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1. PURPOSE: To provide information of interest and assistance to the medical services of the US Armed Forces in Vietnam.
2. GENERAL: This headquarters does not necessarily endorse the professional views or opinions that may be expressed in this pamphlet apart from official notices. The contents of this pamphlet are not directive in nature.

(AVHSU)

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TABLE OF CONTENTS

	Page
Malaria	1
Hepatitis	6
Enteric Diseases	7
Shigellosis	
Amebiasis	
Nonspecific Gastroenteritis	
Tropical Sprue	
Cholera	
Parasitic Infestations	11
Typhoid Fever	14
Plague	15
Melioidosis	17
Tuberculosis	19
Tetanus	20
Gas Gangrene	21
Leptospirosis	22
Dengue Fever	23
Chikungunya	24
Japanese Encephalitis	25
Scrub Typhus	26

	Page
Murine Typhus	26
Rabies	27
Snake Bites	29
Dermatologic Problems	31
Venereal Diseases	35
Immunization	38
Toxins	39
Diagnosis and Management of Acute Renal Failure	41
Allergies and Asthma	44
Heat Injury	47
Duodenal Ulcer	48
Cardiac Disease	48
Trauma - Early Treatment of Wounds and Injuries	49
Use of Blood and Blood Products in Vietnam	51
New Arrivals in Vietnam	53

INTRODUCTION

This issue of the USARV Medical Bulletin is devoted to diseases likely to be encountered in Vietnam. In general, the subject matter is introductory in nature and appropriate references are listed for detailed study. The practices outlined are taken from the experiences of many and are not presented as the final answer to all problems.

Special recognition is due Major George Breza and Major Jay D. Morton for their editorial assistance on the sections covering enteric disease and acute renal failure.

The following reference material is available at all hospitals in the Command:

TB Med 31-	28 June 1962 (Scrub Typhus)
TB Med 119	2 September 1964 (Shigellosis)
TB Med 124	19 September 1968 (Plague)
TB Med 138	12 May 1967 (Cholera)
TB Med 142	2 March 1962 (Filariasis)
TB Med 164	14 July 1967 (Malaria)
TB Med 175	7 August 1957 (Heat Stroke)
TB Med 206	13 March 1961 (Viral Hepatitis)
TB Med 212	30 June 1959 (Viral CNS Problems)
TB Med 218	6 December 1956 (Epidemic Typhus)
TB Med 230	9 July 1965 (Venereal Disease)
TB Med 237	18 December 1962 (Laboratory Specimens)
TB Med 272	27 May 1966 (Dengue Fever)
TB Med 274	22 March 1967 (Meningococcal Disease)
TB Med 277	9 January 1968 (Intestinal Worms)
TB Med 278	29 October 1968 (Melioidosis)
Emergency War Surgery - NATO Handbook (1958)	

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MALARIA

Malaria is a parasitic disease caused by protozoa of the genus Plasmodium. It is transmitted by the bite of the Anopheles mosquito. Three species infect man in Vietnam: P. falciparum, P. vivax and P. malariae.

In the falciparum malaria the cycle within the liver terminates with the onset of the erythrocytic phase. Thus, the battle between the erythrocytic development of the parasite and the host defense concludes with termination of the illness or death of the host. In the other forms of malaria, the hepatic phase may continue for months and years accounting for relapses. Morphologic distinction of the various forms of malaria is important from the prognostic and therapeutic point. The falciparum variety invades any red cell regardless of age and thus may overtake a vast bulk of the erythrocytic population. Infestation of over 500,000 red cells has been associated with high mortality. Multiple infections occur within a single cell and bizarre forms are not uncommon. The developing schizont is rarely seen in the peripheral blood. Intra-vascular development within various organs occurs with release of the mature gametes. Thus in P. falciparum infection developing trophozoites and occasional gametes are seen. Marked pigmentation is unusual. In P. vivax infection, the younger 10 to 15% of the red cell population is involved. There is usually a single trophozoite per cell occurring in the mid portion of the cell and occupying one third of its diameter. As the schizont develops, pigmentation is easily recognized and known as Shufner's dots. Gametes, when seen, are round and within red cells. All stages from the early trophozoite to the mature gametocyte are often seen on a single blood smear. In P. malariae infection, the developing schizont contains 8-12 merozoites. This schizont has the appearance of a "daisy head". This malariae form usually invades the older cells and thus shares a small segment of the total red cell population.

INCIDENCE: Militarily, malaria has been the most important disease in Vietnam, accounting for the greatest number of man days lost. During the summer and fall of 1970 rates have been 25/1000/annum, of which 30% were due to vivax malaria and 6% due to mixed vivax and falciparum malaria.

TREATMENT: Following the identification of chloroquine-resistant strains of falciparum malaria in 1965, a new management program was developed and the details of which are covered in USARV Regulation 40-33. They are summarized as follows:

A. Radical Cure

1. Falciparum Malaria

Quinine tablets - 650mg q8h x 10 days.
Daraprim tablets - 25mg bid x 3 days.
Dapsone (DDS) tablets - 25 mg daily x 28 days.

2. Vivax and Malariae Malaria

Chloroquine tablets - 1gm stat, 500mg in 6 hours, then 500mg qd on day 2 and 3 for total - 2.5gms. (500mg Chloroquine tab = 300mg Base)
Primaquine (Fase) - 15mg qd x 14 days.

3. Exception: Dapsone (DDS) should not be given in the face of significant leukopenia. Gantrisin - 500mg q6h x 5 days may be substituted.

B. Prophylaxis

Chloroquine-Primaquine Tablets - 1 tablet weekly.

C. Relapses and Critically Ill Patients with Falciparum Malaria

Quinine - I. V. - 650mg in 1000cc 5% Dextrose in D/ W q8h.
Daraprim - 25mg bid x 3 days.
Dapsone (DDS) - 25mg qd x 28 days.

D. Mixed Infections

Therapy should be first directed at the falciparum infection, although it may subsequently be necessary to use a course of chloroquine to prevent vivax breakthrough. Primaquine therapy should be delayed until the course of therapy for falciparum malaria is completed.

E. Effectiveness

A recent study of 400 patients with falciparum malaria revealed an overall relapse rate of 12%. Relapse following an initial attack was 9% and following a second attack 27%.

Complications of Therapy have been multiple and varied, they have ranged from the development of methemaglobinemia from primaquine or dapsone to fulminant hemolytic anemia due to a deficiency of glucose - 6 - phosphate dehydrogenase. Quinine has produced symptoms of cinchonism, fevers, Coombs positive hemolytic anemias and pancytopenia. Dapsone has been incriminated in agranulocytosis. This resulted in the restriction of the use

of this agent to units operating in areas known or suspected to be hyper-endemic with falciparum malaria. (USARV Regulation 40-4).

"Plain chloroquine tablets may be substituted for Chloroquine-Primaquine tablets when it is determined by a physician that an individual is unable to tolerate Chloroquine-Primaquine because of undesirable side effects. In each case, the physician will annotate the individual's health record detailing medical reasons for the exception to this regulation. One chloroquine tablet will be taken each week by individuals authorized to do so by a physician and for eight weeks after departure. Each individual, who has taken plain chloroquine, will be given primaquine therapy, one tablet daily for 14 days upon departure. This will be waived by a physician if it is determined that the individual may have harmful side effects from primaquine."

In the rare case that an individual is allergic or unusually sensitive to both chloroquine and primaquine he should be offered prophylaxis with paludrine 100mg daily. Since this agent does not effect the exoerythrocytic phase the need for primaquine 15mg qd x 14 days on departure remains.

Cerebral Malaria is manifested as marked disorientation, delirium, and coma. Aggregated parasite - RBC clumps are found at post mortum in small cerebral vessels. Intravenous quinine is life saving but must be given slowly (600mg in 500cc 5% D/W q8h). Dexamethasone 8mg intravenously and 4 to 8 mg q6h is effective in management of the cerebral edema. Addition of heparinization is recommended in view of the frequent association of consumptive coagulopathy. This complication resulted in death in 8 patients in 1967, 6 patients in 1968, 4 patients in 1969 and 4 in 1970.

Renal Failure was reported 9 times in 1970 secondary to massive hemolysis and hemoglobinuria. This has occurred secondary to massive infections with falciparum malaria and also hemolysis related to drug therapy (G6PD Deficiency and/or Quinine induced Coombs Positive hemolytic anemia). All patients require careful fluid intake and output charting. The development of oliguria requires therapy with mannitol (20gms, 20% solution). The use of Edecrin or Lasix should be considered early. Renal failure may require communication with the Renal Unit, 3rd Field Hospital (923-2301).

Pulmonary involvement in falciparum malaria is rare (3%), but the onset of severe pulmonary edema in the hospital setting, after starting treatment, has been noted. A low central venous pressure, adequate circulatory system and urine output are generally present. Cardiac decompensation is not part

of the syndrome. Oxygen saturations are quite low. The exact mechanism is unknown but it is postulated that anoxia results in disruption of the vascular wall in septal pulmonary vessels, allowing diapedesis of fluid into the alveolar spaces. The role of histamine, kinins or immune phenomenon have not been adequately explained. Therapy is directed at oxygenation, relief of pulmonary congestion, and large doses of corticosteroids.

Disseminated intravascular coagulation occurs in those patients seriously ill with falciparum malaria. Thrombocytopenia, hypofibrinogenemia, and abnormal PTT tests, and ethanol gelation are noted. When this syndrome is suspected, heparin 0.5mg/kg I. V. q8h is indicated. Heparin is efficacious in the treatment of all severe cases of falciparum malaria. It has a direct effect on parasitemia and is directly effective against the basic pathologic change of small vessel occlusion.

Individuals who have had splenectomy are capable of assignment in low risk areas for falciparum malaria. When the latter have developed malaria it has responded well to conventional therapy.

G6PD Deficiency is now known to occur in over 100 million people. In its partial form it generally constitutes no problem. However, patients with severe hemolytic episodes are generally evacuated from Vietnam. The finding of G6PD deficiency per se does not require profiling or reassignment.

Disposition of patients with vivax malaria is straight forward and consists of returning the patient to duty when he is afebrile for two days and an enlarged spleen is not palpable. Those with persistent splenomegaly should have a longer period of observation. In patients with falciparum malaria the persistence of a syndrome of lethargy and hypotension in a number of these patients and the association of anemia with slow resolution requires reconditioning of these individuals before returning to duty and most will be referred to the 6th Convalescent Center at Cam Ranh Bay after control of symptoms. Transfer cannot be effected until the patient is afebrile for 48 hours and has a hematocrit over 30%.

References:

- TB Med 164, Malaria, July 1967.
- USARV Reg No 40-33, 15 Mar 1968, Change 1, 10 April 1969.
- USARV Reg No 40-4, Change 1, 20 October 1969.
- 44th Med Bde Message Unclassified AVHSU-M 45490, 5 Jul 1967.

Sheehy, T. W., Treatment of Chloroquine Resistant Plasmodium Falciparum Infaction in Vietnam, Ann. Int. Med. 66:616, 1967.

Sheehy, T. W., Complications of Falciparum Malaria and Their Treatment, Ann. Int. Med., 66:807, 1967.

Blount, R. E., et al, Management of Chloroquine - Resistant Falciparum Malaria, Arch. Int. Med., 119:557, Jun 1967.

Conrad, M., et al, Coomb's Positive Hemolytic Disease in Malaria, Ann. Int. Med., 68:32, 1968.

Brooks, M. H., et al, Acute Pulmonary Edema in Falciparum Malaria, New England Journal of Medicine, 279:14, 3 Oct 1968.

Response to Steroids in Pulmonary Involvement in Falciparum Malaria by Newman and Hall in the Sep-Oct 1969 USARV Medical Bulletin.

Preventive Medicine Symposium, Jul-Aug 1969 USARV Medical Bulletin.

Panel on Malaria, Ann. Int. Med. 70:127-154, Jan 1969.

Malloy, J. P., et al, Pathophysiology of Acute Falciparum Malaria, II. Fluid Compartmentalization, Am. J. Med., 43:745, 1967.

Dukes, D. C., et al, Oliguric Renal Failure in Blackwater Fever, Am. J. Med., 45:899, 1968.

NOTES:

HEPATITIS

This disease ranks next to malaria as a cause of noneffectiveness due to hospitalization for disease in this command. The average length of stay at the 6th Convalescent Center had been approximately 20 days. Clear cut epidemiologic patterns have evolved in only a few instances, the most consistent patterns stemming from consumption of non potable water and ice. The basic diagnostic and therapeutic principles are as outlined in most textbooks and TB Med 206. The backbone of treatment has been adequate diet and rest. Studies at the 6th Convalescent Center have strengthened Chalmer's report that bed rest is not essential in patients who have become asymptomatic. Generally these patients may have mess hall privileges and are permitted early ambulation. Isolation beyond hand washing is not enforced. The management of the patient requires that the prothrombin time be carefully scrutinized to separate early the severe patient likely to develop a stormy course. This patient should be held in an evacuation hospital until stable. Further, the differentiation from amebiasis or hemolytic disease is mandatory. Jaundice is not synonymous with hepatitis.

Gamma globulin prophylaxis is no longer used routinely but is prescribed in accordance with DA Circular 40-45. Special Forces personnel are presently the only troops in USARV authorized routine gamma globulin administrations. They receive two 5.0 ml injections, the first 30 days after arrival in RVN and the second five months after the first. Gamma globulin can continue to be used for the control of localized outbreaks of hepatitis and also may be used in individuals subjected to intimate exposure to known cases.

The incidence of serum hepatitis in RVN is unknown. Casualties requiring multiple transfusions have been evacuated out-of-country as soon as stable, and to date there has been no feed back from offshore hospitals indicating any significant problem. Gamma globulin has not been given routinely to patients receiving multiple transfusions.

References:

- TB Med 206, Viral Hepatitis, 13 Mar 61.
- DA Circular 40-45, 22 Mar 68.
- USARV Reg 40-7, Change 1, 21 Jun 67.
- Doff, R. S. and Isselbacher, K. J., Changing Concepts in the Epidemiology of Viral Hepatitis, NEJM, 278:1371, 1968.
- Treatment of Acute Infectious Hepatitis, Chalmers et al, Journal of Clinical Investigation, 1955.
- Bulletin of the New York Academy of Science, Symposium on Hepatitis in the Tropics, 1969.
- Hillis, W. D., Viral Hepatitis, Military Medicine, 133:343, 1968.

ENTERIC DISEASES

Diarrheal disease has traditionally been of major military importance because of the severe debilitating effects and alarming speed with which epidemics may occur. Our experience in Vietnam has been in keeping with this principle. Diarrheal outbreaks have in the past caused considerable morbidity, and have been studied extensively. In 90% of patients studied no specific etiologic agent could be isolated. Shigellosis was the most common specific cause. *Salmonella* infections have not been a significant problem. Staphylococcal food poisoning outbreaks have occurred sporadically and differ in no way from those in CONUS. Tropical sprue has been recognized frequently. To date schistosomiasis has not been encountered. With this general background, an approach to evaluation of the patient with diarrhea diseases will be presented followed by comments on specific diseases.

A precise etiologic diagnosis of diarrheal disease is vital both to the patient and to the military community in which he serves. In most instances an adequate history and physical examination will be invaluable in ruling out specific etiologies. The epidemiologic setting may suggest staphylococcal enterocolitis or *Salmonella* infection. Nocturnal diarrhea should suggest diabetes mellitus. Multisystem complaints with chronic diarrhea i. e. arthritis, skin rashes, abdominal pain, weight loss must invite consideration of regional enteritis, Whipple's disease, and ulcerative colitis. Diseases such as hyperthyroidism, chronic pancreatitis, celiac disease, tuberculosis, lymphosarcoma, acute and chronic appendicitis must not be forgotten.

Certain procedures, readily available, but requiring individual effort can be most rewarding in the search for an etiologic diagnosis.

For example:

1. Gross examination of stool - Presence of blood or mucus in an adult in this area suggests Shigellosis or Amebiasis. A rice water stool in this area is characteristic of cholera.
2. Proctoscopy - especially valuable in the differentiation of acute bloody diarrhea, the bowel mucosa of patients with shigellosis is edematous and fiery red with sharply circumscribed ulcerations penetrating to the muscularis mucosa. In contrast amebic colitis is characterized by scattered ulcerations with ragged, elevated and deeply undetermined margins and the intervening mucosa appears normal.
3. Microscopic examination - of aspirates from colonic ulcers may demonstrate trophozoites of *E. histolytica*. Amebic colitis is characterized by predominate mononuclear cells on smear of the aspirate whereas in

Shigellosis, polys predominante. Gram stain of the stool is useful in the diagnosis of staphylococal enterocolitis.

4. The lab may give further assistance via stool cultures and special procedures for the demonstration of Ova & Parasites.

I. SHIGELLOSIS:

The peak incidence months for diarrhea are April, May, and June - probably due to a seasonal increase in shigellosis. This disease may occur, however, in both sporadic and epidemic fashion throughout the year. Patients usually present with a febrile illness, often with headache; progress to nausea and vomiting; and finally develop severe abdominal cramps and diarrhea. Watery green stools are most common although bloody diarrhea may occur. The proctoscopic and x-ray findings usually revert to normal in two to six weeks.

Most patients with shigellosis will respond to parenteral fluids to correct the dehydration, and codeine or Lomotil to control the cramps. In the severely ill patient, antibiotics should be added. Tetracycline, 0.5 gm every six hours, is the antibiotic of choice although recent studies have demonstrated cases of tetracycline resistance. In the Saigon area Neomycin 0.5 gm every six hours for five days has been most effective. A failure to respond to tetracycline in 48 hours is ample justification for instituting neomycin if culture and sensitivity are not available. Neither sulfas nor Furoxone have been used routinely in this command.

References:

TB Med 119, Shigellosis, 2 Sep 64.

Heggers, J. P. and Smith, C. D., Sensitivity of Shigellae to Various Antibiotics, USARV Med Bul, May - Jun 67, p 53.

Stone, G. D., Shigellosis in Saigon, Vietnam, USARV Med Bul, Jan - Feb 68, p 57.

II. AMEBIASIS:

The most difficult problem faced in amebiasis is that of accurate diagnosis. Of key importance is the distinction between phagocytes engulfing red cells, as seen in shigellosis, and the classic motile trophozoite of Endamoeba histolytica. The diagnosis may be further obscured by the frequency with which antibiotics are dispensed or are purchased on the local economy, and the use

of chloroquine in malaria prophylaxis. The spectrum of clinical syndromes varies from the totally asymptomatic cyst passer to fulminant amebic colitis or extra-intestinal amebiasis, i. e., acute amebic hepatitis or amebic liver abscess. Facilities for liver scan are not available in Vietnam and when this study is required patients must be evacuated to Camp Zama Army Hospital, Japan.

Standard therapy for amebic colitis, amebic hepatitis and liver abscess consists of Flagyl 750 mg tid for 7-14 days. With liver involvement, therapy must be individualized, depending on clinical response. Some patients may require longer course of therapy. Common side effects of Flagyl are anorexia, nausea, and dizziness, which may be controlled by a lower dose schedule. Cures of hepatic amebiasis have been reported using 250 mg tid of Flagyl for 7 days. In the acutely ill patient unable to take oral medications, emetine 65 mg I. M. or subcut. may be given for 7 days. This drug should be used with extreme caution in patients with cardiac disease. All patients on emetine should have daily EKG's and therapy discontinued if conduction abnormalities appear. Emetine should be discontinued when the patient is able to take medication orally and a course of Flagyl instituted. Tetracycline 250 mg qid for 20 days should be added to the above regimens to treat possible bacterial hepatic abscess. In all patients, subsequent re-evaluations to include proctoscopy and LFT should be accomplished after completion of therapy at monthly intervals.

References:

TB Med 159, Amebiasis, 21 May 1959.
American Journal of Tropical Medicine, Sep 1970.

III. NONSPECIFIC GASTROENTERITIS:

As implied by the designation, no specific etiologic factor is identified in this group of diarrheal diseases. The role of pathogenic E. coli, Vibrio parahemolyticus and various viruses is speculative. Failure to respond to symptomatic therapy within 48 hours should result in more aggressive efforts to establish a specific cause. Of particular importance is the recognition of a cluster of cases which may indicate a break in mess sanitation in a given unit and call for prompt remedial action. The use of antispasmodics and bowel rest is usually effective.

IV. TROPICAL SPRUE:

This disease has been of unique military interest since WW II. The clinical picture was well defined by Steffanini based on a study of Italian POW's confined

in India. Subsequently the United States Army has been keenly interested in the subject. The precise etiology remains obscure although an infectious basis is strongly suspected. Studies have shown that Special Forces troops are at a particularly high risk although sporadic cases have occurred throughout the command. Cases comparable in severity to those seen in WW II are seen infrequently, due perhaps to the widespread use of tetracycline. Tropical sprue should be considered in any unusually refractory case of non-bloody diarrhea with bulky, greasy or malodorous stools. Facilities for performing d-xylose studies are available at most hospitals in the command. The definitive diagnosis rests on small bowel biopsy. Therapy consists of folic acid, 5 mg, three times a day indefinitely and tetracycline 250 mg, four times a day for two to four weeks. Vitamin B12 may be required in selected cases. At the present time tropical sprue does not preclude an individual from assignment to RVN. The therapy outlined above must be maintained. Long term antibiotic therapy is under study.

References:

Stefanini, M., Clinical Features and Pathogenesis of Tropical Sprue, Medicine, 27:379, 1948.

Sheehy, T. W., et al, Tropical Sprue in North Americans, JAMA, 194:1069, 1965.

Lindenbaum, V., et al, Malabsorption and Jejunitis in American Peace Corps Volunteers in Pakistan, Ann. Int. Med, 65:1201, 1966.

Klipstein, F. A., Tropical Sprue, Gastroenterology, 54:275, 1968.

French, A. B., Tropical Sprue - Specific Disease or Extreme of a Spectrum, Ann. Int. Med., 68:1362, 1968.

V. CHOLERA:

Cholera is a threat to the indigenous population but has not involved any US military personnel to date. Therapy consists of adequate parenteral fluid replacement and oral tetracycline. Introduction of the latter has dramatically reduced the morbidity and the requirement for parenteral fluid.

References:

TB Med 138, Cholera, 12 May 1967.

Carpenter, C. C. J., et al, Clinical Studies in Asiatic Cholera I-VI, Bull Hopkins Hospital, 118:165, 1966.

NOTES:

PARASITIC INFESTATIONS

Parasitic infestations are common in southeast Asia. Careful examination of warm stool specimens in suspect individuals generally results in high yields of positives. Careful attention to rapid processing of stool specimens is required if positives are to be achieved.

Clinical presentation and laboratory diagnosis are adequately covered in TB Med 277. Therapy for the various parasitic infestations is continually changing; therefore some comment of the drugs of choice and alternate therapy seems warranted. Because most of the drugs listed can have serious adverse effects, dosage recommendations are also given and should be exceeded only with special caution.

TABLE OF DRUGS

<u>INFECTING ORGANISM</u>	<u>DRUG</u>	<u>DOSAGE (Adult)</u>
	*(1) Drug of choice	
	*(2) alternate drug	
A. Roundworms		
1. <i>Ascaris lumbricoides</i>	(1) Piperazine citrate (2) Thiabendazole	- 2.4 gm daily x 2 days - 25 mg/kg bid x 2 days
2. <i>Trichuris trichiura</i>	(1) Thiabendazole (2) Hexylresorcinol (.01% solution)	- 25 mg/kg bid x 2 days - 500 ml by rectal retention for 1 hour
3. <i>Necator americanus</i>	(1) Tetrachlorethylene (2) Bephenium (3) Thiabendazole	- 0.12 ml/kg single dose (max - 5 ml) - 5 gm packet bid x 3 days - 25 mg/kg bid x 2 days
4. <i>Ancylostoma duodenale</i>	(1) Same as for #3 (2) Combined RX (Very good) Bephenium Tetrachlorethylene	- - 1/2 packet -- qd x 3d
5. <i>Strongyloides stercoralis</i>	(1) Thiabendazole (2) Pyrvium pamoate	- 25 mg/kg bid x 2 days - single dose, 5 mg/kg (max-250mg); repeat after 2 weeks.
6. <i>Enterobius vermicularis</i>	(1) Piperazine citrate (2) Thiabendazole	- 65 mg/kg (max-2.5 gm) qd x 8 days - 25 mg/kg bid x 2 days

7. *Trichinella spiralis* (1) No specific therapy (2) Thiabendazole - Adrenal corticosteroids for severe symptoms - 25 mg/kg bid until symptoms subside or toxic effects occur

8. *Cutaneous Larva migrans* (1) Thiabendazole (Dog and Cat hookworm) - 25 mg/kg bid x 2 days; repeat in 2 days if necessary

9. *Visceral larva migrans* (1) No specific therapy; (Dog and Cat roundworm) adrenal corticosteroids for severe symptoms - 20-40 mg prednisone daily for 3-5 days (2) Thiabendazole - 25 mg/kg bid until symptoms improve or toxicity develops.

10. Filaria
 a. *Wuchereria bancrofti* (1) Diethylcarbamazine - 2 mg/kg tid for 14 days
 b. *W. (Brugia) malayi*
 c. *Loa loa*

B. Flukes

1. *Clonorchis sinensis* (1) Chloroquine phosphate - 250 mg tid x 6 wks (2) Bithionol - 30-50 mg/kg qod x 15 doses

2. *Paragonimus westermani* (1) Bithionol - 30-50 mg/kg qod x 15 doses (2) Chloroquine phosphate - 250 mg tid x 6 wks

C. Protozoa

1. *Entamoeba histolytica* (1) Flagyl - 750 mg tid x 10 days (2) Emetine HCL plus tetracycline - 1 mg/kg (IM) qd x 10 days followed by Diiodohydroxyquin - 250 mg qid x 5 days - 650 mg tid x 20 days

2. *Dientamoeba fragilis* (1) Diiodohydroxyquin - 650 mg tid x 10 days (2) Tetracycline - 250 mg qid x 7 days

3. *Giardia lamblia* (1) Quinacrine HCL - 100 mg tid x 5-7 days (2) Metronidazole - 250 mg tid x 10 days

4. <i>Trichomonas vaginalis</i>	(1) Metronidazole	- 250 mg tid x 10 days
5. <i>Balantidium coli</i>	(1) Oxytetracycline (2) Diiodohydroxyquin	- 500 mg qid x 10 days - 650 mg tid x 20 days

Although this list is incomplete it does cover the major parasites encountered in South Vietnam. For toxic effects of the sited drugs refer to standard references.

Human schistosomiasis has not been demonstrated in South Vietnam. This is fortunate because, if it were present, operation in watery terrain would be seriously endangered. *S. mansoni* has been found in Americans but it has not been contracted in Vietnam.

Other tissue-flukes do occur in Vietnam. The liver-fluke, Clonorchis sinensis, and the lung-fluke, Paragonimus westermani, are present. They are transmitted to man by ingesting the encysted larvae in uncooked fresh water fish and crustaceans. The intestinal fluke, Fasciolopsis buski, also occurs and is contracted by eating raw aquatic plants, such as the water chestnut.

Filariasis due to the tissue roundworms, Wuchereria bancrofti and W. malayi, is endemic in some localities. The disease is transmitted when a mosquito bites an infected individual who is circulating microfilariae in his blood. The microfilaria must undergo about two week maturation in the mosquito before it can be transmitted to man when the infected mosquito bites again. The initial presentation with recurring fever and lymphadenitis can be quite confusing, and diagnosis by demonstrating microfilariae in the peripheral blood may not be possible until a year and a half after the infection is acquired. Retrograde lymphangitis is a clinical clue to the presence of infection. This disease could be seen among soldiers after return from Vietnam. There are a number of mosquito vectors (Culex, Aedes and Anopheles species) and primary prevention obviously centers upon not being bitten by these.

References:

- TB Med 142, Filarial Infections of Man, 1962.
- TB Med 277, Human Infections Caused by Common Intestinal Worms, 9 Jan 1962.
- Spencer, F. M. and Monroe, L. S., The Color Atlas of Intestinal Parasites, Charles C. Thomas, Springfield, IL 1966.
- Hunter, Frye and Swartzwalder, A Manual of Tropical Medicine, 4th E., W. B. Saunders, Philadelphia, PA 1966.
- U. S. Navy Medical News Letter, vol 54, Nov 1969, pp 16-25.

NOTES:

TYPHOID FEVER

Typhoid fever occurs in U. S. troops in Vietnam. There were eight cases in 1969 and 21 cases in 1970. Chloramphenicol remains a specific drug for treatment of the acute infectious process. Relapse is high even with two weeks of therapy. Intestinal perforation remains a most serious complication. Surgical intervention has been necessary. Attention to fluid balance is mandatory. Ampicillin has been effective in carrier states although cholecystectomy may be required.

Reference:

Management of Typhoid Fever and its Complication, Woodward, T. E., and Smadel, J. E., Annals of Internal Medicine, Volume 60 No. 1, 1964.

NOTES:

PLAQUE

In contrast to world wide decline in the number of plague cases reported in the last decade, the incidence has shown a steady and alarming increase in Vietnam since 1960. From a few hundred cases in the early 1960's there was an increase to over 8000 cases. Although techniques of reporting have improved, this reflects a real and significant increase. Plague is primarily a problem of the civilian populace but has had a secondary impact on US military operations due to the involvement of CIDG personnel and their dependents, as well as third country nationals. An epidemic in late February and early March 1967 brought port operations at Cam Ranh Bay to a halt for 14 days. In addition to the classic reservoir, two new rodents have been implicated. These are the oriental rat, Rattus exulans, and the common house shrew, Suncus murinus. The vector continues to be the infected rat flea, Xenopsylla cheopis. Pneumonic plague can, however, be transmitted from man to man through the respiratory tract by means of infected droplets or aerosols.

Bubonic plague is the most frequently encountered, although the spectrum runs the gamut from pneumonic to "asymptomatic" pharyngeal plague. Bubonic plague has been diagnosed only seven times in immunized US servicemen while serving in Vietnam.

The diagnosis of Bubonic Plague may be established by bubo aspiration. The organism is a gram negative rod, pleomorphic, non-motile, and appears either single, in pairs, or occasionally in chains. It takes a bipolar stain with Giemsa or Wayson, but not with gram stain. It grows well on blood agar, is non-hemolytic and produces small, round transparent glistening colorless colonies. The organism is pathogenic for rats and guinea pigs. Death occurs in 2-5 days following subcutaneous inoculation. Post mortum reveals cutaneous ulceration, regional lymphadenopathy, congestion of liver and spleen, and pleural effusion. The organism can be recovered from the spleen. Cultures can be identified by bacteriophage typing or agglutination with specific antisera.

Streptomycin remains an effective mode of therapy, although tetracycline and chloramphenicol in doses of 2-4 gms per day are equally effective. Again, the ready availability of antibiotics in RVN often obscures the cultural confirmation of the disease.

An intelligent understanding of preventive measures is critical. Environmental sanitation is the biggest problem. The proper sequence of control measures during an epidemic (real or suspected) warrants emphasis. While rodenticides are used routinely, at the time the rat flea index

climbs and an epidemic appears imminent, flea control (dusting) should be accomplished first. An increased rodenticide program will kill the rats and drive the fleas to new (human) hosts compounding the problem. The isolation of contacts is of minor importance in bubonic plague but is of major significance in outbreaks of pneumonic plague in order to limit respiratory transmission of disease from man to man.

U. S. personnel are routinely immunized using a vaccine derived from dead P. pestis organisms. Immunization programs for the civilian populace have been implemented in scattered areas of the country. These should not be done without proper clearance and approval of the province medicine chief. The Pasteur Institutes have developed a one dose vaccine using live attenuated organisms. This vaccine must be kept refrigerated, and there is a marked variation in potency between lots. The serological assessment of the latter shows an immune response in as few as 50 percent in some studies.

References:

TB Med 124, Plague, 13 May 60.
Hunter, D. W. and Dangerfield, H. A., Plague in Vietnam, USARV Medical Bulletin, Jan-Feb 1967, p. 34.
Marshall, J. D., et al Ecology of Plague in Vietnam, Military Medicine, 132:896, 1967.
Courad, F. G., et al A Recent Epidemic of Plague in Vietnam, Arch. Int. Med., 122:193, 1968.

NOTES:

MELIOIDOSIS

Melioidosis is an infectious disease caused by the gram-negative bacillus, Pseudomonas pseudomallei. More cases have been reported from Southeast Asia than any other site in the world. The disease has a broad clinical spectrum including subclinical infection, pulmonary involvement, osteomyelitis, infected wounds, and a fulminant septicemic form. The most common clinical manifestation is an acute pneumonic process. With melioidosis pneumonitis, the patient usually appears acutely ill with temperatures as high as 103 degrees and has a cough productive of scant amounts of yellow, occasionally blood-streaked sputum. When the patient has primary pulmonary involvement, he frequently has pleuritic pain, but rarely is there evidence of pleural effusion. Pleural effusions, however, are encountered when the disease presents as septicemia. Usually there is involvement of the upper lobes and/or the superior segments of the lower lobes with a cavitary infiltrate. The radiologic picture may be confused with tuberculosis. Septicemia can be rapidly fatal and requires prompt, vigorous parenteral therapy. A significant number of septicemic cases have been associated with burn patients, although the apparent site of initial infection may be relatively minor wound. Cultures of sputum, blood, urine, and wounds may lead to a definitive diagnosis. A hemagglutination test is available in Vietnam and is a useful adjunct in establishing a diagnosis of melioidosis. This serological test should be requested in all cases of fever of unknown origin.

Effective drugs in the treatment of melioidosis are tetracycline, novobiocin, chloramphenicol, and gantrisin. Tetracycline appears to be the drug of choice since almost all strains of Pseudomonas pseudomallei are sensitive to this drug. Although some strains of Pseudomonas pseudomallei are sensitive to kanamycin, because of the toxicity in the dosages required to be effective, it is not recommended in initial therapy. Practically all strains of Pseudomonas pseudomallei are resistant to penicillin, streptomycin, colistimethate (Coly-mycin) and cephalothin (Keflin). Combination therapy should be considered in the first stages of treatment in an acutely ill patient. Prolonged therapy is essential to prevent relapse. Tetracycline and gantrisin have been effective for prolonged therapy without adverse side effects. In cultural proven disease, therapy should be continued at least 60 days after clinical response. In practice, in pulmonary melioidosis 90 days of therapy have kept relapses to a minimum and six months of therapy have been used for septicemic disease.

Melioidosis centers have been established at Fitzsimons General Hospital and Valley Forge General Hospital for patients evacuated from Vietnam. Following treatment, patients are usually fit for duty.

References:

Blohm, R. W., Melioidosis, USARV Medical Bulletin, Aug - Sep 1966, p 39.
Conte, N. F., Human Melioidosis, USARV Medical Bulletin, Sep - Oct 1967, p 45.

Krebs, R. A., Diverse Manifestations of Melioidosis, USARV Medical Bulletin, Nov - Dec 1967, p 41.

Copper, E. B., Medioidosis, JAMA 200:452, 1967.

Spotnitz, M., An analysis of Nine Cases of Benign Melioidosis Pneumonitis, JAMA 202:950, 1967.

TB Med 278, Medioidosis, 29 Oct 1968.

Cooper, E. B., Treatment of Melioidosis, JAMA (Letter to the Editor) 204:176, 1968.

Spotnitz, Treatment of Melioidosis, JAMA (Letter to the Editor) 204:176, 1968.

Consultants Corner, USARV Medical Bulletin, Mar-Apr 1969, p 23.

Weber, et al, Acute Varities of Melioidosis Occurring in US Soldiers in Vietnam, Amer. J. of Med., Feb 1969.

NOTES:

TUBERCULOSIS

Tuberculosis remains a major problem among the Vietnamese. Treatment of Vietnamese civilians outside the framework of projects designed by the Ministry of Health and in conjunction with the province chief are to be condemned. These are generally haphazard and self-limited attempts which serve only to develop resistant strains in the population. They provide no follow up or lasting benefit.

Maximum effective utilization must be made of such basic tools as the history, physical examination, skin test and chest film. Bacteriologic study should be restricted to acid fast stain of the sputum. Many tuberculosis suspects are evacuated from country before results of cultures can be made available. The 9th Medical Laboratory has therefore requested that AFB culture requests be kept to a minimum. Therapy will not be initiated in RVN except in circumstances where it is considered lifesaving. Early evacuation of suspected tuberculosis patients is mandatory.

References:

TB Med 236, Tuberculosis.

Sproul, J., Antituberculosis Chemotherapy in Developing Countries; and Approach, USARV Medical Bulletin Sep - Oct 1969.

NOTES:

TETANUS

Adequate immunization has prevented tetanus in U. S. military personnel, but a number of cases have been seen in Vietnamese including several neonates. Meticulous supportive care is the foundation of treatment. Convalescence may be extremely slow. Mortality remains high even in the best university centers.

The following approach has been utilized in one of our hospitals and has resulted in survival of 10 out of the last 12 patients treated at that institution. Curare has not been used. This has eliminated hypostatic pneumonia as a complication. The program is as follows:

1. Admit to an intensive care area.
2. Perform a tracheostomy.
3. Debridement of suspect areas of infection.
4. Valium (IM or IV) - 5 to 10 mg stat and every 2 hours to control increased muscle tone or seizural activity.
5. Control respiration for 48 hours using an Emerson Respirator.
6. Hypertet (Hyperimmune Globulin) - 5000-6000 units.
7. Active immunization with Tetanus toxoid - begin with treatment.
8. Aqueous penicillin G - 10 million units every day for 5 to 7 days.

NOTES:

GAS GANGRENE

Gas gangrene, a myonecrosis caused by Clostridium perfringens should be considered a surgical disease. U.S. personnel are not spared and sound basic surgical management is the best prevention. Perfringens antitoxin is valueless except as a medico-legal token measure in civilian practice; it was dropped from the Army medical stock list years ago. Penicillin is the systemic antibiotic of choice and should be given in massive doses of 20 to 30 million units or more daily. Topical Sulfamylon solution will control local progression and often spare the patient mutilative excision. Hyperbaric oxygen has not been proved to add much to the regimen. If it is readily available, it probably will not hurt, but a critically ill patient is better off with uninterrupted care right where he is.

NOTES:

LEPTOSPIROSIS

In Southeast Asia leptospirosis is a relatively mild disease. Smith and Metzger have reported serological differences between those cases seen in RVN and those seen in CONUS. The disease is characterized by prominent headache, myalgia, and some degree of photophobia and is generally self-limited. The most prominent signs are conjunctival suffusion and hepatic tenderness. Leptospiral pneumonia has been seen. Weil's disease is infrequently seen, although a few patients with both hepatic and renal involvement associated with severe oliguria were hospitalized during 1969. Although a spontaneous and brisk diuresis occurred in some patients a requirement for either hemo - or peritoneal dialysis does occur. Isolation of the causative agent is not practical under field conditions so the diagnosis must be established by a rise in antibody titre of paired (acute and convalescent) sera. Identification in the urine has been possible. Inoculation of guinea pigs can be performed at the 9th Medical Laboratory. Treatment is symptomatic. Penicillin and tetracycline therapy has been used but the precise relation of these drugs to clinical improvement is hard to assess.

References:

Smith, C. W. and Metzger, J. F., Leptospirosis, A Serological Study of RVN Patients, Geographic Pathology Division, AFIP, Washington, D. C. and 406th Medical Laboratory, US Army Medical Command, Japan.

Allen, G. L. and Weber, D. R., Leptospirosis in South Vietnam, USARV Medical Bulletin, Jan - Feb 1967, p 23.

Allen, G. L. et al The Clinical Picture of Leptospirosis in American Soldiers in Vietnam, Military Medicine, 133:275, 1968.

NOTES:

DENGUE FEVER

Dengue fever is an infectious disease caused by a Group B arbovirus. The most common vectors are the Aedes aegypti and Aedes albopictus mosquitoes. The disease is characterized by the acute onset of fever, severe splitting frontal headaches, ocular pain, myalgia, arthralgia, non-tender lymphadenopathy, and rash. The fever may last five to six days and may be diphasic. Leukopenia develops during the course of the disease. A mild form of this classical picture exists and is usually less than 72 hours in duration. Therapy of the mild and classical forms of dengue fever is symptomatic and mortality is nil. A serious form with hemorrhagic manifestations has been seen in children in the Philippines and Thailand with a significant mortality, but this syndrome has not been recognized in U.S. troops.

References:

Sanford, Jay P., Harrison's "Principles of Internal Medicine," 6th edition, chapter 234, pp 1014-1015, McGraw-Hill, New York, 1970.

Clarke, D. H. and Casals, J., Horsfall and Tamm's "Viral and Rickettsial Infections of Man," 4th edition, chapter 27, pp 615-622, J. B. Lippincott Co., Philadelphia, 1965.

World Health Organization, Arboviruses and Human Disease, WHO Chronicle, 21:460, 1967.

TB Med 272, Dengue Fever.

NOTES:

CHIKUNGUNYA

Chikungunya is an infectious disease caused by a Group A arbovirus. It was first recognized in Africa, but also has been found in Southeast Asia. The disease is characterized by the sudden onset of fever, myalgia, and arthralgia. The joint pains are sometimes severe enough to be incapacitating. There actually may be objective arthritis involving the knees, ankles, shoulders, wrists and proximal interphalangeal joints. An irritating maculopapular rash is common. Treatment is symptomatic. No deaths have been ascribed to the disease.

References:

Sanford, Jay P., Harrison's "Principles of Internal Medicine," 6th edition, chapter 239, pp 1024-1025, McGraw-Hill, New York, 1970.

Casals, J. and Clarke, D. H., Horsfall and Tamm's "Viral and Rickettsial Infections of Man," 4th edition, chapter 26, p 584-586, J. B. Lippincott Co., Philadelphia, 1965.

NOTES:

JAPANESE ENCEPHALITIS

Japanese encephalitis is caused by a Group B arbovirus. The major vector is the Culex tritaeniorhynchus mosquito. Usually there is the abrupt onset of fever, rigors, severe headache, and nausea and vomiting. This is followed by confusion and disorientation. The disease may progress to coma, but some patients show only lethargy and somnolence. Within the first few days of illness, there may be oculomotor muscle paresis, nystagmus, facial weakness, difficulty in swallowing, hemiparesis, or monoparesis. There is predominantly upper extremity involvement. A specific diagnosis can be made only by isolation of the virus or by demonstration of a rise in titer in the hemagglutination test or complement-fixation test. The treatment is mainly supportive. Parenteral adrenal cortical steroid has been effective in certain cases. In 1970 there were 61 cases of viral encephalitis reported in the U.S. Army Medical Command facilities with peak incidence between May and September. A prospective study in 1970 showed 36 cases had a selective titer rise to Japanese B arbovirus.

References:

Sanford, Jay P., Harrison's "Principles of Internal Medicine," 6th edition, chapter 218, pp 978-982, McGraw-Hill, New York, 1970.

Clarke, D. H. and Casals, J., Horsfall and Tamm's "Viral and Rickettsial Infections of Man," 4th edition, chapter 27, p 626-631, J. B. Lippincott Co., Philadelphia, 1965.

NOTES:

SCRUB TYPHUS

This entity has an appeal to physicians not only because of its tongue twisting synonym (tsutsugamushi disease) but also because of the interesting clinical picture which it presents. It has been of particular concern in Vietnam as a possible cause of FUO, as commented on by Deller et al, and also as a concurrent disease in malaria patients (6%). The clinical picture and diagnostic methods are well outlined in TB Med 31. One significant variation is that it is difficult to find the classic eschar at the site of the infected mite bite. Due to perspiration and maceration a crust may never form or may slough, and the "eschar" may consist only of a denuded papule. Fever is generally to 104 and 105°. Backache and muscle pain are prominent. The conjunctiva may be suffused and a blotchy macular torsal rash may be apparent. Prior to the development of generalized tender adenopathy by the fifth day a careful physical examination will reveal local tender inguinal adenopathy early in a large number of cases. Tetracycline is a specific therapeutic agent for scrub typhus and provides prompt control of the disease. A five gram total dose consisting of a loading dose of up to three grams (25mg per kg) and 0.5 gram every six hours is usually adequate. Both chloramphenicol and tetracycline are rickettsiostatic therefore the suppressive effect may be relatively short lived. Relapses may occur if the disease is treated early, i. e., before immunity develops. Such relapses respond promptly to retreatment and can be prevented by routine retreatment six days from the end of the first course of therapy or delaying initial therapy for 72 hours while under hospital observation.

References:

TB Med 31, Scrub Typhus (Mite-borne Typhus Fever, Tsutsugamushi Disease), 28 Jun 1962.
Deller, J. J. and Russell, P. K., An Analysis of Fever of Undetermined Origin in American Soldiers in Vietnam, Ann. Int. Med., 66:1129, 1967.
Elisberg, B. L., Typhus Fevers, Current Therapy, 1967 (H. F. Conn, ed.), Philadelphia: W. B. Saunders Co., 1967.

MURINE TYPHUS

Murine typhus due to *R. typhi* (*mooseri*) and transmitted by bite of infected rat fleas, is similar in presentation to scrub typhus with the exception that the disease is generally milder, no eschar or regional adenopathy is noted. The rash is much less apparent. Treatment is the same as for scrub typhus. Serological studies to confirm the diagnosis is mandatory. This disease is quite common in US troops.

RABIES

Rabies is hyperendemic in Vietnam. Approximately 25 percent of dog heads examined by the 9th Medical Laboratory have been positive. Therapy of dog bite in Vietnam should be based on an acute awareness of this figure. Every exposure to possible rabies infection must be individually evaluated. Each major medical treatment facility should have a Rabies Control Board on orders to perform this evaluation (Reference USARV Reg 40-15, para. 9, 30 Jul 70). Annex A of USARV Reg 40-15 is reproduced on the next page; it is a guide for post-exposure anti-rabies treatment.

References:

USARV Reg 40-15, 30 Jul 70
DA Technical Bulletin 8-21, p 10, 15 Oct 67.
USARV Medical Bulletin, Sep-Oct 69.

NOTES:

ANNEX A

(USARV Reg No 40-15)

GUIDE FOR POST-EXPOSURE ANTI-RABIES TREATMENT

The following recommendations are intended only as a guide. Every exposure to possible rabies infection must be individually evaluated. It must be borne in mind, however, that rabies is hyperendemic in RVN.

Biting Animal		Treatment		
Species	Status at Time of Bite	Exposure		
		No lesion	Mild*	Severe*
Domestic dog or cat	Healthy	None	None ¹	S ¹
	Signs of rabies	None	V ²	S&V
	Escaped or unknown	None	V	S&V
	Rabid	None	S&V	S&V
Wild animals or bats	Regard as rabid in unprovoked attack	None	S&V	S&V

V = Rabies Vaccine

S = Antirabies Serum

1 = Begin vaccine at first sign of rabies during quarantine period.

2 = Discontinue vaccine if animal is healthy 5 days after exposure, or if brain is negative in animal killed at time of attack. If quarantined animal dies after 5 days and brain is positive, resume treatment.

*Definition of Exposures:

SEVERE: Multiple or deep puncture wounds, and any bites on the head, face, neck, hands or fingers.

MILD: Scratches, lacerations or single bites on other areas of body than the head, face, neck, hands or fingers. Open wounds, such as abrasions, which are suspected of being contaminated with saliva also belong in this category.

SNAKE BITES

The magnitude of the snakebite problem in Vietnam has been overplayed. Nevertheless, correct management of even the occasional bite is crucial. Common poisonous snakes include the cobra, king cobra, bamboo viper, Malayan pit viper and the banded krait. Extremely rare are sea snakes and Russell's viper. As a rough rule of thumb, the most common bites in Military Region I are bamboo viper, in the central highlands banded kraits (the vipers and cobras are also found), in Military Region III bamboo viper. In the jungle any snake can be found. Viper bites cause severe local pain, swelling and hemorrhage followed by systemic hemorrhagic phenomenon. Bites of cobras and kraits will be followed in minutes by pain and numbness at the site and within a short while signs of neurotoxicity appear. Radical, incisional treatment is not indicated. Transporting a patient with a tourniquet may be hazardous.

Treatment has two goals; reassurance and inactivation of the venom. For this reason Haffkine polyvalent cobra, krait antiserum is available for use at all medical treatment facilities. It must never be used except for krait or cobra bites. It is not to be used unless neurotoxicity is noted (ptosis, glossopharyngeal paralysis). Evacuation to a snakebite center (24th Evacuation Hospital, Long Binh; 67th Evacuation Hospital, Qui Nhon; 91st Evacuation Hospital, Chu Lai; and 95th Evacuation Hospital, Da Nang) is indicated early. The polyvalent crotalid antivenom made by Wyeth has shown effectiveness against bamboo viper poisoning. Poisoning is a result of a hemotoxin causing abnormal clotting and is reversed by antiserum. Cryotherapy does not appear to be effective nor have prednisone, epsilon-amino caproic acid or fibrinogen been helpful in managing the coagulation defect associated with viper bites. Close observation and careful nursing support are essential. Because of the low order of toxicity from viper bites there is time to evacuate the patient to a snakebite center for therapy and stocks of antiserum are kept at these centers.

Currently available antivenom and the recommended dosage:

	FSN	
1. Cobra (Neurotoxic)	6505-689-0373	Haffkine Polyvalent (Bombay), 10-50 ampules.
2. Krait (Neurotoxic)		Haffkine Polyvalent (Bombay), 10-50 ampules.
3. Malayan Pit Viper (Hemotoxic)	6505-689-0567	Malayan Pit Viper Antivenom (Thai Red Cross Society) 2-10 ampules.

4. Bamboo Viper (Hemotoxic) 6505-680-2787 Crotaline Polyvalent Antivenom (Wyeth), 2-10 ampules.

5. Sea Snake (Myotoxic) 6505-689-0373 Haffkine Polyvalent (Bombay), 10-50 ampules
or
Sea Snake Antivenom 1-4 ampules CWLTH Serum Labs Melborne, Australia

References:

Travis, L. L., and Blohm, R. W., Snakebite in Vietnam, USARV Medical Bulletin, Mar - Apr 1967, p 14.

Poisonous Snakes in Vietnam, Animal Kingdom, Volume LXIX: 34, 1966.

Reed, A., Snakebites in the Tropics, British Medical Journal, August 1968.

Frazier, C. A., Diagnosis and Treatment of Insect Bites, CIBA Clinical Symposia 20, Jul - Sep 1968.

USARV Medical Bulletin, May - Jun 1970.

NOTES:

DERMATOLOGIC PROBLEMS

Several problems that are common but not unique to RVN warrant comment. Trichophyton infections are widespread but are particularly frequent in the Mekong Delta. The causative agent there is largely T. mentagrophytes while in central Vietnam T. rubrum is encountered with equal frequency. Involvement may be widespread, but is most frequent in the inguinal area, the feet and legs. It presents as reddish lesions, but can produce vesicles on the feet. Sometimes so-called "id", or allergic, reactions in the form of vesicles are found concurrently on the hands. Treatment includes frequent bucket soaks with plain water if there is oozing or secondary infection; griseofulvin-V, 500 mg once or twice daily with meals; erythromycin, 250 mg every six hours for secondary infection; and topical medication such as hydrocortisone-Vioform cream, Desenex, Whitfield's ointment, Tinactin solution or cream. The same is applicable for the hands. Resistant cases are being encountered and require persistent, prolonged treatment or evaluation at a dermatology center for addition of steroids if indicated.

Tinea versicolor appears in the caucasian individual as multiple light pink lesions located mainly on the trunk. In the negro the lesions are whitish to silvery in color and pruritic. Treatment is with Selsun and the following directions should be given to the patient.

1. Get in the shower and start working lather in your scalp, neck, chest and arms. Scrub well with small towel.
2. Rinse off.
3. Apply thin film of shampoo at bed time and leave over night.
4. Rinse off in the morning.
5. Do the same procedure once a week for four weeks.
6. There will be white marks left and these will take several months to re-color.

If showers are unavailable, topical Tinactin rubbed into the lesions is simple and acceptable. Verdefam solution applied twice daily is also recommended.

Moniliasis occurs mainly in the inguinal and axillary areas. It is deeply red, has an exudate, and satellite lesions outside the large area of involvement. It is treated with Mycostatin cream, Fungizone lotion, Mycolog cream, hydrocortisone-Vioform cream, or Sporotacin cream or lotion. (CAUTION: An ointment base will lead to further maceration; Tinactin will worsen the erupt-

Acute vulgaris causes much discomfort and pain. Cystic degeneration as well as pustules are encountered on the back, chest, shoulders, face, neck, arms, and even the buttocks and thighs. Tetracycline, starting with four tablets daily and lowering to one daily will often control the disease if given as long term therapy. Its effect is to lower the total sebum content and free fatty acid content of the sebum, rather than functioning as an antibiotic. Topical treatment includes Fostex soap, a lotion containing up to eight percent sulfur, and thorough washing.

Tropical ulcers are caused by a mixed bacterial flora. Treatment consists of frequent compresses, oral antibiotics and hydrocortisone-Vioform cream or bacitracin ointment. Several weeks of treatment are usually necessary, in order that the lesions may reepithelialize.

Contact dermatitis presents with erythema, vesication or dry eczematous changes at particular locations. Treatment includes compresses, topical hydrocortisone cream, hydrocortisone-Vioform cream. Oral prednisone (short-term course) may be required if the reaction is severe.

Urticaria is caused primarily by drugs or foods. Various antihistamines should be tried before systemic steroids are employed.

Alopecia areata is not uncommon in our troops. Tension and stress (military personnel) is often elicited in the history. Advice to the patient concerning regrowth of hair, and reassurance are necessary.

Warts are caused by infection with a DNA virus. Treatment of choice depends on the location of the lesions. Plantar warts should be treated weekly by paring down with a #15 blade, and the application of a strip of 40% salicylic acid plaster held in place by tape. An alternate method is the daily application, by the patient, of 20% salicylic acid in collodion (with applicator). Treatment may last several months but will avoid scarring or infection of the soles. These methods can also be used for warts elsewhere on the body. At a dermatology center, certain lesions may be desiccated and curetted, or treated by freezing with liquid nitrogen.

Warts are not an emergency skin problem and may wait for definitive care. Exceptions include warts in critical areas (trigger finger) or those causing pain.

Herpes progenitales is characterized by small vesicles on a swollen erythematous base. The lesion is tender and the patient describes burning. It may occur on any part of the penis and lasts two to three weeks. At times there may be secondary infection. It is best treated by cooling soaks using a glass of cold water twice daily followed by application of topical antibiotic.

ion; and griseofulvin is contraindicated for this.) Sitzbaths several times daily are also helpful for scrotal involvement, when available. Medication should be applied twice daily and applications can be continued for weeks after clearing has occurred to prevent recurrence.

Pyodermas occur mainly on the lower legs and do not respond favorably to topical antibiotics. Oral broad spectrum antibiotics are required (tetracycline, erythromycin, Lincocin). Use of anti-bacterial soap is routinely recommended. Local therapy in the form of pHisoHex scrubs are helpful.

Miliaria rubra (heat rash) presents as multiple red papules on the trunk, arms and thighs and is pruritic. Topical therapy, while not entirely curative, includes hydrocortisone lotion (60 gm hydrocortisone 1% cream (2 oz tube) and 60 cc water), frequent showers and dousing the arms and trunk with water randomly. Vitamin C 500 mg b. i. d. may be beneficial in mild cases and some have found the tetracycline effect on sebum (1 tablet t. i. d.) helpful. A simple effective lotion that can be utilized in addition as a coolant is as follows:

Prickly Heat Lotion

Menthol and Camphor	0.5
Salicylic Acid	4.0
Glycerin	10.0
Alcohol 70%	qs 100.

Pseudofolliculitis barbae presents as multiple small nodules of papules with pustular formation. This is quite common in negroes. Findings are confined to the beard and chin. Soaking the beard area for five minutes before shaving and shaving only in the direction the hair grows is helpful. Depilatory cream or powder such as Magic Shave, Nair, or Surgex (Ciba) is recommended for this. It can be used every two to three days in place of razor shaving, since razor shaving may propagate the eruption. Tetracycline in low dose (250 mg b. i. d.) and topical steroids to reduce inflammatory reactions are useful.

"Blisterbug" dermatitis (Paederus family) produces burn-like lesions that can be up to five inches in diameter. Center is gray with vesicles and has a surrounding erythema. It takes two to three weeks to heal. A topical antibiotic-steroid preparation is helpful.

"Moth" dermatitis is produced by elements of the moths egg. Intensely pruritic papules are produced on exposed areas. It is seasonal and epidemic. Topical steroids are helpful.

Condylomata acuminata in the anal region or on the penis should carefully be treated with 25% podophyllin in tincture of benzoin, and washed off by patient in eight hours. (CAUTION: Divide treatment of large encircling lesion) Treat once or twice weekly until lesions have regressed.

Molluscum contagiosum presents as multiple small nodules in areas adjacent to the pubis, and on the penis. They resolve completely after removal with a curette.

Cutaneous larva migrans should be treated with oral Mintezol.

Generally, the preceding skin problems do not need immediate referral to a dermatologist, except in cases of treatment failure. The following problems should be referred to a Dermatology Clinic: psoriasis, vitiligo, dermatitis herpetiformes, and recurrent urticaria.

NOTES:

VENEREAL DISEASES

Venereal diseases have assumed new importance in Vietnam due to the poor response of gonococcal infections to the regimen outlines in TB Med 230, 1965. The situation is further complicated by the prevalence of other infections such as chancroid and lymphogranuloma venereum. Mixed infections are frequently encountered. Fortunately, syphilis is infrequent. Reasons for the poor response of *Neisseria* infections to conventional doses of penicillin remains obscure. The presence of true penicillin resistance still has not been demonstrated, however relative resistance is common. The basic TB Med remains an excellent outline for the diagnosis of all venereal infections.

Specific treatment programs have been implemented for use in-country. For acute gonococcal urethritis 2.4 million units aqueous procaine penicillin I. M. plus a priming dose of probenecid 1.0 gm, orally, followed by 0.5 gm probenecid 6 and 12 hours later. Treatment failures should be given 4.8 million units procaine penicillin I. M. in conjunction with the same dose schedule of probenecid. Individuals allergic to penicillin may be given tetracycline according to one of two schedules. Two doses of 2500 mg (10-250 mg tablets) given 12 hours apart has proven effective. Reactions are minimal and consist of transient nausea, light headedness, and occasional vomiting.

An alternate form of tetracycline therapy is a 1.5 gm priming dose followed by 0.5 gm every six hours for four days. The total dose is 8.5 gm. Because many patients cannot be relied upon to continue several days of treatment, this schedule should be used only after other methods have proven ineffective. A third alternative schedule that may be considered in penicillin sensitive or treatment failures is five to eight capsules of doxycycline monohydrate (50 mg) as a single dose.

Previous studies at 9th Medical Laboratory demonstrated that 50 of 100 bar girls at Vung Tau harbored *N. gonorrhoea*. Sensitivity studies on these organisms showed that high dose penicillin remains effective. Cephalin resistance was noted. Chloramphenicol was ineffective. Tetracyclines remained effective.

There is insufficient data to indicate the efficacy of these forms of treatment in the eradication of coincidentally acquired syphilis. All patients treated for gonorrhea should have a serologic test for syphilis (STS) at a minimum of six weeks after treatment, particularly when tetracycline is used. Patients should be advised to report promptly to a physician at the development of any suspicious genital or extragenital lesions. It

must be remembered that dark fields are not positive if treponema are seen. They must be identified as *T. pallidum*. All "chancres" should be seen and treated at the Dermatology Centers when possible so that confirmation of diagnosis and registry is accomplished.

Chancroid has been encountered with increasing frequency in recent months. It has posed several problems including refractoriness to single drug therapy. Chancroid has a tendency to complicate such procedures as circumcisions or even eradication of venereal warts with podophyllin. In uncircumcised males it is associated with tears of the frenulum. Two independently conducted studies indicate that a combination of sulfisoxazole in a dose of 1.0 gm four times a day for two weeks and tetracycline 0.5 gm four times a day for the same time period is highly effective. Refractory cases should be hospitalized and retreated with the same regimen. Specific attention being given to local cleansing. Occasionally circumcision will be required to control this problem. This program is effective in therapy of LGV. These patients also require serologic tests for syphilis as indicated earlier and repeat titer for LGV. Retreatment may be required in LGV cases.

There are several important administrative implications of venereal disease. The keynote of the Army's present policy is that it will not be used as the basis for punitive action against an individual. This is spelled out in TB Med 230, paragraph 2(b). Loss of duty time or quarters status is not generally required. Referral of complicated cases should be made to the nearest medical service and generally not to a Urology Service. "Refractory" urethritis patients are generally reinfection, herrela, gleat or non-specific urethritis. One full course of therapy (PCN 2 courses with Benemid and tetracycline or doxycycline, 1 course) should be used before labeling the patient refractory. Therapy through a whole tour without loss duty time is possible, if the patient insists on reinfecting himself. Penile discharge alone should not be used as a reason for relieving an individual from duties. Reinfection should not be considered as a "refractory" case and treatment is the same as for a primary case.

A number of problems have arisen regarding personnel who wish to go on R&R while under treatment for a venereal infection or who develop a venereal infection at the time of DEROS. A policy letter published 10 February 1968, requests that all physicians inform venereal disease patients that they will not be allowed to proceed on R&R until non infectious. It is not required that they be cured. This same letter spells out the follow up policy. Under provisions of USARV Reg 40-13, physicians are authorized to withhold clearance for return to CONUS in an individual with an infectious disease until therapy has rendered the disease non in-

fectious or up to 72 hours. Contact reporting is conducted in accordance with USARV Reg 40-6.

REFERENCES:

- TB Med 230, Treatment and Management of Venereal Disease, 9 Jul 65.
- TB Med 243, Interviewer's Aid for VD Contact Reporting, Jun 54.
- USARV Message 53282, AVHSU-M, 3 Jun 68.
- USARV Letter, Venereal Disease at Offshore R&R Sites, 10 Feb 68.
- USARV Reg 40-6, Prevention and Control of Venereal Diseases, 15 Mar 68.
- USARV Reg 40-13, Medical Clearance, 19 May 68.
- Public Health Report, Volume 84, No. 2, Feb 69.

NOTES:

IMMUNIZATION

The Army's basic immunization policy is covered in AR 40-562. Specific requirement for RVN are outlined in USARV Reg 40-7 revised 27 June 1968. An individual on duty in RVN will not be evacuated because he is unable to comply with the immunization requirements for this area. This applies particularly to cholera and plague vaccines. The requirement for yellow fever and typhus boosters has been dropped. By and large these individuals should not regularly participate in MILPHAP, MEDCAP or other activities that demand maximum protection against these diseases and are related to maximum contact with the local population. Reassignment within Vietnam is not indicated unless very special circumstances exist.

References:

AR 40-562.

USARV Reg 40-7, Immunization Requirements and Procedures, 27 Jun 68.
USARV Surgeon's Letter, AVHSU-M, Subject: Assignment Limitations and Fitness for Duties in RVN for Individuals with serious Allergic Reactions or Other Medical Contraindications to Immunizing Agents, 2 Feb 68.

DA Message, OTSG, MEDPS-SX, No. 177050, Subj: Medical Fitness for Service in Vietnam, 032100Z Oct 69.
Change 1, USARV 40-562, 20 Feb 71.

NOTES:

TOXINS

Three substances have caused serious problems due to their unusual character or frequency. Appropriate preventive measures and troop education have been implemented but it is anticipated occasional cases may still be encountered.

a. Ingestion of Composition C-4 Plastic Explosive. This syndrome was first encountered in early 1968 and presently C-4 intoxication has been one of the most prevalent causes of convulsions in US troops. The primary constituent of C-4 is RDX which is insoluble in water but which undergoes rapid hydrolysis in ethanol and is reduced to its constituents methylamine, nitrous acid, ammonia and formaldehyde. Manifestations include gastrointestinal irritation with nausea, vomiting and abdominal pain. Associated symptoms include hyperirritability, convulsions, coma and transient hematuria, oliguria and hepatic damage. The SGOT is generally always markedly elevated. These all reverse spontaneously although mental confusion and amnesia for events during the acute phase are significant. To date patients have recovered within 10 days to two weeks and have been returned to duty. EEG's have normalized prior to return to duty. Therapy is symptomatic consisting of anti-convulsants, hydration and careful nursing care. Careful attention to renal function is required.

References:

Medical Consultants Report, Jan 1968.

Merrill, J. L., Ingestion of an Explosive Material Composition C-4, USARV Medical Bulletin, Mar - Apr 1968, p 5.

Hollander, A. I. and Colbach, E. M., Composition C-4 Induced Seizures, USARV Medical Bulletin, Mar - Apr 1969.

b. Methyl Alcohol. This syndrome differs in no way from that encountered in CONUS. The usual source is bootleg or adulterated liquor although some clever label forgeries as well as forged tax stamps have made careful detective work essential. Correction of the profound metabolic acidosis pathognomonic of methanol ingestion is the primary therapeutic goal and will invariably require intravenous bicarbonate. Treatment of shock, convulsions and secondary pulmonary problems are all managed in a conventional fashion. Severe pancreatitis is frequently seen.

c. Barbiturate Poisoning. Both accidental and purposeful over-dosage of barbiturates have been frequently encountered. Barbiturates are readily available in civilian pharmacies and have been a mainstay in suicide attempts. When drug ingestion is suspected both gastric lavage and administration of a cathartic are indicated. Lavage is contraindicated when a caustic is ingested. In the absence of a gag reflex, lavage should not be attempted in the field. Support of respiration, vasopressors as needed and induced diuresis with mannitol are the most important measures. The use of analeptic agents is controversial. When mannitol is administered and diuresis ensues, sodium replacement is important. Both peritoneal and hemodialysis are of limited value, the former because of poor diffusion, the latter because of the delay before it can be instituted. The prognosis is largely dose-related. It is adversely affected by secondary complications such as aspiration pneumonia.

NOTES:

DIAGNOSIS AND MANAGEMENT OF ACUTE RENAL FAILURE

DIAGNOSIS:

The early recognition of acute renal failure is a significant factor in survival. The hallmark is a rising serum creatinine and urea. For practical purposes in Vietnam, if the serum creatinine is 4 the patient is in renal failure. The BUN is a less specific test. If the BUN is 60, the patient may well be in renal failure. Non-renal failure causes of an elevated BUN include: (1) tetracycline administration; (2) corticosteroids; (3) G. I. bleeding; (4) diuretics; (5) excessive protein intake; (6) increased catabolism as in high fevers; (7) congestive heart failure; and (8) dehydration.

The most common pitfall obscuring early diagnosis is the patient with good urine output and a rising BUN and creatinine. He is in renal failure. Very frequently patients referred to the renal unit in renal failure and requiring hemodialysis have urine outputs greater than 1000 ml/24 hours. This non-oliguric form of renal failure can be observed in the following clinical situations:

1. Traumatic injury. The patient has tolerated initial surgery well with no hypotension during anesthesia, no evidence of a transfusion reaction, an insignificant history of nephrotoxic drugs, and has a normal post-operative BUN with good urine output. Anywhere from one to six weeks post injury the BUN starts to rise with no fall in urine output. This is invariably associated with areas of tissue necrosis and infection. An occult intra-abdominal abscess might be present, or a bandaged extremity might require further debridement.
2. Medical causes of non-oliguric renal failure that have been observed include (1) hemoglobinuria as in blackwater fever or G6-PD deficiency and (2) nephrotoxins such as colistin, kanamycin, gentamycin, and C-4 plastic explosive.

Oliguric renal failure is much more obvious. The urine output has fallen to less than 20 ml/hour or less than 400 ml/24 hours. A precipitating cause is usually apparent: (1) severe hemorrhagic shock between time of injury and resuscitation, (2) hypotension during surgery, (3) septic shock, (4) severe hemolysis, or (5) dehydration. The dilemma facing the clinician with an oliguric patient is distinguishing between prerenal and renal failure. Post renal failure is very unusual in the young population in Vietnam and can be excluded unless there is a question of trauma or inadvertant ligation of the ureters or renal blood vessels. Retrogrades should only be done if there is a strong suggestion of obstruction, and then only unilaterally.

There are two ways to distinguish prerenal from renal failure: (1) urine chemistries and (2) a therapeutic trial of mannitol and fluids. The latter will be discussed under Management of Renal Failure. In the oliguric patient simultaneous blood and urine Na, urea, creatinine, and osmolalities should be determined. The prerenal failure kidney of dehydration or ischemia from shock will put out (1) urine with a Na < 10 (the kidney is conserving Na) and (2) very concentrated urine with a U/P ratio (urine/plasma) that is high for urea, $> 20/1$; and high for creatinine, $> 70/1$. Urine osmolality should be high, > 600 osms.

The renal failure kidney has had tubular damage, and the tubule cells cannot conserve sodium and concentrate urine hence the urine Na is high > 20 ; the U/P urea low, < 10 ; and U/P creatinine low, < 30 . Urine osmolality is also low, 300-400.

MANAGEMENT OF RENAL FAILURE:

1. Prerenal. This type of renal failure is rapidly reversible. If prerenal failure is suspected the treatment should be to correct what is causing the decreased perfusion of the kidneys. If the patient is in shock his blood pressure should be raised to at least 80 systolic. If there is a question that the patient might be dehydrated he should receive 25 grams of mannitol IV, followed by the rapid infusion of 500 ml of saline. If his urine output increases to over 40 ml/hour, sustained hydration should be instituted to rehydrate the patient.

2. Renal or Acute Tubular Necrosis. This form of renal failure is not immediately reversible. If the urine chemistries and failure to respond to mannitol indicate this is present, the patient should be placed on a renal failure regimen. This includes:

A. Water Restriction. Daily fluid intake should be restricted to 700 ml/24 hours plus the 24 hour urine output. Forced hydration should not be continued because diuresis will not ensue and water intoxication and/or pulmonary edema may result.

B. Sodium. Sodium should be severely restricted.

C. Potassium. Hyperkalemia is the most immediate threat to life in the renal failure patient. Potassium intake should be severely restricted. Common sources of exogenous potassium include dietary protein, salt substitute, old bank blood, potassium penicillin (1.5 mEq K/1 million units). Hyperkalemia should be treated by the immediate infusion of two amps of sodium bicarbonate (88meq), two amps of calcium gluconate 10% (20 ml), 50 grams of 50% glucose (100 ml), and a 50 gram Kayexalate enema (200 ml) if available. This is the most common form of death of patients who die

on route to the renal unit and the above regimen should be given to all patients transferred to the renal unit regardless of serum potassium.

D. Protein. Protein should be restricted to 20 grams of animal protein (eggs)/day.

E. Drugs. If renal failure is severe enough to alter drug dosage, the patient should be transferred to the renal unit. Digitalis is very ineffective in improving the pulmonary edema of fluid overload in the patient who is oliguric and can give rise to arrhythmias as the electrolytes are altered during dialysis.

METHOD TO ARRANGE FOR TRANSFER TO 629th MEDICAL DETACHMENT (RENAL UNIT), 3rd FIELD HOSPITAL, SAIGON:

The importance of communication is to be stressed. If we are notified as soon as possible by the physician in charge of the patient we can alter our dialysis schedule and alert the surgical team.

TELEPHONE 923-2301

The patient should be transferred early rather than late. Physician to physician communication can best determine the time to do so. Patients are currently started on hemodialysis when a rising BUN and creatinine reach 100 mg% and 10 mg%, respectively.

SUMMARY:

1. Early referral of patients in renal failure to the artificial kidney team is an essential factor in survival.
2. The keystone to diagnosis is not urine output. It is serum creatinine and BUN.
3. If the creatinine is 4 the patient is in renal failure. The BUN is a less specific test. If the BUN is 60 the patient may well be in renal failure.
4. The most common pitfall in diagnosis is the patient with good urine output and rising creatinine and BUN. He is in renal failure. Very frequently patients referred to the renal unit in renal failure and requiring hemodialysis have urine outputs greater than 1000cc/24 hours.
5. Communication is available on a 24 hour basis. Telephone 923-2301.

ALLERGIES AND ASTHMA

In general, patients with allergies are fit for duty in USARV. Individuals undergoing desensitization need only provide their serum to their supporting medical unit and administration will be effected. If the individual has received serum from an Army allergy clinic the patients unit physician may request re-supply through military channels. Primary allergy evaluations are deferred to return to CONUS.

Asthmatics are fit for duty in USARV unless there is a requirement for repeated hospitalization and failure to respond to therapy. Decision for evacuation will be based on the findings of the consulting internist at the supporting hospital in conjunction with documented information provided by the unit physician.

Insect allergy does not preclude duty in the Republic of South Vietnam. A vaccine prepared by the allergy clinic, Walter Reed General Hospital for mixed stinging insects is available through the Medical Consultant USAMED-COM V (P), APO #96384. This vaccine should not be substituted 1 for 1 with vaccine from any other source. Skin testing is not warranted prior to instituting hyposensitization in this condition and will be based on history alone.

The following general and specific instructions for use of the vaccine are offered:

A. General Instructions.

1. A physician must always be immediately available.
2. Use a 26 - 27 gauge needle and give injections into the lower deltoid area subcutaneously.
3. Record date, dosage, and reaction to injections. Observe at least 20 minutes after injections.
4. Do not increase any dose which causes a skin reaction or which causes hives, rhinitis, or asthma. (See reactions.)
5. Reactions may be controlled by placing a tourniquet above the site and giving 0.2 cc of 1:1000 aqueous adrenaline into the injection site. Notify the physician!!

B. Method of Administration.

1. Keep extract refrigerated.
2. Give injections from vial #1 every 2 - 3 days. Start with 0.05 cc and increase by 0.05 cc until a dose of 0.5 cc is given. Then proceed to the next stronger vial and follow the same schedule until the full strength. The full strength vial should be started with 0.02 cc and then increased by 0.02 cc every 7 - 14 days until maintenance dosage (1 cc) is reached. Vial #7 is full strength and contains a total of 10,000 PNU per cc. Maintenance dosage should be continued for at least 2 years, at 7 to 30 day intervals.
3. The extract is dispensed in the following concentrations:

Vial 1 contains 1:1 million dilution of full strength						
"	2	"	1:100,000	"	"	"
"	3	"	1:10,000	"	"	"
"	4	"	1:1,000	"	"	"
"	5	"	1:100	"	"	"
"	6	"	1:10	"	"	"
"	7	"	full strength			

C. Reactions.

1. Negative - increase dosage according to schedule.
2. "A"-Small wheal (less than 1/2") - repeat the same dosage.
3. "B"-Wheal 3/4" - return to the last dosage which caused no reaction.
4. "C"-Wheal 1" or larger - give 1/3 of the last dosage which caused no reaction.
5. Systemic - Hives, angioedema, etc. - recheck strength of vial. Generally treat as in 4 above.

D. Management of missed injections.

1. One week - increase according to schedule.
2. Two weeks - repeat the last dose.
3. Three weeks - reduce by 0.10 cc.

4. Four weeks - reduce by 1/3 of last dose.

5. More than 4 weeks - re-start.

If further questions arise call the Medical Consultant, HQ USAMEDCOM V, APO #96384, telephone 926-4456/4815/5546.

NOTES:

HEAT INJURY

Heat injuries are a recurring problem despite years of medical and command interest. Adequate acclimatization combined with proper water replacement will prevent most if not all problems. Under some circumstances the tactical situation precludes adequate water supply and heat casualties will occur. The spectrum of clinical syndromes and their management are well outlined in TB Med 175. Every medical officer should thoroughly familiarize himself with its contents. Simple heat cramps and heat exhaustion are the most frequently encountered syndromes. Oral intake of 0.1 percent saline will adequately handle most cases; intravenous administration of normal saline can be used in more severe cases. It is easy to overload people with salt in a hot environment. Salt loading leads to serious potassium depletion associated with weakness, lethargy and cardiac arrhythmia. There may well be a distinct relationship between potassium depletion and heatstroke. Potassium can be added to intravenous fluids if urine output is maintained. The management of true heat stroke is more critical and includes lowering the body temperature, extremely judicious fluid replacement and treatment of associated consumptive coagulopathy. Acute renal failure associated with heat stroke has occurred in recruit populations in CONUS. If encountered in RVN such patients should be promptly evacuated to the Renal Unit, 3d Field Hospital (call 923-2341).

References:

TB Med 175, The Etiology, Prevention, Diagnosis and Treatment of Adverse Effects of Heat, 26 Nov 57.

Schrier, et al, Nephropathy Associated with Heat Stress and Exercise, Ann. Int. Med., 67:356, 1967.

Knockel, J. P., and Vertel, R. M., Salt loading as a Possible Factor in the Production of Potassium Depletion, Rhabdomyolysis and Heat Injury, Lancet I:659, 25 Mar 67.

Boaz, Colonel T., Jr., Some Comments on Water and Salt, Military Medicine, June 69.

NOTES:

DUODENAL ULCER

Duodenal ulcer disease has not been a major problem in Vietnam and is in keeping with previous findings that ulcer disease is not enhanced in a war zone. In USARV these patients are expected to perform, eat in mess halls and consume combat rations. Patient food selection advice should be provided by his physician. Unit physicians provide first line support and encouragement. When hospitalization is required it is generally brief and results in return to duty in all cases except for severe bleeding episodes, organic obstruction or perforation.

CARDIAC DISEASE

Patients on anticoagulants cannot be maintained in USARV except in special positions in areas with a dependable laboratory. Cardiac dysrhythmia should not be treated outside of hospitals except in an emergency. Patients in USARV hospitals with myocardial infarction are always evacuated by litter and not prior to 30 days hospitalization. All departments of medicine are required to have monitoring equipment. The 3rd Field Hospital has a fully equipped coronary care unit. A transvenous pacemaker is available in USARV.

NOTES:

TRAUMA - EARLY TREATMENT OF WOUNDS AND INJURIES

Although not specifically within the province of a review of the medical specialties certain principles require emphasis for all physicians serving in Vietnam.

Military medical care is provided in echelons. Standardized methods of managing wounds and injuries not only prevent the patient from suffering because of ill-conceived procedures, but also provide successive medical officers with the assurance that previous treatment has been sound. The DOD publication "Emergency War Surgery, NATO Handbook" provides clear guidance on the early treatment of trauma in a military environment. The local ground rules are further spelled out in USARV Reg 40-21 which has been revised on the basis of recent experience in RVN and was published 30 May 1968. Both the NATO Handbook and this regulation should be required reading for all medical officers.

The primary mission of battalion, brigade and division medical units is to provide the majority of patients only with sufficient treatment and resuscitation to permit their safe evacuation to a hospital. Treatment by these medical units should not go beyond the immediate necessities except for those patients whose wounds are minor enough to permit their retention and return to duty from these medical units. Strict adherence to this primary mission is essential if wounded in action (WIA) patients are to receive optimal care. In the prevention of death due to trauma it must be recognized that almost half the deaths (KIA) are due to brain wounds.

Most of the wounds to the brain are due to fragments and the steel helmet is capable of deflecting most of these fragments. If penetration does occur, the velocity of the missile has been markedly reduced and thus many more are then stopped by the skull and do not enter the brain. It cannot be stressed too greatly that the wearing of the steel helmet in all situations of combat or attack is of tremendous value towards the preservation of life and brain function, in all individuals, in all circumstances, and without regard to rank or pride.

In the management of the expectant head injured patient early evacuation is not indicated. This type of patient is best described as being deeply comatose, having fixed dilated or fixed constricted pupils, corneal anesthesia, irregular respirations not due to airway obstruction, no reaction to intense painful stimulation or decerebrate or decorticate posturing. This type of patient will receive expectant care at the neurosurgical center as well and emergency transfer is not indicated and in fact is contraindicated during inclement flying weather or at dangerous visibility. If this patient's condition improves over a several hour period and neurosurgical consultation is felt indicated, then transfer should be reconsidered.

References:

Emergency War Surgery, NATO Handbook.
USARV Reg 40-21, Early Treatment of Wounds and Injuries, 30 May 1968.
Current Problems in Surgery, Heaton, et al, November 1966.
CINCPAC Conferences on War Surgery 1967, 1968, 1969.
Fifth Conference on War Surgery, CINCPAC, March-April, 1971.

NOTES:

USE OF BLOOD AND BLOOD PRODUCTS IN VIETNAM

The most desirable blood for transfusion is as fresh as possible, group and type specific and completely cross-matched. Availability of this desirable situation requires about 40 minutes. Group specific blood with an immediate spin saline cross-match requires 15 minutes. Low titer O positive blood may be obtained immediately. During periods of increased need for blood, care should be taken to request no more than immediate contingencies dictate so that the cross-matching capacity of the laboratory is conserved and utilization of available units is maximized. A cross-matched unit on the shelf is usually not available to another patient. Group AB patients should be transfused with one of the following blood groups in order of decreasing preference AB, A, O (without reverting to a "more preferable" group as blood supplies again become available).

Low titer O positive (universal donor) blood is indicated for patients with acute severe hemorrhage, in mass casualty situations, and at small medical units where grouping and cross-matching are impractical. Because of its limited supply O negative blood is given only in group specific transfusions. No complications have been observed in-country to result from this practice, and sensitization of males to RH factors should not lead to clinical disease given the standard blood banking practices in CONUS. As a rule of thumb, all patients receiving four or more units of group O blood should continue to be transfused with group O blood unless a minimum of two weeks has elapsed since the last transfusion and the new cross-match is compatible.

Recent radioactive tracer studies have shown that whole blood stored in plastic bags has an acceptable shelf life of up to 31 days. Blood 21 days or less is ordinarily used. Blood between 21 and 31 days of age is retained as a back-up in mass casualty situations.

In the treatment of burns plasma from bank blood, aged plasma and/or Plasmonate are optimal. Fresh frozen plasma (FFP) should not be used.

In a patient with uncontrollable oozing following massive transfusion a coagulation work-up should be carried out prior to, or simultaneously with, treatment and should consist of at least an examination of a peripheral blood smear, platelets, a prothrombin time (PT) and a partial thromboplastin time (PTT). Generally, a simple dilution of coagulation factors is characterized by a prolonged PT and PTT with normal platelet and fibrinogen levels. There is a rapid response to 2 - 3 units of FFP.

In some instances platelets may also be depressed and fresh blood (FB) would be indicated. Hemorrhage due to disseminated intravascular coagulation is characterized by depressed platelet and fibrinogen levels and a lengthened PT and PTT. There is little or no response to FB and FFP. Heparin is the treatment of choice.

Hemorrhage due to fibrinolysis is indicated by a prolonged PT and PTT and a diminished euglobulin lysis time, shows little or no response to FB and FFB and is treated with epsilon amino caproic acid. In hemorrhage due to combinations of the above, the laboratory results may be inconclusive. In that case a trial with FB (3 - 4 units) and/or FFP may well be indicated.

NOTES:

NEW ARRIVALS IN VIETNAM

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NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Aaron, Richard A.	CPT/MC	20 Dec 70	23d Inf Div
Alcalay, Michael M.	CPT/MC	20 Nov 70	11th ACR
Alexander, Leslie	CPT/VC	24 Nov 70	4th Vet Det
Allison, Barrie H.	1LT/ANC	8 Jan 71	93d Evac Hosp
Alonso, Kenneth B.	CPT/MC	15 Nov 70	23d Arty Gp
Amos, Paul D.	2LT/MSC	22 Nov 70	23d Inf Div
Andrews, Charles E.	MAJ/ANC	2 Dec 70	3d Field Hosp
Antilla, Betty J.	MAJ/ANC	10 Nov 70	95th Evac Hosp
Aton, James K. Jr.	LTC/MC	17 Dec 70	95th Evac Hosp
Aupke, Susanne C.	CPT/ANC	31 Dec 70	3d Field Hosp
Baccus, Virgil T.	CPT/MC	20 Dec 70	23d Inf Div
Baker, Daniel C.	CPT/MC	10 Nov 70	22nd CAG
Baxter, David A.	CPT/MC	23 Nov 70	93d Evac Hosp
Beene, Jerry D.	1LT/MSC	5 Dec 70	58th Med Bn
Bennett, James R.	CPT/MSC	4 Jan 71	32d Med Depot
Benoit, Donna M.	LTC/ANC	17 Dec 70	3d Field Hosp
Bentley, William R.	LTC/MSC	4 Jan 71	61st Med Bn
Berger, Bruce W.	CPT/MC	27 Oct 70	93d Evac Hosp
Berger, William C.	CPT/MC	20 Dec 70	93d Evac Hosp
Berger, Winfried N.	MAJ/MC	4 Nov 70	18th Surg Hosp
Bergman, Stephen E.	CPT/MC	12 Nov 70	17th CAG
Bezold, Henry	1LT/MSC	21 Nov 70	58th Med Bn
Bishop, James A.	CPT/MSC	10 Nov 70	11th ACR
Boecker, Frederick W.	CPT/MSC	3 Dec 70	172d PMU
Boeders, William S. Jr.	1LT/MSC	8 Dec 70	61st Med Bn
Boone, Charles A.	CPT/ANC	12 Nov 70	18th Surg Hosp
Boyers, Stephen P.	CPT/MC	28 Oct 70	67th Med Gp
Brainerd, Henry D. Jr.	CPT/MC	20 Dec 70	101st Abn Div
Braun, Stephen E.	CPT/MC	23 Dec 70	212th CAB
Braverman, Howard L.	CPT/MSC	18 Nov 70	11th ACR
Brescia, Frank J.	CPT/MC	12 Nov 70	1/5th Mech Inf
Brinley, Jerald L.	CPT/MC	7 Dec 70	525th MI Gp
Brown, John R.	CPT/MC	27 Oct 70	18th Surg Hosp
Bryant, Goldon V. Jr.	1LT/MSC	28 Nov 70	1st Cav Div
Bryant, William D. Jr.	2LT/MSC	1 Jan 71	67th Med Gp
Bunch, James S.	CPT/MSC	28 Oct 70	58th Med Bn
Bux, Robert B.	CPT/MC	10 Nov 70	1st Cav Div

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Byrne, Richard R.	CPT/MC	28 Oct 70	68th Med Gp
Cafferata, Harold T.	MAJ/MC	15 Nov 70	3d Field Hosp
Caporusso, Frank J.	2LT/MSC	22 Nov 70	101st Abn Div
Carey, James P.	MAJ/MC	20 Dec 70	27th Surg Hosp
Carter, Samuel C.	LTC/MC	4 Nov 70	91st Evac Hosp
Chan, Patrick J.	MAJ/MC	27 Oct 70	24th Evac Hosp
Cherry, Edmond B. III	1LT/MSC	7 Jan 71	USAMEDCOMV
Ciffin, William F.	CPT/VC	20 Nov 70	175th Vet Det
Clore, Lawrence H.	CPT/MSC	22 Nov 70	32d Inf Div
Cochran, Henry S.	1LT/MSC	10 Nov 70	164th CAG
Cooley, William L.	2LT/MSC	4 Dec 70	1st Cav Div
Cooper, James O.	CPT/MSC	19 Nov 70	101st Abn Div
Copeland, Keith E.	MAJ/MSC	19 Nov 70	58th Med Bn
Crane, David L.	2LT/MSC	12 Nov 70	MACV
Cromartie, Robert S. III	CPT/MC	5 Jan 71	93d Evac Hosp
Cross, Ralph E. Jr.	CPT/MSC	23 Dec 70	101st Abn Div
Cunningham, Florence L.	1LT/ANC	4 Nov 70	3d Field Hosp
Curtis, Jimmy C.	CPT/MSC	15 Nov 70	222nd PSC
Dabbs, Richard W.	2LT/MSC	31 Dec 70	173d Abn Bde
Davis, Carl E.	MAJ/AMSC	4 Nov 70	67th Med Gp
Davis, Michael	CPT/MC	3 Nov 70	67th Med Gp
Deardorff, Virginia	2LT/ANC	21 Nov 70	67th Evac Hosp
Deem, Clark W.	CPT/MC	20 Dec 70	23d Inf Div
Deitch, Michael J.	CPT/VC	21 Dec 70	4th Vet Det
Deleese, Joseph S.	CPT/MC	12 Nov 70	12th CAG
Dionne, Raoul B.	CPT/MSC	24 Nov 70	23d Inf Div
Dumais, Helen C.	1LT/ANC	1 Jan 71	95th Evac Hosp
Eastling, Timothy A.	1LT/MSC	12 Nov 70	17th CAG
Eaton, Gregory L.	CPT/MSC	6 Nov 70	25th Inf Div
Edmondson, Lionel J. Jr.	CPT/MSC	10 Nov 70	23d Inf Div
Ehlers, Sara E.	1LT/ANC	8 Jan 71	95th Evac Hosp
Eiffler, Darrell D.	CPT/ANC	22 Nov 70	91st Evac Hosp
Everett, Bruce A.	CPT/MC	27 Oct 70	95th Evac Hosp
Fearon, Douglas T.	CPT/MC	27 Oct 70	68th Med Gp
Feldstein, Leslie M.	1LT/ANC	31 Dec 70	3d Field Hosp
Foust, Jerome V.	CPT/MSC	7 Jan 71	61st Med Bn

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Freeman, Michael A.	CPT/MC	20 Dec 70	1st Bde/5th Inf
Friedman, Alan D.	CPT/MC	27 Oct 70	3d Field Hosp
Furia, Frederick A.	CPT/MC	19 Nov 70	23d Inf Div
Furlow, Bruce M.	CPT/MSC	28 Oct 70	20th PMU
Furtney, Jennifer J.	1LT/ANC	22 Nov 70	67th Evac Hosp
Gailans, Ivars J.	CPT/MC	27 Oct 70	101st Abn Div
Gans, Jason Y.	CPT/MSC	21 Nov 70	68th Med Gp
Garcia, Benito	1LT/MSC	4 Dec 70	1st Cav Div
Garner, Mary B.	1LT/ANC	20 Nov 70	6th Conv Ctr
Garrard, Richard S.	1LT/MSC	19 Nov 70	932d DPS
Gennaro, Josephine	MAJ/ANC	21 Nov 70	24th Evac Hosp
Gilbert, Derl H.	2LT/ANC	31 Dec 70	3d Field Hosp
Gilbert, Roy G.	CPT/MSC	6 Nov 70	9th Med Lab
Gilden, Carl	CPT/MC	20 Dec 70	101st Abn Div
Gildersleeve, John W.	CPT/MC	15 Nov 70	18th Surg Hosp
Gillick, John S.	CPT/MC	13 Dec 70	93d Evac Hosp
Giron, Louis T. Jr.	CPT/MC	15 Nov 70	101st Abn Div
Goldsmith, Joel W.	CPT/MC	28 Oct 70	93d Evac Hosp
Goldsmith, John M. Jr.	1LT/MSC	4 Dec 70	1st Cav Div
Goodman, Francis D.	CPT/MSC	19 Nov 70	9th Med Lab
Gordh, Martina E.	1LT/ANC	1 Jan 71	95th Evac Hosp
Gordon, Allen F.	CPT/ANC	1 Jan 71	3d Field Hosp
Graunke, David A.	2LT/MSC	19 Nov 70	23d Inf Div
Green, Linda L.	1LT/ANC	5 Nov 70	93d Evac Hosp
Green, William E.	CPT/MSC	16 Dec 70	68th Med Gp
Greer, Terry A.	1LT/MSC	4 Dec 70	58th Med Bn
Grimson, Baird S.	CPT/MC	20 Dec 70	101st Abn Div
Griswold, Richard H.	2LT/MSC	12 Nov 70	101st Abn Div
Gross, Richard C.	CPT/MC	20 Dec 70	23d Inf Div
Gula, Elaine M.	2LT/ANC	10 Nov 70	24th Evac Hosp
Gunson, Kathleen S.	1LT/ANC	8 Jan 71	85th Evac Hosp
Gushwa, Richard L.	MAJ/MC	4 Nov 70	173d Abn Bde
Haas, Erwin J.	CPT/MC	10 Nov 70	17th CAG
Hallstead, Byron T.	CPT/MSC	20 Nov 70	58th Med Bn
Hannon, Bruce G.	2LT/MSC	12 Nov 70	America Div
Harrison, Nathan J.	CPT/MC	20 Dec 70	101st Abn Div
Harrison, Sandra J.	1LT/ANC	2 Jan 71	3d Surg Hosp
Hayes, Cheryl L.	CPT/ANC	28 Oct 70	95th Evac Hosp
Heitman, Kenneth L.	LTC/DC	30 Oct 70	437th Dental Det
Higgins, Walter E.	CPT/MSC	27 Oct 70	68th Med Gp
Hinds, Ervin A.	CPT/MC	27 Oct 70	91st Evac Hosp
Hodges, Don D.	CPT/MC	10 Nov 70	12th CAG

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Hoffman, Albert T.	1LT/MS	13 Nov 70	61st Med Bn
Hoffman, Herman W.	CPT/MS	31 Dec 70	9th Med Lab
Hofmeister, John F.	CPT/MC	27 Oct 70	68th Med Gp
Holliday, Michael J.	CPT/MC	10 Nov 70	1st Cav Div
Hood, Marion G.	MAJ/MC	28 Oct 70	3d Field Hosp
Horsley, Norma P.	MAJ/ANC	10 Dec 70	67th Evac Hosp
Hudson, Clifford C.	CPT/MC	23 Dec 70	17th CAG
Hunt, Ramon E.	CPT/CH	5 Nov 70	HQ 85th Evac Hosp
Jerke, Leonard V.	2LT/MS	10 Nov 70	101st Abn Div
Johnson, Stewart M.	MAJ/MC	15 Nov 70	3d Field Hosp
Jolly, Walter W.	MAJ/MC	20 Dec 70	93d Evac Hosp
Jones, Malin F.	CPT/MS	9 Jan 71	HQ USAMEDCOMV
Jost, Frank A. III	CPT/MC	27 Oct 70	68th Med Gp
Kaplan, Martin J.	CPT/MC	20 Dec 70	1st Cav Div
Kautzman, Dennis J.	CPT/MS	8 Jan 71	1st Cav Div
Kelly, Patrick C.	CPT/MC	22 Nov 70	23d Inf Div
Kias, Thomas N. Jr.	CPT/MC	20 Dec 70	67th Evac Hosp
King, William H.	CPT/ANC	20 Nov 70	85th Evac Hosp
Kozma, Marie G.	CPT/ANC	2 Dec 70	24th Evac Hosp
Krenzke, Cynthia M.	CPT/ANC	13 Nov 70	85th Evac Hosp
Krueger, James S.	CPT/MS	13 Nov 70	USAMEDCOMV
Kuehn, Dorothy M.	LTC/ANC	7 Dec 70	93d Evac Hosp
Kuharich, Richard	CPT/MC	8 Oct 70	224th Avn Bn
Labhart, Diane M.	2LT/ANC	1 Nov 70	67th Evac Hosp
Landeen, Robert H.	CPT/MC	15 Nov 70	101st Abn Div
Lathan, Gladys S.	MAJ/ANC	9 Jan 71	3d Surg Hosp
Leary, William J. Jr.	CPT/MS	7 Dec 70	68th Med Gp
Leath, Mary K.	LTC/AMSC	11 Dec 70	93d Evac Hosp
Leecy, Donna A.	CPT/ANC	20 Nov 70	93d Evac Hosp
Litt, Abraham A.	CPT/MC	20 Dec 70	164th CAG
Livingston, Allen S.	1LT/MS	20 Nov 70	61st Med Bn
Long, Jack C.	CPT/MC	20 Dec 70	12th CAG
Long, Reuel S.	CPT/MC	3 Dec 70	93d Evac Hosp
Longden, Paul K.	CPT/MS	11 Dec 70	67th Med Gp
Lonigan, Robert M.	CPT/MC	10 Nov 70	12th CAG
Lowden, Roland G.	MAJ/MC	1 Nov 70	9th Med Lab
Lunghofer, Victoria	CPT/ANC	29 Nov 70	3d Field Hosp
Lutz, Thomas W.	CPT/MC	27 Oct 70	68th Med Gp

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Mac Donald, Janette	2LT/ANC	16 Nov 70	3d Field Hosp
Malewski, Edward	CPT/MSB	31 Oct 70	101st Abn Div
Manelis, Joseph	LTC/MC	28 Oct 70	3d Field Hosp
Mannard, Enn	CPT/MC	15 Nov 70	Americal Div
Maravola, Patricia A.	CPT/ANC	20 Nov 70	3d Field Hosp
Marcieski, Stanley C.	1LT/MSB	5 Dec 70	58th Med Bn
Marko, Michael W.	CPT/MC	28 Oct 70	68th Med Gp
Marshall, Robert A.	CPT/MC	28 Oct 70	68th Med Gp
Martin, Hugh M. Jr.	CPT/MC	15 Nov 70	173d Abn Bde
Mathews, Robert D.	CPT/MC	20 Dec 70	24th Evac Hosp
McAdam, Lawrence P.	CPT/MC	10 Nov 70	17th CAG
McCarthy, Elizabeth J.	2LT/ANC	1 Nov 70	95th Evac Hosp
McGann, Martha E.	2LT/ANC	8 Jan 71	3d Field Hosp
McGannon, Michael L.	CPT/MC	21 Dec 70	164th CAG
McGinty, John D.	CPT/MSB	31 Dec 70	32d Med Dep
McGrew, Rae E.	1LT/ANC	20 Nov 70	6th Conv Ctr
Mercurio, Annette L.	2LT/ANC	22 Dec 70	91st Evac Hosp
Mielcarek, Donald J.	CPT/MC	20 Dec 70	12th CAG
Miller, Dwayne E.	2LT/ANC	20 Nov 70	3d Surg Hosp
Millican, Joe T.	CPT/MSB	4 Nov 70	172d Prev Med Unit
Mioduszewski, Diane	1LT/ANC	20 Nov 70	3d Field Hosp
Morphy, John C.	CPT/MC	27 Oct 70	24th Evac Hosp
Mumper, Diane L.	2LT/ANC	21 Nov 70	67th Evac Hosp
Musher, David R.	CPT/MC	9 Dec 70	93d Evac Hosp
Nark, Janis A.	2LT/ANC	17 Nov 70	6th Conv Center
Neary, Jack M.	CPT/ANC	1 Jan 71	95th Evac Hosp
Nelson, John C.	CPT/MC	8 Dec 70	3d Field Hosp
Newman, Philip H.	MAJ/MC	27 Oct 70	18th Surg Hosp
Newton, Douglas F.	CPT/MC	10 Nov 70	17th CAG
Noble, Patricia A.	1LT/ANC	20 Nov 70	3d Field Hosp
Normile, James P.	CPT/MSB	23 Nov 70	1st Cav Div
Norris, Jimmy A.	1LT/MSB	24 Nov 70	15th Med Bn
Novotny, Valerie E.	2LT/ANC	5 Dec 70	27th Surg Hosp
Nachtshem, Daniel A.	CPT/MC	20 Dec 70	93d Evac Hosp
Oakes, Roger M.	CPT/MC	28 Oct 70	68th Med Gp
Ogg, Michael J.	CPT/MC	27 Oct 70	27th Surg Hosp
Omahan, Kathleen R.	2LT/ANC	17 Nov 70	93d Evac Hosp
O'Neill, Martin J. Jr.	CPT/MC	20 Dec 70	1st Cav Div
Ordons, Kathleen L.	2LT/ANC	13 Nov 70	95th Evac Hosp
Ossmann, Paul M.	CPT/MC	10 Nov 70	17th CAG
Otis, Robert J.	LTC/MSB	10 Nov 70	USAMEDCOMV

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Paclik, Michael J.	CPT/VC	22 Nov 70	4th Vet Det
Park, Richard E.	CPT/MC	20 Dec 70	91st Evac Hosp
Parks, Justin D.	2LT/MSC	21 Nov 70	23d Inf Div
Paschia, Monica	2LT/ANC	12 Nov 70	93d Evac Hosp
Peiser, Robert S. Jr.	1LT/MSC	9 Jan 71	23d Inf Div
Peterson, Dan D.	1LT/MSC	22 Nov 70	1st Bde/5th Inf
Pharr, William F.	MAJ/MC	28 Oct 70	91st Evac Hosp
Poliakoff, Claude S.	MAJ/MC	27 Oct 70	91st Evac Hosp
Poole, Jimmy K.	1LT/MSC	21 Nov 70	23 Inf Div
Presto, Barbara N.	2LT/ANC	22 Nov 70	3d Surg Hosp
Price-Williams, Joan E.	1LT/ANC	1 Jan 71	3d Field Hosp
Proctor, Russell J.	CPT/MC	20 Dec 70	17th CAG
Quarton, Sally	2LT/ANC	12 Nov 70	93d Evac Hosp
Queen, Henry	CPT/ANC	12 Nov 70	91st Evac Hosp
Rainey, Edward C. Jr.	CPT/MC	15 Nov 70	1st Bde/5th Mech Inf
Reeves, Waverley B.	1LT/MSC	19 Nov 70	58th Med Bn
Reid, Roy M. Jr.	MAJ/MC	15 Nov 70	24th Evac Hosp
Reynolds, Mary T.	1LT/ANC	20 Nov 70	24th Evac Hosp
Rice, Robert E.	2LT/MSC	2 Dec 70	11th ACR
Richardson, Joel K.	2LT/MSC	22 Nov 70	68th Med Gp
Richter, Fred T.	1LT/MSC	31 Dec 70	172d PMU
Rigler, Wilson F.	CPT/MC	15 Nov 70	1st Bde/5th M Inf
Robertson, Howard T. II	CPT/MC	9 Dec 70	93d Evac Hosp
Rodgers, Richard W.	CPT/MSC	15 Nov 70	67th Med Gp
Roe, Rodney A.	CPT/MC	10 Nov 70	164th CAG
Romans, Michael H.	CPT/VC	24 Nov 70	175th Vet Det
Roth, Robert S.	CPT/MC	20 Dec 70	1st Bde/5th Inf
Rothgeb, Eric J.	CPT/MC	21 Dec 70	17th CAG
Rouse, Kenneth G.	1LT/MSC	12 Dec 70	11th ACR
Russell, James T. Jr.	CPT/MSC	8 Jan 71	68th Med Gp
Sanchez, Aida N.	MAJ/AMSC	23 Dec 70	95th Evac Hosp
Santana, Jorge A.	CPT/MC	28 Oct 70	67th Med Gp
Sasaki, Gordon H.	CPT/MC	17 Dec 70	93d Evac Hosp
Schlicke, Lutz H.	CPT/MC	15 Nov 70	101st Abn Div
Schroeder, Dannette E.	1LT/ANC	7 Nov 70	67th Evac Hosp
Sellards, Robert R.	CPT/MSC	10 Nov 70	173d Abn Bde
Sharkey, Daniel J.	1LT/MSC	21 Nov 70	61st Med Bn
Shively, Ellen M.	CPT/ANC	6 Nov 70	93d Evac Hosp
Shucart, Mary N.	CPT/ANC	30 Nov 70	91st Evac Hosp
Silver, Donna F.	1LT/ANC	24 Nov 70	3d Field Hosp

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
Sims, James A.	CPT/MC	15 Nov 70	101st Abn Div
Singh, Alvin R.	CPT/MSC	1 Dec 70	101st Abn Div
Sittingdown, Quincy	1LT/MSC	4 Dec 70	61st Med Bn
Smarr, Janet M.	2LT/ANC	16 Nov 70	3d Surg Hosp
Smith, Gayle M.	1LT/ANC	16 Nov 70	3d Surg Hosp
Smith, Maude M.	COL/ANC	2 Nov 70	USAMEDCOMV
Smith, Roy R.	MAJ/MSC	17 Nov 70	32d Med Dep
Smith, William J.	CPT/MC	29 Nov 70	XXIV Corps
Snell, Joan	MAJ/ANC	21 Nov 70	67th Evac Hosp
Sontag, Adolph J. Jr.	CPT/MSC	31 Dec 70	935th Med Det
Soyak, John A.	CPT/MSC	11 Jan 71	9th Med Lab
Sparks, Paul C.	CPT/MC	28 Oct 70	91st Evac Hosp
Stamps, Thomas E.	CPT/MC	20 Dec 70	11th ACR
Stewart, Elaine N.	2LT/ANC	22 Nov 70	6th Conv Cen
Strayer, Wendy D.	2LT/ANC	4 Dec 70	8th Field Hosp
Streb, Richard A.	CPT/MC	7 Dec 70	3d Field Hosp
Sway, Daniel H.	CPT/MC	20 Dec 70	108th Arty Gp
Szanto, Philip A.	MAJ/MC	1 Nov 70	9th Med Lab
Tabor, Barbara A.	2LT/ANC	21 Nov 70	27th Surg Hosp
Talley, Gaylyn L.	CPT/ANC	9 Jan 71	3d Field Hosp
Tardio, Philomina M.	LTC/ANC	2 Dec 70	93d Evac Hosp
Tate, Bessie M.	CPT/ANC	20 Nov 70	95th Evac Hosp
Thoesen, Mary A.	1LT/ANC	13 Nov 70	93d Evac Hosp
Thoesen, Richard C.	CPT/MSC	13 Nov 70	20th PMU
Thomas, David R.	CPT/MC	10 Nov 70	212th CAG
Thompson, Willard R. Jr.	CPT/MC	10 Nov 70	164th CAG
Trenouth, Roland S.	CPT/MC	20 Dec 70	1st Cav Div
Tukey, Sheila E.	1LT/ANC	23 Dec 70	93d Evac Hosp
Turtzo, Douglas F.	CPT/MC	20 Dec 70	1st Cav Div
Tyler, George	CPT/MC	20 Dec 70	1st Cav Div
Van Dyke, Alan R.	CPT/MC	21 Dec 70	101st Abn Div
Varela, Fausto R.	1LT/MSC	9 Dec 70	101st Abn Div
Varney, Kathryn E.	1LT/ANC	1 Dec 70	93d Evac Hosp
Voges, Peter W.	MAJ/MC	18 Nov 70	67th Evac Hosp
Vogt, William J. II	2LT/MSC	9 Jan 71	101st Abn Div
Wait, Ardis E.	1LT/ANC	1 Jan 71	95th Evac Hosp
Webb, Rita K.	1LT/ANC	16 Nov 70	3d Field Hosp
Webber, Charles E. Jr.	CPT/MC	27 Oct 70	27th Surg Hosp
Weiss, Ken H.	CPT/MC	22 Nov 70	11th ACR
Wheeler, Sondra K.	2LT/ANC	1 Nov 70	67th Evac Hosp

NAME	GRADE & BRANCH	ARRIVED	ASSIGNED TO
White, James O.	1LT/MSC	22 Nov 70	932d Den Prof Svcs
White, Stephen R.	CPT/MC	28 Oct 70	95th Evac Hosp
Williams, Barbara N.	1LT/ANC	22 Nov 70	6th Conv Cen
Willis, Robert H.	LTC/MSC	13 Nov 70	32d Med Depot
Wirth, Barbara	MAJ/MC	3 Dec 70	3d Field Hosp
Wisgerhof, Max V. II	CPT/MC	15 Nov 70	1st Bde/5th M Inf
Yep, Ronald	CPT/MC	12 Nov 70	3d Surg Hosp
Yarosh, Patricia M.	1LT/ANC	22 Nov 70	3d Surg Hosp
Zolezzi, Michael A.	1LT/MSC	8 Dec 70	61st Med Bn

USARV MEDICAL BULLETIN

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