surgery. One patient has shown improvement, and the fourth is fully recovered.

COLLECTED REVIEW:

A number of reports related to carotid arterial injury have been published since 1963.^{1,2,3,4,6,8,13,16,17} The preoperative and postoperative neurological status and operative management were determined in each series. Those cases with associated head injury or any significant injury which could influence the outcome of the patient were eliminated. A total of two hundred fifteen cases was obtained through this search. Combined with the cases from this study a total of two hundred thirty-three cases were included in the following analysis.

The cases were divided into four groups, cross matching the presence or absence of preoperative neurological deficit with the method of management; i.e., restoration of vascular continuity or ligation of the damaged carotid vessel.

One hundred and seventy patients had a normal neurological examination prior to surgery. One hundred and sixty-one of them had direct repair of their injury. There were no deaths in this group but two patients developed hemiplegia as a residual neurological deficit¹⁶ (Table 3).

Nine patients who had normal neurological examinations had the injured carotid vessel ligated. This was usually due to technical difficulty associated with attempts at repair. There was one death in this group and two patients developed permanent neurological deficits^{4,13} (Table 3).

Sixty-three patients had abnormal neurological examinations preoperatively. The deficits were monoplegia, hemiplegia, quadriplegia, aphasia, or coma. The comatose state was often found in conjunction with lateralizing signs. Forty-nine patients had restoration of vascular continuity. There were twelve deaths in this group and all of them were related to cerebral injury. The overall results are shown in Table 4.

The remaining fourteen patients were managed by ligation of the carotid artery (Table 4). Forty-three percent were no worse following surgery but fifty-seven percent deteriorated or died following carotid artery ligation.

If those patients who were comatose at the time of surgery are excluded, then the results following vascular repair as opposed to ligation are exceptionally good (Table 5). There were thirty-four patients who had significant preoperative neurological deficits (monoplegia, hemiplegia, aphasia) but without coma. Twenty-nine of them (85%) were improved or showed no deterioration from their preoperative state. Twenty-two of them (65%) had a complete recovery with subsequent normal neurological examination. There was progression of the neurological deficit in two patients and three of them died.⁴ It should be noted that in these three deaths no mention of the presence or absence of coma was made in the preoperative assessment. It is assumed that no coma was present; therefore, any bias would be directed against the final conclusions of this study. The fate of the comatose individual is bleak. Seventy percent died regardless of the mode of therapy (Table 6).

DISCUSSION:

The current surgical literature including the most recent reports from Southeast Asia contains a number of pleas for more conservative approaches to management of patients with penetrating carotid injuries in association with severe neurological deficits.^{2,4} Bradley, in an excellent review² which included a pathophysiological study of injury based largely on the primate experience of Denny-Brown and Meyer,^{5,9-12} proposed that any injury with neurological lesions more severe than mild paresis, should be managed by ligation. Cohen⁴ advocated the same approach following his military experience, except that he favored ligation in every patient with any degree of neurological impairment. Thal¹⁶ recommended vascular repair only if flow was present in the carotid system at the time of exploration. The recent experience at the Medical College of Virginia and the review of published data do not support these conservative recommendations.

It is logical to assume that the presence of a preoperative neurological deficit is an indication of inadequate cerebral collateral blood flow. However, this assumes that those factors which influence collateral blood flow are corrected before determining the extent of the deficit. These factors include systemic hypotension, reduction in arterial oxygen tension and accumulation of acid metabolites.⁹ On this basis, patients with penetrating carotid injuries can be divided into two groups: A. Those patients with adequate collateral flow as evidenced by the absence of neurologic signs. B. Those patients with inadequate collateral circulation as demonstrated by the presence of a neurological deficit. The exception to this would be those patients with injury to the carotid system without total disruption of flow. In this group, the adequacy of collateral flow cannot be assessed.

Ninety percent of the cerebral blood flow is contributed by the carotid vessels; ten percent by the vertebral system. If flow is interrupted in one of

these vessels, there is a compensatory increase in flow in the other vessels supplying the circle of Willis.⁷ However, only twenty percent of normal individuals have what is considered to be the standard anatomical configuration.¹⁴ Other collateral pathways exist, but their importance in the acute disruption of flow is unknown.²

It seems apparent from the clinical results that the neurological state which correlates best with an irreversible injury is coma. Motor weakness or paralysis may be reversible with a very acceptable risk factor. Improvement therefore may be dependent on re-establishing blood flow to the ischemic area. Many believe that there is an area of relative ischemia surrounding an infarcted region which, although physiologically inert, can improve with an increase in or restoration of cerebral blood flow. There is experimental evidence that permanent interruption by ligation of a major vessel decreases the reserve capacity of the brain to tolerate any vascular stress.¹⁵ Consequently, experimental evidence and the clinical data presented argue against carotid ligation.

The management of the comatose patient remains difficult. Results are similar regardless of which surgical approach is taken. It seems reasonable that if prograde flow is present in the injured carotid at the time of exploration, then revascularization should be attempted. If no flow is present and the patient is comatose, restoration of flow to this avascular area could be deleterious.

This study has focused on isolated carotid injuries in an effort to delete any bias that associated injuries might have on the end result. Our experience with patients having carotid injury in association with multiple system involvement is small, but the extension of this treatment plan to the multiple injured patient appears logical. The carotid injury must take high priority in the immediate management of these individuals.

CONCLUSIONS:

I. All patients who do not have a neurological deficit should have restoration of vascular continuity.

This method of treatment is associated with uniformly good results, does not subject the patient to permanent interruption of flow and the possible sequelae, and does not place the patient at greater risk due to the progression of atherosclerotic disease.

II. All patients who have a significant neurological deficit (monoplegia, hemiplegia, quadriplegia, or aphasia) short of coma, should also have primary vascular repair. This method of treatment has been shown to be significantly superior to carotid artery ligation.

III. In comatose patients, repair or reconstitution should be undertaken if prograde flow is present. Neither repair nor ligation appear to change the basic poor prognosis significantly.

IV. At the present time, ligation of the carotid artery is indicated only:

- (a) for the comatose patient with no prograde flow.
- (b) for technical reasons when repair is impossible.

REFERENCES

1. Beall, Arthur C., Jr., Shirkey, Albert, L., and DeBakey, Michael, E. Penetrating wounds of the carotid arteries. *J. Trauma* 3:276-287, 1963.
2. Bradley, E.L. Management of penetrating carotid injuries: An alternative approach. *J. Trauma* 13:248-255, 1973.
3. Buchman, R.J., Thomas, P.A. and Park, B. Carotid artery injuries. *Angiology* 23:97-102, 1972.
4. Cohen, A., Brief, D. and Mathewson, C. Carotid artery injuries. *Am. J. Surgery* 120:210-214, 1970.
5. Denny-Brown, D. and Meyer, J.S. The cerebral collateral circulation. II *Neurology* 7:567-578, 1957.
6. Flax, R.L., Fletcher, H.S. and Joseph, W.L. Penetrating injuries of the neck. *The American Surgeon* 148:150, 1973.
7. Hardesty, W.H., Whitacre, W.B., Toole, J.F, Randall, P. and Royster, H.P. Studies on vertebral artery blood flow in man. *Surgery, Gynecology and Obstetrics* 116:662-664, 1963.
8. Hewitt, R.L., Smith, A.D., Becker, M.L., Lindsey, E.S., Dowling, J.B. and Drapamds, T. Penetrating vascular injuries of the thoracic outlet. *Surgery* 76:715-722, 1974.
9. Meyer, J.S. and Denny-Brown, D. The cerebral collateral circulation I. *Neurology* 7:447-458, 1957.
10. Meyer, J.S. Importance of ischemic damage to small vessels in experimental cerebral infarction. *J. Neuropathology* 17:571-584, 1958.
11. Meyer, J.S. Circulatory changes following occlusion of the middle cerebral artery and their relation to function. *J. Neurosurgery* 653-673, 1958.
12. Meyer, J.S., Terauks, T., Marx, P., Hashi, K. and Sakamoto, K. Experimental cerebral infarction. *Brain* 95:833-852, 1972.

13. Menson, D.O., Saletta, J.D. and Freeark, R.J. Carotid vertebral trauma. *J. Trauma* 9:987-997, 1969.
14. Roberts, B., Hardesty, W.H., Helling, H.E., Reivich, M., and Toole, J.F. Studies on extracranial cerebral blood flow. *Surgery* 56:826-833, 1964.
15. Sengupta, D., Harper, M. and Jennett, B. Effect of carotid ligation on cerebral blood flow in baboons. *J. Neurology, Neurosurgery, and Psychiatry* 36:736-741, 1973.
16. Thal, E.R., Snyder, W.H., Hays, R.J. and Perry, M.O. Management of carotid artery injuries. *Surgery* 76:955-962, 1974.
17. Williams, J.W. and Sherman, R.T. Penetrating wounds of the neck. *Surgical Management. J. Trauma* 13:435-442, 1973.

Table 1. Results of Surgical Repair of Carotid Artery Injuries in Patients without Preoperative Neurological Deficits at the Medical College of Virginia 1966-1976

Sex	Age	Mode of Injury	Injury ^a	Assoc Injury	Operation	Preoperative Neuro Status	Postoperative Neuro Status	Shunt	Comment
M	19	GSM	C.C.	None	Resection & Primary Anastomosis	Normal	No Deficit	Yes	Normotensive: ischemic time 4 minutes
M	70	GSM	C.C.	None	Lateral Arteriorrhaphy	Normal	No deficit	Yes	Normotensive
F	58	GSM	C.C.	None	Resection & Primary Anastomosis	Normal	No deficit	No	Normotensive: ischemic time 12 minutes
M	24	GSM	C.C.	Left Linear Skull	Resection & Primary Anastomosis	Normal	L. Hemiparesis	No	Hypotension corrected pre-operatively; developed hemiparesis on 5th post-op day. Emergency right frontal lobectomy
M	20	GSM	C.C.	None	Saphenous Vein Graft CC to IC	Normal	Transient Paresis, right arm	Yes	Normotensive: cleared completely
M	50	Stab	C.C.	None	Resection & Primary Anastomosis	Normal	No deficit	No	Hypotension corrected pre-operatively
M	32	GSM	C.C.	None	Ext. Jugular Vein Patch C.C.	Normal	No deficit	Yes	Normotensive
M	35	GSM	C.C.	A-V Fistula	Resection & Primary Anastomosis	Normal	No deficit	Yes	Normotensive
M	24	GSM	I.C.	None	Lateral Arteriorrhaphy	Normal	No deficit	No	Normotensive
M	20	Stab	C.C.	None	Dacron Patch Graft to C.C.	Normal	No deficit	No	Hypotension corrected pre-operatively. Respiratory arrest necessitating tracheostomy in E.R.

^a CC = Common carotid artery
IC = Internal carotid artery

Table 2. Results of Surgical Repair of Carotid Artery Injuries in Patients with Preoperative Neurological Deficits at the Medical College of Virginia 1966-1976

Sex	Age	Mode of Injury	Injury ^a	Assoc Injury	Operation	Preoperative Neuro Status	Postoperative Neuro Status	Shunt	Comment
M	20	GSM	C.C.	Fracture Mandible	Ligation Common Carotid Bifurcation	Unresponsive sluggish pupils	No deficit	Yes	Normotension corrected preoperatively. Ischemic time 10 minutes
M	21	GSM	C.C.	None	Resection & Primary Anastomosis	R. monoplegia (arm)	Deficit Cleared	Yes	Normotensive
M	19	GSM	C.C.	Transsected Spinal Cord @ C-5 level	Resection & Primary Anastomosis	Unresponsive Flaccid extremities	Death at 72 hrs.	Yes	Hypotension corrected in O.R. prior to surgery
M	26	GSM	C.C.	None	Sphenous Vein Graft C.C. to I.C.	Left hemiparesis	Deficit Cleared	Yes	Normotensive
M	46	GSM	C.C.	None	Ligation Common Carotid	Comatose with right hemiparesis	Death	No	Normotensive - Generalized seizure activity postoperatively. Death at 72 hours Autopsy confirmed intracerebral hemorrhage
M	21	GSM	C.C. I.C.	None	Resection & Primary C.C. to I.C.	Comatose	Decerebration & Death		
M	20	GSM	C.C.	Int. jug vein, soft tissue	Ligation Right Common Carotid	Comatose	Comatose		Hypotension corrected preoperatively: DIC
M	30	GSM	C.C.	C.C.	Ligation Common Carotid	Comatose left hemiplegia	Unchanged		Hypotension corrected preoperatively. Death 2 ^{1/2} months postoperatively. Remained comatose

^aCC - Common carotid artery
IC - Internal carotid artery

Table 3. Collected Results of the Treatment of Carotid Artery Injuries in the Absence of Preoperative Neurological Deficit

<u>Treatment</u>	<u>Revascularized</u>	<u>Ligated</u>
Previously Published Cases	151	9
Current Report	<u>10</u>	<u>0</u>
Total Cases	161	9
 <u>Results</u>		
No Deficit	153	6
Transient Deficit	6	0
Permanent Deficit	2	2
Death	0	1
Favorable Outcome	99%*	67%*

*p < 0.05

Table 4. Collected Results of Treatment of Carotid Artery Injuries
in the Presence of Preoperative Neurological Deficit

<u>Treatment</u>	<u>Revascularized</u>	<u>Ligated</u>
Previously Published Cases	45	10
Current Report	<u>4</u>	<u>4</u>
Total Cases	49	14
 <u>Results</u>		
Improved	23	5
Unchanged	10	1
Progressive Deficit	4	1
Death	12	7
Favorable Outcome	67%*	43%*

*Not Significant

Table 5. Collected Results of Treatment of Carotid Artery Injuries in the Presence of Preoperative Neurological Deficit Exclusive of Coma

Total Cases Reported - 40

	<u>Revascularized</u>	<u>Ligated</u>
Improved	22	2
Unchanged	7	1
Progressive Deficit	2	1
Death	3	2
Favorable Outcome	85%*	50%*

*p < 0.05

Table 6. Collected Results of Treatment of Carotid Artery Injuries in the Presence of Coma

Total Cases Reported - 23

	<u>Revascularized</u>	<u>Ligated</u>
Improved	4	2
Unchanged	0	0
Progressive Deficit	2	0
Death	9	6
Favorable Outcome	27%*	25%*

*Not Significant

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER 14 GRS7-76- 0 42	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER 9 rept.
4. TITLE (and Subtitle) 6 Protocol for Management of Penetrating Carotid Artery Injury.	5. TYPE OF REPORT & PERIOD COVERED Technical Report December 1975 - 31 December 1976	
7. AUTHOR(s) 10 Lazar J. Greenfield, M.D. William G. Liekweg, Jr., M.D.	8. CONTRACT OR GRANT NUMBER(s) 15 N00014-76-C-0422	9. PERFORMING ORG. REPORT NUMBER
9. PERFORMING ORGANIZATION NAME AND ADDRESS Medical College of Virginia Virginia Commonwealth University Box 786, Richmond, Virginia 23298	10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 105-821	11. CONTROLLING OFFICE NAME AND ADDRESS Office of Naval Research Department of the Navy 800 N. Quincy Street, Arlington, Virginia 22217
11. CONTROLLING OFFICE NAME AND ADDRESS Office of Naval Research Department of the Navy 800 N. Quincy Street, Arlington, Virginia 22217	12. REPORT DATE 11 8 February 1977	13. NUMBER OF PAGES
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office) Office of Naval Research Resident Representative John Hopkins University, Room 98 Garland Hall, 34th & Charles Street, Baltimore, Maryland 21218	15. SECURITY CLASS. (of this report) Unclassified	15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) 12 19p. Distribution of this report is unlimited		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number)		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Results of treatment of patients with penetrating isolated carotid artery injuries with or without neurological damage have been reviewed at the Medical College of Virginia and compared with published experience from the surgical literature. On the basis of these outcomes a Protocol For Management is described, emphasizing the value of revascularization surgery in all cases including coma when prograde flow is present. Ligation of the carotid artery following injury may still be indicated for technical reasons and in comatose		

402539 LB

#20 → patients in the absence of prograde flow.



OFFICE OF NAVAL RESEARCH
BIOLOGICAL & MEDICAL SCIENCES DIVISION
MEDICAL AND DENTAL SCIENCES PROGRAM, CODE 444
DISTRIBUTION LIST FOR TECHNICAL, ANNUAL AND FINAL REPORTS

Number of Copies

- (12) Administrator, Defense Documentation Center
Cameron Station
Alexandria, Virginia 22314
- (6) Director, Naval Research Laboratory
Attention: Technical Information Division
Code 2627
Washington, D. C. 20375
- (6) Director, Naval Research Laboratory
Attention: Library Code 2029 (ONRL)
Washington, D. C. 20375
- (3) Office of Naval Research
Medical and Dental Sciences
Code 444
Arlington, Virginia 22217
- (1) Commanding Officer
Naval Medical Research and Development Command
National Naval Medical Center
Bethesda, Maryland 20014
- (1) Chief, Bureau of Medicine and Surgery
Department of the Navy
Washington, D. C. 20375
- (2) Technical Reference Library
Naval Medical Research Institute
National Naval Medical Center
Bethesda, Maryland 20014
- (1) Office of Naval Research Branch Office
495 Summer Street
Boston; Massachusetts 02210

Enclosure (3)

- (1) Office of Naval Research Branch Office
536 South Clark Street
Chicago, Illinois 60605
- (1) Office of Naval Research Branch Office
1030 East Green Street
Pasadena, California 91101
- (1) Office of Naval Research
Contract Administrator for Southeastern Area
2110 G Street, N.W.
Washington, D. C. 20037
- (1) Commanding Officer
Naval Medical Research Unit No. 2
Box 14
APO San Francisco 96263
- (1) Commanding Officer
Naval Medical Research Unit No. 3
FPO New York 09527
- (1) Officer in Charge
Submarine Medical Research Laboratory
Naval Submarine Base, New London
Groton, Connecticut 06342
- (1) Scientific Library
Naval Medical Field Research Laboratory
Camp Lejeune, North Carolina 28542
- (1) Scientific Library
Naval Aerospace Medical Research Institute
Naval Aerospace Medical Center
Pensacola, Florida 32512
- (1) Commanding Officer
Naval Air Development Center
Attn: Aerospace Medical Research Department
Warminster, Pennsylvania 18974
- (1) Scientific Library
Naval Biomedical Research Laboratory
Naval Supply Center
Oakland, California 94625

(1)

Commander, Army Research Office
P. O. Box 12211
Research Triangle Park
North Carolina 27709

(1)

Director, Life Sciences Division
Air Force Office of Scientific Research
1400 Wilson Boulevard
Arlington, Virginia 22209

(1)

Commanding General
Army Medical Research and Development Command
Forrestal Building
Washington, D. C. 20314

(1)

Department of the Army
U. S. Army Science and
Technology Center - Far East
APO San Francisco 96328

(1)

Assistant Chief for Technology
Office of Naval Research, Code 200
800 N. Quincy Street
Arlington, Virginia 22217