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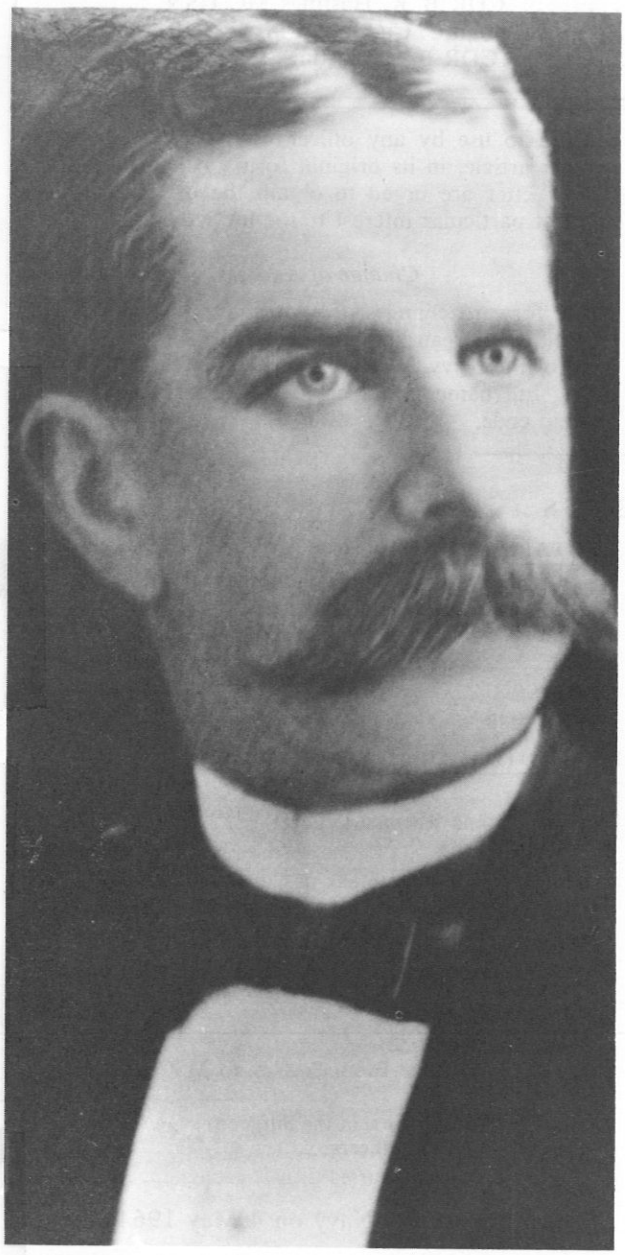

Surgeons General of the Past

(The seventeenth in a series of brief biographies)

Presley Marion Rixey was born at Culpeper, Va., 14 July 1852, graduated from the University of Virginia Medical School, and was commissioned an Assistant Surgeon in the Navy on 28 January 1874. He served through the various grades and was appointed the 13th Surgeon General and 17th Chief of the Bureau of Medicine and Surgery on 5 February 1902, continuing in that office until 4 February 1910. During his term, all naval hospitals were renovated and modernized, and new ones were constructed at Puget Sound (1903), Canacao, P.I. (1903), Las Animas, Col., for tuberculosis patients (1906), Great Lakes (1907), and Guam (1910). He also established medical supply depots at Brooklyn, San Francisco, and Cavite, P.I. Rear Admiral Rixey doubled the size of the Medical Corps and instituted the Naval Medical School at Washington in 1902 for the postgraduate instruction of officers in military medicine. He sent officers abroad to study tropical medicine and to civilian institutions for specialized study. He founded the United States Naval Medical Bulletin in 1907 to disseminate naval and general medical knowledge throughout the Medical Department. In 1908, the female Nurse Corps was established, the nurses being employed in naval hospitals, on hospital ships and at training schools for corpsmen and native nurses in America's island possessions. Surgeon General Rixey was instrumental in having a second hospital ship (RELIEF) fitted out for West Coast service and to support the fleet President Roosevelt sent around the world. He served Presidents McKinley and Roosevelt as personal physician, having entire charge of Mr. McKinley's case following his assassination, and left an important historical record of the event. He died 17 June 1928.

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The issuance of this publication approved by the Secretary of the Navy on 4 May 1964.

PROPHYLAXIS OF WARM-WATER-IMMERSION FOOT

Larry J. Buckels MD, Kenneth A. Gill, Jr., MD, and Gustave T. Anderson MD,
JAMA 200(8):681-683, May 22, 1967.

Warm-water-immersion foot has caused a large number of casualties among our troops fighting in Vietnam. A prophylaxis against this condition was studied. A silicone grease was applied to the feet of 52 men every 24 hours. Their feet were exposed to an occlusive, wet environment for five days. Mild or early changes of immersion foot developed in six of the men and all of the subjects completed the test. Six volunteers were exposed to the same wet conditions without using silicone grease; symptomatic immersion foot developed in five of these subjects and four were unable to continue in the test beyond the third day due to the severity of their symptoms.

In late 1965, one of us (G.T.A.) called attention to the problem of casualties arising from a condition known as "immersion foot" which was considered of operational significance by US troop leaders in Vietnam. The feet were white, wrinkled, and so painful that it was difficult to walk. This was first noted after an operation which took place during days of constant, heavy rainfall in the coastal lowlands. The men went for several days without being able to adequately dry their feet.

It is well known that in individuals who have the proper facilities and time to administer proper foot care wet-foot injury will not develop. However, there are always combat situations where proper foot care is not practical, feasible, or possible, despite command orders to the contrary, and thus the search for a practical prophylaxis against the development of immersion foot. The "immersion foot" term, as used in this communication, refers to warm-water-immersion foot and should not be confused with classical immersion foot, which is associated with injury following exposure to a wet, cold environment.

From the Physiology Division, Naval Medical Field Research Laboratory, Camp Lejeune, NC (Dr. Buckels), the Department of Dermatology, Naval Hospital, Camp Lejeune, NC (Dr. Gill), and the Bureau of Medicine and Surgery, Navy Department, Washington, DC (Dr. Anderson).

Recognizing the command's concern about the morbidity associated with this condition, the Research Division of the Bureau of Medicine and Surgery, Navy Department, initiated further investigation by this laboratory. Two investigators had recently carried out successful studies in preventing tropical immersion-foot syndrome (warm-water-immersion foot) by using a silicone preparation (high vacuum silicone grease) applied to the feet every 12 hours.

Initial studies with prophylactic measures in wet-foot injury date back to at least 1916 when lard and tallow with 4 percent to 10 percent sodium chloride were applied to the feet. Since that time many agents have been suggested as a prophylactic including whale oil, petrolatum, linseed oil, waterproof leg bags of silk, wax paper, zinc oxide paint, etc. None of these agents are reported to be of any value in retarding or preventing the development of wet-foot injury.

Silicones are a group of polymers composed of silicon, carbon, hydrogen and oxygen. They have a wide range of physical properties depending on the molecular structure of the silicone. Any material to be used for the prevention of immersion foot under combat conditions obviously demands that it have certain desirable qualities such as adhesiveness, water repellency, and ease of application. Since high vacuum silicone grease used in an earlier study does not contain an adhesive, a silicone compound with all three specifications was requested from a manufacturer and subsequently used in prototype studies.

This compound consisted of methylsilicone, fluorosilicone and silica. This preparation was made available in the form of an aerosol, a liquid, and a grease.

The frequency with which these materials were applied, the effect of a specially treated sock, and the possible difference in prophylactic effects between leather and jungle boots were evaluated. The results

of this pilot study revealed that (1) silicone grease applied every 24 hours afforded the most protection; (2) the jungle boot and leather boot difference with regard to the development of immersion foot required further study; (3) silicone treated socks, in addition to local application of silicone grease, may slow the development of immersion foot; (4) symptomatic immersion foot can be created in the majority of subjects exposed to an occlusive, wet environment for 48 hours or more; and (5) clinical (dermatologic) and laboratory (cultures for bacteria and fungi) examinations performed daily gave no indication that harmful results should be expected from the application of these silicone preparations to the skin of the feet. Based on these preliminary observations, a second study was devised. This is a report on that study.

Methods

A total of 61 Marine volunteers were tested; 54 of these finished the test. Physical examinations were done on all volunteers before the test began. These Marines were exposed to a constant wet environment for a total of five days (120 hours) and the area selected for this test was a swamp at Camp Lejeune, NC. All men wore standard issue leather boots or tropical jungle boots and all were given two pairs of socks each. Twenty-eight of the treated subjects had standard issue socks (composed of 50 percent cotton, 20 percent wool and 30 percent nylon) treated with a special silicone liquid preparation, and 24 treated subjects used standard issue nontreated socks (Table 1). Each man put on his second pair of socks 2½ days after the test began. No one was allowed to remove his boots except during the daily examining and treatment period which lasted approximately five to seven minutes. The men's feet were wet at all times. They walked five to ten miles per day and slept in their wet boots and socks. The authors monitored the feet at random times to insure that constant wet occlusion was being maintained. A silicone-grease preparation was thoroughly applied to the soles, between the toes, and up to the level of the malleoli on each subject at the beginning of the test and every 24 hours thereafter. Six volunteers acted as controls and had no preventive treatment of any type. A dermatologist examined all feet daily during the test and at 24, 48, and 96 hours after completion of the test.

Three weeks after the test was completed all available subjects were reexamined. At the end of the test, two dermatologists performed separate examinations on each subject. The examiners had no knowledge of the type of treatment each individual received. Detailed notes were recorded during each examination and color photographs were made of the feet at the beginning and at the end of the test. Appropriate additional follow-up photographs were made when indicated. An immersion-foot casualty was one who could no longer continue marching due to pain and discomfort in his feet. Attempts were made to eliminate subjects who had foot discomfort other than that arising from immersion foot (eg, blisters, poorly fitting boots, etc). Cultures for fungus were made on the first and last test days.

The dry bulb temperatures, relative humidity, and water temperature 18 inches below the water surface were measured at the same time each day. Water temperatures ranged from 70 to 80 F, relative humidity ranged from 57 percent to 60 percent, and dry bulb temperature varied between 77 and 88 F for all five days of the test period.

TABLE 1.—SUMMARY OF RESULTS OF DIFFERENTLY TREATED GROUPS *

Group	Immersion Foot	No Immersion Foot
Silicone-treated sock with leather boot	0	15
Silicone-treated sock with jungle boot	1	12
Regular sock with leather boot	4	9
Regular sock with jungle boot	1	10

*All subjects had silicone grease applied to their feet every 24 hours.

Results

Warm-water-immersion foot is characterized by a painful, white, wrinkled sole. Of the six controls, immersion foot developed in five and of these five, four were classified as casualties after three days and required evacuation. Of the remaining 55 treated subjects, three were unable to finish the test because of conditions not related to immersion foot (ie, plantar warts, blisters, and a foreign body in the eye). In the remaining 52 treated subjects, all completed the test (five days' duration).

Out of a total of 52 treated subjects, mild cases of immersion foot developed in six. There were six Negroes in the study; immersion foot did not develop in any of them.

Mild or early changes of immersion foot developed in one person out of 28 who wore silicone-treated socks and in five out of 24 who wore untreated

socks (Table 1). No subject in whom immersion foot developed had any complaint referable to poor-fitting boots. Supervisory personnel, who were exposed to the same frequency of immersion and marching, removed their wet boots and socks and air-dried their feet each night. They had no subjective or objective changes in their feet. Examination of the 46 treated subjects in whom immersion foot did not develop revealed that 90 percent of them showed mild whiteness and wrinkling by the fifth test day. All treated subjects stated that they could have continued beyond the test termination date.

All subjects had 96-hour follow-up examinations and all available subjects were reexamined three weeks later. No untoward effects were produced by the silicone grease.

Scrapings for fungi cultures were taken between the fourth and fifth digits on each subject's foot at the beginning and end of the test. Of the 52 treated subjects, 19 had positive pathogenic fungus cultures before the test and seven had positive pathogenic cultures at the end of the study (Table 2).

TABLE 2.—NUMBER OF PATHOGENIC FUNGI IN THE TREATED SUBJECTS AT THE BEGINNING AND END OF THE TEST PERIOD

Pathogenic Fungi	Before	After
Candida albicans	8	6
Epidermophyton floccosum	1	0
Trichophyton rubrum	9	1
Trichophyton mentagrophytes	1	0
Total	19	7

It is interesting to note that at the beginning there were 28 positive cultures of nonpathogenic fungi and only eight at the end of the immersion period. The contaminants were *Penicillium, Aspergillus, Trichosporum, Scopulariopsis,* and *Hormodendrum.* In contradistinction, there were 11 positive cultures with bacterial contaminants at the beginning and 19 at the end of the immersion period. The bacterial contaminants were *Aerobacter, Pseudomonas, Escherichia coli,* and *Klebsiella.* Cultures of the swamp water grew the following bacteria: *Aerobacter, Pseudomonas,* and *Klebsiella.*

Comment

The ability to create immersion foot was demonstrated when the condition developed in five of six nontreated subjects. The group treated with silicone grease every 24 hours was remarkably resistant to this condition. Out of a total of 52 treated subjects, immersion foot developed in only six and each was a mild case. The possibility that the silicone grease only retarded the development of immersion foot is suggested by the high frequency of whiteness and wrinkling in the remainder of the group. Even though these subjects had varying degrees of whiteness and wrinkling, they had no subjective complaints.

Daily application of the silicone grease is required because it is removed by the scrubbing, surging action of the water in the boot while moving. This was evident by the appearance of a refractive film assumed to be silicone grease floating on the water when the troops moved through the swamps after application of the material.

There is no difference between the two types of boots (leather vs jungle boot) in their ability to alter the development of immersion foot. The silicone treatment of the socks was apparently ineffectual in modifying the development of immersion foot, although this requires additional studies. The results of the cultures support the authors' clinical impression that the conditions of the subjects with fungus infections at the beginning of the test were unchanged or improved by the end of the test. This supports data reported elsewhere that a silicone grease applied to the foot does not enhance the growth of mycotic organisms on the foot.

Observations on the supervisory personnel have confirmed the perhaps obvious statement that immersion foot will not develop in feet that are allowed to dry overnight. Those formulating field orders and military directives have sometimes gone to great lengths to see that drying facilities are available to the front line troops. During our earlier studies, immersion foot developed in subjects who were allowed to remove their wet boots and socks for 30 minutes, three times a day, and dry their feet just as readily as in other control subjects. The feet must be allowed to dry a long enough period of time (eg, overnight) before such measures will prevent the development of this condition.

Individual variability was striking even between the feet of the same subject. Careful examination of large numbers of subjects is therefore necessary for such a study to have any meaning at all. Efforts to "score" degrees of severity of pain, pinching, and

burning were discarded as these symptoms did not appear reliable.

All subjects showing symptoms of immersion foot were asymptomatic one to three days after removal from the wet environment. The natural history of immersion foot is under preparation (K. A. Gill, MD, and L. J. Buckels, MD, unpublished data).

The authors acknowledge that there are many areas as yet unexplored in the understanding of warm-water-immersion foot, including its prevention. At the present time, this silicone grease applied every 24 hours to the bottom of the feet markedly retards or prevents the development of this condition for up to five days.

(The omitted references may be seen in the original article.)

PERFORATION OF THE TRANSVERSE COLON FOLLOWING EXTERNAL CARDIAC MASSAGE

Sidney Tobias MD, Philadelphia, Arch Surg 94(3):335–336, March 1967.

Closed chest cardiac massage introduced by Kouwenhaven and associates in 1960 has rapidly replaced open chest massage in the management of cardiac arrest. Many hospitals now have trained resuscitation teams on call at all times. These teams respond within minutes to a prearranged signal and arrive at the patient's bedside with the equipment and the personnel necessary to manage such an emergency. As a result, salvage rates for the acute cardiac arrest are continually improving and further efforts in this field should be even more rewarding. In our institution the salvage rate (patients who leave the hospital alive following resuscitation) is now 15% (unpublished data). Closed chest massage, however, is not without complications. Since 1960, occasional reports have appeared describing complications associated with this procedure. It is obvious that many of these complications go unrecognized, either in the patient who dies in whom post-mortem examination is not performed or in the patient who sustains minor complications and is discharged alive without further investigation. It is the purpose of this paper to report a previously unrecognized complication arising as a direct result of blunt force applied to the sternum. In this case a traumatic perforation of the transverse colon was recognized and surgically corrected 16 days following successful closed chest cardiac massage. A review of complications previously reported in the literature is also discussed.

From the Department of Surgery, Hahnemann Medical College and Hospital, Philadelphia.

Report of Case

A 47-year-old white male physician with a five-year history of coronary artery disease and hypertension was sitting in the hospital staff room when he suddenly collapsed. His distress was witnessed by another physician who immediately noticed that peripheral pulses were absent and breathing had stopped. External cardiac massage and mouth-to-mouth resuscitation were begun within seconds of collapse, and in less than three minutes the patient was awake and breathing spontaneously with normal blood pressure and pulse rate. An electrocardiogram showed a sinus tachycardia, evidence of an old posterior wall myocardial infarction, and recent ischemic changes. Chest x-ray film showed no evidence of rib fracture or pneumothorax. The patient was treated for a myocardial infarction. During the first four hospital days he complained bitterly of pain in the chest wall, so that intercostal nerve blocks and narcotics were necessary to control his discomfort. On the 16th hospital day, his temperature suddenly rose to 103 F (39.4C) and he complained of pain in the right upper abdomen. There was a tender mass in the right upper quadrant. He was thought to have acute cholecystitis and treatment was begun with antibiotics, intravenous fluids, and nasogastric suction. His fever and symptoms persisted and an abdominal exploration was performed 24 hours later. There was an abscess in the right upper quadrant arising from a perforation of the transverse colon just distal to the hepatic flexure. *Escherichia coli* was cultured from the abscess. The region of perforation was exteriorized as a

right transverse loop colostomy. The postoperative course was complicated by a small bowel obstruction and on the 13th postoperative day the abdomen was reexplored. He was found to have a mechanical small bowel obstruction secondary to volvulus of the ileum around a fixed Meckel's diverticulum. The postoperative course was complicated by massive bleeding from the colostomy stoma ten days later. This was thought to be bleeding from a stress ulcer and was treated with milk, antacids, and blood replacement. The patient improved rapidly and was discharged eight days later. After a month of convalescence at home, he was readmitted for closure of the colostomy. Barium enema before closure of the colostomy showed no evidence of diverticular disease. Pathological examination of the resected colostomy stoma also revealed no evidence of diverticular disease. The patient is now alive and well.

Comment

Serious complications of cardiac massage have been previously reported in the literature. Clark reported 19 autopsy cases in which he found five cases of fractured liver and two cases of ruptured spleen. In six of these cases there was definite evidence of vascular trauma, either in the chest, abdomen, or retroperitoneum. One of his cases had a hematoma of the right transverse mesocolon. Baringer and associates reported a series of 84 cases of patients who died following cardiac massage. An autopsy was performed on 46 of these. There were rib fractures in 15, 6 had bone marrow emboli, 5 had lacerations of the liver, 4 had hemothorax, and 2 had a hemoperitoneum. It is apparent from these reports, as well as those of others that the most common injuries resulting from closed chest resuscitation are those associated with trauma to the bony thorax, such as fractures of either ribs or sternum or fat emboli. The most frequently reported injuries to intra-abdominal organs are fractures of either the liver or spleen. There have been a few reports of vascular trauma as a result of this procedure, one resulting in a large hematoma of the right transverse mesocolon. The only previously reported case of a ruptured hollow viscus is that of Demos and Poticha in which perforation of the stomach occurred during external massage and was treated successfully. This case, however, was not the result of direct trauma but more likely of the simultaneous application of mouth-to-mouth resuscitation and compression of the sternum. These authors postulated that perforation of the stomach was secondary to overdistention by air forced through the esophagus.

Rupture of a hollow viscus is a common occurrence after blunt trauma to the chest or abdomen. The more commonly involved viscera are those in a relatively fixed position, such as the retroperitoneal duodenum, the jejunum just distal to ligament of Treitz, and the distal ileum in the region of the ileocecal valve. In such cases the mechanism of injury is compression of a distended viscus in a relatively fixed position with resultant rupture from a sudden increase of intraluminal pressure. In our case we postulate that the initial trauma caused edema and interstitial hemorrhage of the bowel wall as well as a hematoma of the mesentery of the transverse colon, which ultimately progressed to necrosis and perforation of the bowel wall. A similar injury was reported in Baringer's case of hematoma of the transverse mesocolon. Another explanation for intestinal infarction was suggested in a case report by Tulgan and Budnitz in which there was a large area of small bowel infarction secondary to an embolus in the superior mesenteric artery. This embolus was thought to originate from a mural thombus forced into the systemic circulation by closed chest compression. This does not seem to have been the mechanism of injury in our patient. Careful inspection of the colon in our patient at the time of surgery and postoperatively by barium enema failed to reveal any evidence of diverticular disease, thus excluding the possibility of a perforated diverticulum.

Summary

A case of perforation of the transverse colon secondary to external cardiac massage is reported. The mechanism of this injury is discussed. A brief review of the complications arising from this procedure is presented.

E. Dallet Sharpless, MD, and John H. Davie, MD, attended this patient and permitted the use of this material for publication.

(The references may be seen in the original article.)

ACCELERATION OF HEALING WITH ZINC SULFATE

MAJ Walter J. Pories MC USAF, CAPT John H. Henzel MC USAF, Charles G. Rob MD, and William H. Strain PhD. From the Department of Surgery, USAF Hospital Wright-Patterson, Wright-Patterson AFB, Ohio and the Departments of Surgery and Radiology, School of Medicine and Dentistry, University of Rochester, Rochester, New York. *Ann Surg 165(3): 432–436, March 1967.*

Zinc is a potent catalyst of wound healing and zinc deficiency may be a common cause of delayed tissue repair. It is curious that this element has been ignored for so long in studies of human nutrition. Physiologically, zinc is similar to iron in many respects and is present in nearly the same concentrations. It is more plentiful in the human body than other metals such as cobalt, iodine and fluorine and is intimately involved with such vital processes as protein synthesis and cellular respiration. To a great extent, knowledge has been limited by difficulties in analysis for only recently with the advent of sophisticated chemical technics, could careful studies of zinc metabolism be made. As these investigations have progressed in the last few years, zinc has been identified as an important factor in repair of tissue.

The use of zinc to promote healing is not new. The ancient Egyptians used it topically in the form of calamine and since then zinc oxide, zinc sulfate and zinc stearates in the form of powders, salves and ointments have continued to be used. It is likely that some of the element is absorbed through the skin, particularly through injured and granulating tissues.

Our interest in the systemic use of zinc began with a laboratory accident. During a series of wound healing studies in rats, a remarkable acceleration of healing was noted in a group fed a contaminated diet. After considerable searching, zinc was finally identified as the beneficial impurity and further experiments in rats confirmed that zinc was a potent factor in healing. Subsequent observations in patients suggested that zinc also played a role in human healing. The purpose of this report is to present the results of a carefully controlled study which suggests that dietary zinc supplements can accelerate the rate of healing in man.

The contents of the paper reflect the authors' personal views and are not to be construed as a statement of official Air Force policy.

Materials and Methods

Extension of zinc therapy from animal studies was a problem until it was found that the wound formed by excising pilonidal sinuses in young airmen was very satisfactory for healing studies. These wounds should reflect the behavior of normal granulating tissues as the excisions were carried into normal tissues on all sides and as the lesions were left open to heal by second intention. The excision sites were dressed daily with Balsam of Peru and the volumes were measured by taking impressions of each cavity at approximately 5-day intervals with a rapidly setting, innocuous alginate hydrocolloid, Jeltrate, as previously described, and as shown in Figure 1 (omitted). After 25 days, many volumes were less than 1 ml. and accurate measurement of volume by this method was no longer possible.

The second method of quantifying the rate of wound healing was the determination of the number of days required for complete healing. Healing was considered complete when the entire surface was covered with epithelium and no further scab formation took place which proved to be a remarkably clear end point.

Twenty patients were studied. These airmen were young and healthy and continued their usual diets during the study. They were randomly divided into treatment groups on the basis of serial numbers, with odd numbers assigned to control and even numbers to zinc therapy. Ten patients became controls and were maintained on their usual diets *ad lib* without medication during the period of healing. The remaining ten formed the therapy group and were given daily dosages of zinc sulfate in addition to their usual diets *ad lib*. The medicated patients were supplemented with capsules containing 220 mg. of zinc sulfate USP ($ZnSO_4 \cdot 7H_2O$) at a dosage of one cap-

TABLE 1.—WOUND HEALING IN CONTROLS AND AIRMEN MEDICATED WITH ZINC SULFATE USP

Patient	Age	Wound Volume (ml.)	Days for Complete Healing	Healing Rate (ml./day)
		Controls		
1	27	10	40	0.25
2	21	2.5	46	0.05
3	40	40	48	0.83
4	23	47	53	0.89
5	23	28	63	0.44
6	24	52	71	0.73
7	23	42	85	0.50
8	24	7.5	93	0.08
9	23	45	121	0.37
10	22	49	181	0.27
MEAN ± SE	25.0	32.3	80.1 ± 13.7	0.44 ± 0.09
		Zinc Sulfate USP, 220 mg. t.i.d.		
1	19	100	33	3.03
2	24	28	34	0.82
3	22	30	43	0.70
4	18	44	44	1.00
5	28	35	46	0.76
6	40	140	46	3.04
7	28	30	48	0.63
8	23	50	51	0.98
9	22	49	52	0.94
10	22	39	61	0.64
MEAN ± SE	24.6	54.5	45.8 ± 2.6	1.25 ± 0.30
Significance			$p < 0.02$	$p < 0.01$

sule three times a day in order to supply the equivalent of an additional 150 mg. of elemental zinc per day.* This dosage was well tolerated by all patients. The capsules were taken either with milk or after a meal to avoid possible gastro-intestinal irritation by zinc sulfate; the salt has an emetic dose of 2 Gm. All patients were maintained on regular house diets until three days after operation, when they were returned to full duty and resumed their usual dietary habits. All patients healed without difficulty and, to date, there have been no recurrences.

Results

The significant acceleration of wound healing produced by zinc therapy is emphasized by the data in mean healing values summarized in Table 1. In the control group, wounds healed in 80.1 + SE 13.7 days.† In the zinc-medicated patients, wounds healed in 45.8 + SE 2.6 days ($\rho < 0.02$). Thus wounds of patients receiving 150 mg. of metallic zinc daily were healed 34.3 days earlier than the controls even though their initial wounds were almost twice as large (54.5 ml. vs. 32.3 ml.). Acceleration is also shown in the rate of wound closure. The control wounds closed at the rate of 0.44 ml./day, whereas those of patients medicated with 150 mg. of zinc closed at the rate of 1.25 ml./day, or almost three times faster ($\rho < 0.01$).

There was a considerable difference in the appearance of the wounds between the two groups. The wounds of the airmen on zinc sulfate therapy demonstrated cleaner, pinker and *healthier* granulations with considerably less purulent exudate.

Although the medicated group healed more rapidly throughout the course of the experiment, the differences were small during the first 15 days. Acceleration of epithelization in the terminal stages of healing is similar to the pattern observed in our earlier animal studies.

Discussion

It is not surprising that zinc should play an important role in human wound healing. Considerable experimental evidence has been available that this

* Supplied by Smith, Kline & French Laboratories, Philadelphia, Pa.

† Standard error.

element is essential for wound repair. Zinc-deficient cattle, poultry, sheep and swine spontaneously develop ulcerating lesions, especially of their legs, which heal only if adequate dietary zinc is supplied. Miller *et al.,* noted the dependency of healing on adequate zinc stores, and suggested that poor healing in cattle can be used as a sign of zinc deficiency.

Zinc probably acts primarily at the wound site where it is incorporated into enzyme systems. Savlov *et al.* demonstrated that radiozinc is preferentially concentrated in healing tissues with a peak of activity during the first 7 days after injury, followed by a gradual decrease. The migration of zinc into the wound appears to be temporary since at the time of complete scarring, 100 days after injury, these investigators found no evidence of zinc-65 in the scar although the radioisotope was still detectable in most tissues.

Zinc is the metal moiety in a number of essential enzyme systems. Metalloenzymes known to contain zinc include alkaline phosphatase, carbonic anhydrase, carboxypeptidase and the following dehydrogenases: alcohol, glutamic acid and lactic acid. In addition, zinc acts as a cofactor to a number of enzymes including arginase, carnosinase, dehydropeptidase, glycyglycine dipeptidase, histidine deaminase and tripeptidase, as well as oxaloacetic carboxylase and some of the lecithinases and enolases. Evidence accumulated from studies in microorganisms also supports the thesis that the action of zinc is in the area of protein and nucleic acid synthesis. Zinc deficiency in *Mycobacterium smegmatis,* for example, interferes with the synthesis of RNA and thus secondarily inhibits the synthesis of DNA and other proteins. Whether similar pathways are affected in humans remains to be shown.

It is desirable to offer readily available zinc to the organism during periods of wound healing as zinc appears to be essential to protein synthesis. Studies on rats first demonstrated that such an approach was sound. The addition of extra zinc to a commercial rat feed accelerated the healing rate of wounds and burns in these animals by 30 percent. The present studies on young airmen on normal diets suggest that zinc salts are also beneficial during tissue repair in humans. The healing of standard granulating wounds was accelerated by 34.3 days, or 43 percent, over that in controls (80.1 + SE 13.7 days vs. 45.8 + SE 2.6 days). Most of this acceleration occurred during the stage of epithelization. Perhaps this is related to the fact that 20 percent of the body's zinc stores is in the skin, concentrated in the epithelium and epithelial structures.

It is becoming apparent that zinc deficiency may be a common problem in our patient population. Studies in 47 patients with severe burns have shown a marked zinc deficit beginning shortly after the burn and lasting as long as 2 to 3 months after injury. More recent observations in postoperative patients have shown a marked zincuria with a fall in zinc stores during the early postoperative period.

Zinc is obtained only in a limited number of foods and in these the zinc content is often variable; the element is primarily absorbed and excreted by the small intestine. In the chronically malnourished patient such as the aged, the obese, the faddist or those with serious debilitating illnesses severe zinc deficits due to the negative zinc balance can and probably do easily develop. This may account for wound dehiscences, poor healing and frequent hypoproteinemia so often seen in this group. Since zinc medication was so beneficial in young, healthy airmen with relatively minor wounds, it is likely to be even more so in seriously ill patients with severe nutritional problems. Further studies of these groups of patients are now underway.

Fortunately, zinc sulfate is an extremely safe compound for oral use and toxicity levels are about 30 times the dosages used in these studies. It is well tolerated and offers a safe and effective method of promoting healing, especially in the marginally nourished patient with deficient zinc stores.

Summary

Zinc is intimately involved in healing. Zinc-deficient animals heal poorly, zinc-65 localizes preferentially in healing tissues and the addition of extra readily available zinc salts accelerates healing in rats.

Zinc also appears to play an important role in human healing. Patients with severe burns develop significant zinc deficits and it is likely that patients with other healing problems also have zinc stores inadequate to meet the demands of healing tissues.

Oral medication with 220 mg. of zinc sulfate USP three times a day, resulted in the acceleration of healing by 43 percent in a well controlled study of young men with granulating wounds. Zinc therapy deserves a trial as an adjunct in the treatment of patients with healing problems.

(The omitted figures and references may be seen in the original article.)

ACUTE MELIOIDOSIS IN A SOLDIER HOME FROM SOUTH VIETNAM

CAPT Matthew C. Patterson USAF (MC), MAJ Charles L. Darling USAF (BSC), CAPT Jerome B. Blumenthal USAF (MC), JAMA 200(6):447-451, May 8, 1967.

A fatal case of acute melioidosis occurred in a soldier who had recently returned from Vietnam. *Pseudomonas pseudomallei* was identified by culture of blood specimens taken before and after death, animal inoculation, and fluorescent antibody tests. Chloramphenicol is the drug of choice in this disease.

It is imperative that military and civilian physicians be aware of the exotic infections which may confront our servicemen in Southeast Asia. The rapid transportation of military personnel to our shores literally brings the exotic diseases of the East to our doorstep. Melioidosis is one such disease. This paper reports this infection in a soldier who recently returned home from South Vietnam.

Report of a Case

This 22-year-old white man (Register No. 166 349) had a transient febrile illness of obscure etiology four months before his death but otherwise had been in good health. He had been stationed in South Vietnam since January 1966 (with the exception of temporary leave to the United States in June 1966), but was sent home on Oct 6, 1966, for emergency leave. Soon after he arrived, severe bilateral frontal headache, anorexia, nausea, prominent sore throat, dysphagia, and generalized myalgia developed. He was seen in the emergency room of a civilian hospital and given what was probably penicillin, orally and intramuscularly, without relief.

On Oct 12, 1966, he was admitted to a civilian hospital. Physical examination revealed a temperature of 105 F (40.6 C), flushed skin, a striking pharyngitis with tonsillar exudate, marked anterior cervical lymphadenopathy, clear lungs on auscultation, right and left upper abdominal tenderness, and a staggering gait thought to be due to weakness.

From the departments of pathology (CAPT Patterson and MAJ Darling) and medicine (CAPT Blumenthal), US Air Force Hospital Wright-Patterson, Wright-Patterson Air Force Base, Ohio.

The hemoglobin level was 13 gm/100 ml and the white blood cell count (WBC) was 8,800/cu mm, with a normal differential. Prothrombin time was 75 percent of normal. Repeated smears for malaria were negative, throat culture grew β-hemolytic streptococcus, and results of urinalysis were normal. An antistreptolysin O titer was 250 Todd units. Febrile agglutinin titers were 1:40 to 1:80 to the typhoid and paratyphoid antigens. Skin test with purified protein derivative tuberculin was negative. Tularemia agglutination titer was 1:20. Stool cultures were negative. Sulfobromophthalein (BSP) retention was 22 percent. Numerous blood cultures were negative. A chest film revealed accentuated hilar markings with perihilar streaking, suggesting to the radiologist a viral pneumonia.

The patient was treated with 6 million units procaine penicillin daily for six days, 5 gm chloramphenicol (Chloromycetin) daily for three days (discontinued after WBC decreased to 5,000/cu mm), and 2 gm sodium methicillin daily for four days. His hospital course was characterized by spiking temperature to 105 F and rapidly progressing weakness. The patient's hematocrit value dropped to 34 percent during his admission.

On Oct 22, 1966, he was transferred to US Air Force Hospital Wright-Patterson for evaluation of fever of obscure etiology. On admission to this hospital, the only additional history obtained was the fact that he had stepped on a punji stick (sharp lance covered by fecal material) one month before his arrival in this country; he apparently had had no subsequent difficulty.

At physical examination, the patient appeared acutely and chronically ill and had a temperature of 105 F. Pharyngeal mucous membranes were pale, but there was no evidence of tonsillitis. No lymphadenopathy was noted. A pleural friction rub was heard on the right. The liver was felt three fingerbreadths below the right costal margin but was not tender.

The spleen could not be felt, but enlargement was noted by percussion. There was no jaundice. The hemoglobin level was 11 gm/100 ml; hematocrit, 35 percent; and WBC, 10,300/cu mm, with a shift in the differential to the left. The reticulocyte count was 1.8 percent. The serum bilirubin level was 2.2 mg/100 ml with 1 mg direct. Thymol turbidity was 8.2 units and cephalin flocculation was 3+ at 24 hours. Serum glutamic oxaloacetic and pyruvic transaminase (SGOT and SGPT) levels were 52 and 41 units/100 ml, respectively. The prothrombin time was 70 percent of the control. The heterophil titer was 1:7, and the cold agglutinin titer was 1:4. Multiple malarial preparations during fever spikes were negative. Febrile agglutination tests revealed no titer to typhoid O and a 1:20 titer to typhoid H antigen. The test for *Proteus* OX–19 antibodies was negative. Tests for brucella, tularemia, and paratyphoids A and B antibodies were negative. Chest films revealed small, nodular infiltrates in both lung fields. Liver scan was negative. Numerous blood and urine cultures were negative during the patient's life. The patient was unable to produce sputum for culture. Bone marrow aspirate was normal on culture and microscopy. Results of lumbar puncture were normal.

The patient spiked daily temperatures to 105 F. He was given tetracycline for 12 hours following admission but this was discontinued so that a diagnosis could be established by culture prior to the institution of antibiotic therapy. On the third hospital day, gram-negative rods were identified in a buffy-coat smear but none could be found on subsequent attempts. On the fourth hospital day, the patient had a slight fall in blood pressure accompanying a temperature spike and was, at that time, placed on a daily regimen of 12 gm of sodium cephalothin given intravenously, 2 mg of streptomycin, given intramuscularly, and 225 mg of sodium colistimethate given intramuscularly. Eighteen hours after initiation of this therapy, the patient experienced severe dyspnea. Chest films showed striking progression of the nodular infiltrates, and the patient was immediately given isoniazid intramuscularly (INH) and steroids intravenously in high dosage. Shortly thereafter he experienced respiratory arrest. Vigorous and prolonged resuscitation was unsuccessful, and the patient was pronounced dead approximately three weeks after the onset of illness.

At autopsy (HPA–82–66) the body was that of a well-developed and well-nourished man. The skin was unremarkable. No lymph nodes were prominent externally. Five hundred milliliters of serous ascitic fluid were found. There was no obvious peritonitis. Each pleural cavity contained 500 ml of serosanguineous fluid and there were foci of fibrinous pleuritis. The superior surface of the left leaf of the diaphragm was studded with 1–3 mm, yellowish-white, slightly raised lesions. Each lung weighed 1,250 gm, and numerous yellowish-white, discrete lesions measuring 0.1 to 2 cm could be seen beneath the pleurae. The pulmonary parenchyma was a deep bluish-red color and contained a marked amount of bloody fluid. Throughout all lobes there were slightly raised, fairly well circumscribed, yellowish-white areas measuring from 1 mm to 4 cm in diameter. These areas were predominately firm but several contained central cavities filled with creamy, yellowish-green material. There was no associated caseous necrosis, although a single lymph node at the right hilum contained caseous necrosis with calcification. The liver was enlarged and weighed 2,100 gm. Scattered throughout the liver in a random fashion were fairly discrete, yellowish-white, soft lesions measuring up to 4 cm in diameter. The largest of these showed extensive creamy, central necrosis, and many of these necrotic areas were surrounded by a thin rim of fibrous tissue. The intervening liver parenchyma was grossly normal in appearance. The spleen was enlarged and weighed 480 gm. There were small foci of fibrinous peritonitis over the capsule. On gross section there were numerous 1-mm, yellowish-white lesions slightly larger than malpighian corpuscles. A small accessory spleen was identified and it, too, contained similar lesions. Grossly, the heart, pancreas, adrenal glands, kidneys, prostate, urinary bladder, gallbladder, gastrointestinal tract, lymph nodes, thyroid, parathyroids, bone marrow and brain were unremarkable.

Histologically, foci of extensive cellular destruction with microabscess formation were noted within the lungs, liver, spleen, accessory spleen, bone marrow, mediastinal lymph nodes, right adrenal gland, and left leaf of the diaphragm. The inflammatory cellular reaction included polymorphonuclear leukocytes, lymphocytes, and macrophages. Within the lungs there was extensive hemorrhage associated with the inflammation. The striking histologic feature of the lesions, however, was marked karyorrhexis. None of the microabscesses contained granulomatous inflammation. However, there was cellular organization

of exudate with fibroblastic proliferation surrounding large abscesses in the lungs and liver. Multiple tissue sections stained by the Brown-Brenn method revealed rare gram-negative rods. Methenamine silver, PAS, and acid-fast stains were negative for organisms. Touch impressions from the lung and liver lesions revealed pleomorphic gram-negative bacilli which demonstrated marked bipolar staining with methylene blue. Acid-fast stains were negative. The lymph node from the right hilum described above contained fibrocaseous necrosis and numerous acid-fast bacilli. (We feel this represents a single focus of tuberculosis quite unrelated to the disease under discussion.)

Three of 11 blood specimens drawn while the patient was alive grew *Pseudomonas pseudomallei* seven days after his death. These three samples had been taken less than 48 hours prior to death. The administered antibiotics may well have been responsible for this retardation of growth. At autopsy, *P pseudomallei* was isolated from both lungs and liver. The organism was a moderately slow grower on sheep blood agar when incubated at 37 C under carbon dioxide tension. At 24 hours the colonies were white to light-cream in color and were surrounded by greenish discoloration (α-hemolysis). After 48 hours' incubation there was complete hemolysis around confluent colonies but not around well-isolated colonies. In three to four days all of the colonies became quite wrinkled. There was a definite ammonia odor. The organism grew fairly well on eosinmethylene blue agar, quite slowly on MacConkey agar, and not at all on salmonella-shigella agar. Good growth was obtained on triple sugar iron agar, but no acid, gas, or hydrogen sulfide was produced. Nitrogen gas was produced on Seller's medium, but growth on the surface of the slant did not fluoresce with ultraviolet light. Citrate utilization was delayed until the third day. On SIM medium the organism was motile, and indole was not produced. The urease test was negative. The catalase test was weakly positive. The oxidase test was interpreted as positive, even though the colonies took on only a light pink color. The color faded completely within 15 minutes. A salient feature was the production of a wrinkled pellicle in thioglycollate broth after incubation at 37 C for 24 hours. This phenomenon did not occur in brain heart infusion broth.

Disk sensitivity tests on Mueller Hinton agar indicated susceptibility to chloramphenicol, tetracycline, and sulfisoxazole and resistance to cephalothin, penicillin, lincomycin, erythromycin, streptomycin, and colistimethate.

The identification of *P pseudomallei* was confirmed at the Communicable Disease Center by culture and fluorescent antibody tests.

Postmortem culture material was used for animal inoculation. One milliliter of undiluted, 48-hour-old brain heart infusion broth culture was injected intraperitoneally into two male guinea pigs. The control animal received sterile broth. The smaller of the two experimental animals (290 gm) died in approximately 12 hours. Postmortem examination revealed microabscesses within the omentum, spleen, and liver, with focal acute peritonitis. *Pseudomonas pseudomallei* was recovered from peritoneal fluid, liver, and heart blood. The larger experimental animal (440 gm) survived 3½ days. At postmortem examination, *P pseudomallei* was recovered from heart blood and peritoneum; microscopy demonstrated abscesses within the spleen, liver, omentum, peritoneum, and tunica vaginalis. The lesions in both experimental animals reproduced those seen in the case under discussion. The control animal was culturally sterile and no pathologic lesions were found.

The final anatomic diagnosis was melioidosis with bilateral, severe, widespread necrotizing bronchopneumonia with abscess formation and dissemination to liver, spleen, accessory spleen, bone marrow, thoracic lymph nodes, pleura of the left leaf of the diaphragm, and right adrenal, with multiple abscess formation. (Pleomorphic, bipolar-staining, gram-negative bacilli were demonstrated on smear and *P pseudomallei* grew on culture.)

Comment

Melioidosis is a fulminating and usually lethal disease first described by Whitmore and Krishnaswami in 1912. It was at first called Whitmore's disease, but Stanton and Fletcher chose the name "melioidosis," meaning "a resemblance to a distemper of asses," in 1921. Melioidosis bears a striking resemblance to glanders etiologically, pathologically and clinically, but the two can be differentiated by bacteriologic and serologic methods. They are also epidemiologically dissimilar, for glanders is principally a disease of horses whereas melioidosis is found in rodents. Melioidosis is caused by the gram-negative bacillus *P pseudomallei* (until recently designated *Malleomyces pseudomallei*), isolated and described in 1913 by Whitmore.

Most of the 300 cases reported in the world's literature have occurred in Thailand, Malaya, Vietnam, Burma, and Ceylon. Cases have been reported sporadically from Guam, South Africa, Panama, Ecuador, United States, Philippines, Great Britain, and Australia. Five previous reports describing a total of six cases of melioidosis occurring in US military personnel stationed in Southeast Asia have appeared in the literature. Only one of these, was in a soldier involved in the Vietnam conflict; the others occurred from 1945 to 1957 in Burma, Guam, and the Philippines. A total of six cases of melioidosis acquired by natural means in the Western Hemisphere have been reported. Biegeleisen and co-workers believe only three cases proven definitely by culture can be regarded as legitimate. In two of these cases, the disease was contracted in Panama and in the third in Ecuador.

In areas of Southeast Asia, melioidosis is found as a natural infection among rodents (principally rats). Human infection is thought to occur mainly through consumption of foodstuffs contaminated by rat excreta. Whitmore consistently obtained a fatal bacteremia with typical lung lesions in guinea pigs which were fed food or water contaminated with *P pseudomallei.* More rarely it is found in drug addicts and is probably inoculated subcutaneously. The apparent immunity of laboratory workers in contact with *P pseudomallei* cultures and of attendants handling infected laboratory animals suggests that man is not readily susceptible. Man-to-man infection has never been reported although patients with severe pulmonary changes and discharging abscesses have been nursed in general wards.

Melioidosis may present as either an acute or chronic disease. In the most acute form (90 percent of reported cases) illness begins suddenly, after an incubation period of 10 to 14 days, with shaking chills, high fever, and marked prostration. The primary clinical manifestation is usually pulmonary. Bacteremia occurs early and there is widespread hematogenous dissemination of organisms throughout the body. Subsequent abscess formation takes place, involving principally the lungs, liver, spleen, bone marrow, lymph nodes, skin, and subcutaneous tissue; rarely is there involvement of kidney, testes, or prostate, and there is no report of brain abscess. The patient may die within three or four days, or the course of the disease may enter a subacute phase with prolongation of life for three to four weeks. In this instance the disseminated lesions may become evident clinically. In rare instances, melioidosis becomes chronic or may actually present initially as a chronic disease. Lesions of skin, subcutaneous tissue, skeletal muscle, and bone predominate, and draining sinuses may persist months or even years. The chronic form of the disease may be more common in Occidentals than Orientals.

Although there was no evidence of diabetes mellitus in our case, Rimington described diabetes mellitus in five of six patients with melioidosis (five cases of acute disease and one of chronic), and Crotty et al described diabetes mellitus in one of their two cases.

Although melioidosis is often described as a granulomatous disease, the lesions are not granulomatous in the classical sense. It is true that epithelioid-like cells are seen at the periphery of many of the older lesions. This merely represents the fibroblastic attempt at organization that is often seen at the periphery of many of the older lesions. This merely represents the fibroblastic attempt at organization that is often seen at the periphery of abscesses, whatever the cause. The striking feature of the lesion in acute and subacute cases is the marked tissue necrosis with cellular karyorrhexis. Certainly, this represents an anatomical reflection of the virulence of this organism. We must emphasize the previously described difficulty in staining organisms in tissue section regardless of method used. Gram-stained touch preparations of lesions need to be done to demonstrate the organism.

Clinically, the disease may be quite similar to disseminated fungus infection, tuberculosis, plague, typhoid, amebic hepatitis, viral pneumonia, or other infections by gram-negative organisms. There is nothing diagnostic about the chest films.

Definitive culture of the organism, animal inoculation, and fluorescent antibody tests are the only means available of making a positive diagnosis in sufficient time so that vigorous and appropriate antibiotic therapy can be instituted. Serological tests are of little value in the acute form because the patient usually dies before the antibody titer is appreciably raised. Recently, Nigg has reported a comprehensive study of the serology of melioidosis. Intraperitoneal inoculation of male guinea pigs with 0.5 ml of undiluted broth culture 24 to 48 hours old produces a fulminating illness with fever, shock, and death usually in four or five days although an

occasional animal will survive eight or nine days. Postmortem examination of these animals reveals multiple abscesses throughout the body and a purulent peritonitis with extension into the scrotal sac and production of an acute orchitis (Straus reaction). Organisms are easily recovered from heart blood, peritoneal exudate, and tissue. The Straus reaction is not specific for melioidosis as it does occur after inoculation with several other bacteria. Fluorescent antibody tests afford a rapid and accurate means of diagnosis.

Melioidosis is an extremely virulent infection with approximately 90 percent fatality in untreated cases. However, in patients treated aggressively with a combination of antibiotic cure can be effected. Sulfonamides, tetracycline, streptomycin, and chloramphenicol appear to be most effective, with chloramphenicol the drug of choice. Khaira et al report cures in three cases of acute melioidosis with several of these agents. A combination of surgery and antibiotic therapy is often necessary in chronic melioidosis involving soft tissues and bone.

Robert Weaver, MD, PhD, Communicable Disease Center, Atlanta, confirmed the identification of the organism by culture and fluorescent antibody studies. John Ford, Miami Valley Hospital, Dayton, Ohio, did the photomicroscopy.

Generic and Trade Names of Drugs

Tetracycline—*Achromycin, Panmycin, Polycycline, Tetracyn.*
Chloramphenicol—*Chloromycetin.*
Sodium methicillin—*Dimocillin-RT, Staphcillin.*
Sodium cephalothin—*Keflin.*
Sodium colistimethate—*Coly-Mycin Injectable.*
Isoniazid—*INH, Niconyl, Nydrazid, Tyvid.*
Lincomycin—*Lincocin.*
Sulfisoxazole—*Gantrisin.*

(The omitted figures and references may be seen in the original article.)

MEDICAL ABSTRACTS

MELANOMA AS A MEDICAL PROBLEM

Larry Nathanson MD, T. C. Hall MD, G. F. Vawter MD, and Sidney Farber MD, (From the Tumor Therapy Clinic of the Children's Cancer Research Foundation, the departments of Medicine and Pathology of the Children's Hospital Medical Center, the Peter Brent Brigham Hospital, and the Harvard Medical School, Boston.) Arch Intern Med 119:479–492, May 1967.

This is another article which, unfortunately, is too long to be reproduced in the U.S. Navy Medical News Letter and is too filled with information about its subject for adequate abstracting. One hundred sixty-five patients with malignant melanoma were reviewed and the authors' summary and conclusions are as follows: "Actinic radiation, possible genetic factors, as well as premalignant pigmented lesions appeared to have a role in the initiation of the disease. Sexual immaturity was associated with a lower incidence of the disease without greatly altering survival. The reverse was true for postmenopausal women who had a poor prognosis. Doubling time of pulmonary metastasis was seven weeks. Delay in diagnosis beyond 12 months was associated with superior survival.

"When added to standard surgical therapy, adjuvant chemotherapy appeared both to delay onset of recurrence and to prolong survival. The best rate and duration of chemotherapeutic response was seen in a small group of patients who underwent perfusion. New drugs may provide chemotherapeutic responses in the future. The changes in tumor histology following chemotherapy, together with preliminary experience with a new therapeutic technique, laser therapy, are briefly reviewed."

However, it includes a great deal of information in addition to that in the summary and conclusions—statistical data; discussions of genetic factors; survival as related to age and sex of patients, location of primary lesion, stage of the disease, delay in diagnosis, pregnancy, and date of presentation; patterns of dissemination including clinical patterns, rate of growth, immunological factors; and medical therapy including chemotherapy and X-ray therapy, surgical adjuvant chemotherapy, histologic changes following chemotherapy and future prospects for medical therapy.

EXPLORATORY GASTROTOMY IN MANAGEMENT OF MASSIVE UPPER GASTROINTESTINAL HEMORRHAGE

R. J. Freeark MD, W. J. Norcross MD, and R. J. Baker MD, (From the Departments of Surgery, Cook County Hospital, Northwestern University Medical School, the University of Illinois at the Medical Center, and the Hektoen Institute of Medical Research of Cook County Hospital, Chicago, Illinois.) Arch Surg 94:684–695, May 1967.

This is a report of 77 patients who required surgery for the control of massive upper gastrointestinal bleeding of undetermined origin. Exploratory gastrotomy was done in each instance. A bleeding site was accurately identified in seventy-four. The patients were divided into two groups depending on the presence or absence of an abnormality noted at the time of laparotomy and before gastrotomy. Group number one was made up of 47 patients without any findings at the time of laparotomy to suggest the site of bleeding and in whom exploration of the widely opened stomach was carried out in an effort to establish the cause. In this group, gastric ulcer was found in 15, Mallory-Weiss syndrome in 11, duodenal ulcer in 8, diffuse mucosal hemorrhage in 5, ruptured sclerotic vessel in 3, duodenal reduplication in 1, pseudoxanthoma elasticum in 1, and in 3 the source of bleeding was not identified. Group two consisted of 30 patients in whom some abnormality was noted prior to gastrotomy but the surgeon elected to do a gastrotomy because of the uncertainty concerning the relationship of the abnormality to the cause of bleeding. Cirrhosis of the liver was found in 12, subserosal hemorrhage (Mallory-Weiss syndrome) in 5, a perigastric mass in 7 (pancreatic pseudocyst) in 4, chronic pancreatitis in 1, bleeding from the gastric mucosa overlying a neoplasm (leiomyosarcoma stomach (?)) in 1, and leiomyoma of the stomach in 1, splenomegaly in 3, and in 3, bleeding occurred one to ten years following partial gastrectomy. A peptic ulcer was found in the vicinity of anastomosis in two of the latter and in the other there was massive bleeding from the entire gastric mucosa but no evidence of discrete ulceration. The authors state that a significant number of the bleeding sources identified could not have been controlled by empiric resection of the distal two-thirds of the stomach. To prevent complications, which were frequent in the early cases, they describe means of minimizing the hazards of long gastrotomy which evolved with continuing experience.

CHOLESTEROL EMBOLIZATION—FROM PATHOLOGICAL CURIOSITY TO CLINICAL ENTITY

Miriam Moldveen-Geronimus MD and J. C. Merriam Jr. MD, (From the Department of Pathology, Veterans Administration Hospital, Boston, Massachusetts.) Circulation 35:946–953, May 1967.

Two cases of cholesterol embolization are described in this article and the authors refer to 121 cases which they found in the English language. Their patients had multiple system disease due to atheromatous embolization, with increasingly severe hypertension due to renal involvement. In addition, one had acute pancreatitis from embolization to the pancreas, and one had mottling of the skin of the legs with purple toes and gangrene in three toes. The authors discuss the "purple toe" syndrome at some length, particularly its development in connection with anticoagulant therapy. They hypothesize that anticoagulant therapy may favor the dissemination of atheromatous fragments, possibly by preventing adequate thrombosis over atheromatous lesions of the aorta, and aggravate if not actually initiate cholesterol embolization. The peripheral manifestations because they may be concomitant with or even precede life-threatening systemic embolization, they feel, are a signal for the clinician to reappraise his treatment.

(See the New England Journal of Medicine 276: 1368–1377, 15 June 1967—Case Records of the Massachusetts General Hospital, Case 25–1967—Editor.)

AN ANALYSIS OF FEVERS OF UNKNOWN ORIGIN IN AMERICAN SOLDIERS IN VIETNAM

J. J. Deller, Jr., LT COL MC USA and P. K. Russell MAJ MC USA, (From the 93rd Evacuation Hospital, Long Binh, South Vietnam.) Ann Intern Med 66:1129–1143, June 1967.

One hundred ten cases of "fevers of unknown origin" presented to a U.S. Military Hospital in South Vietnam over a four month period were studied by serologic, virologic, and bacteriologic methods. This study was designed, the authors state, to unravel the diagnostic puzzle of the febrile diseases being seen in the American troops supported by the 93rd Evacua-

tion Hospital. They were able to make etiologic diagnoses in 74 percent of the large number of tropical diseases endemic in the area characterized by high fever, chills, and headache. The majority fell into three groups: the arborvirus diseases, (dengue and chikungunya), scrub typhus, and falciparum malaria. Other diseases included melioidosis, leptospirosis, amebiasis, drug sensitivity, gonococcal sepsis, Japanese encephalitis, salpingitis, pneumonia, prostatitis, pericarditis, shigellosis, and nonspecific diarrhea. Etiologic diagnoses could not be established in 29 cases. These fell generally into two groups. Half had a significant febrile course persisting for more than 72 hours in 13 and for as long as seven days in some. The patients in this group could not be distinguished clinically from those with scrub typhus and dengue. The other half had fever and variable symptoms less than 72 hours the causes of which appeared to be a heterogenous group of simple febrile illnesses.

The authors conclude: "The high incidence of the arborvirus diseases found in this study suggests the need for a more precise epidemiologic survey that might lead to a consideration of future immunization against these diseases for troops entering the area.

"The prevalence of malaria and scrub typhus and their inclusion in a study of "fevers of unknown origin" indicate a continuing need for awareness of the clinical spectrum of their manifestations and emphasis on prophylactic measures.

"The potential hazard of diseases such as melioidosis and leptospirosis, which represented a small segment of the diagnostic problems in this series, may well cause them to become major medical problems in areas of Vietnam not surveyed by this study."

DIAGNOSTIC SIGNIFICANCE OF LYMPHOCYTES IN PLEURAL EFFUSIONS

Lung T. Yam MD, (From the Blood Research Laboratory, New England Medical Center Hospitals, and the Department of Medicine, Tufts University School of Medicine, Boston, Massachusetts.) Ann Intern Med 66:972–982, May 1967.

The author describes a clinical, cytologic, and histologic study of a group of patients with pleural effusions in which he found various cellular patterns. The degree of lymphocytosis, he feels, was very informative, often offering a clue to or leading to the specific diagnosis.

The following diseases associated with lymphocytosis of the pleural fluid are discussed: tuberculosis, lymphoma, carcinoma, cardiopulmonary diseases, pyogenic infections, others. In addition, typical cytologic findings in transudates are described. In his summary, the author states: "Two hundred twenty effusions from 159 patients were examined cytologically; 123 of them from 85 patients were predominately lymphocytic (50 percent or more of the cells being lymphocytes). Seventy-three percent of the lymphocytic effusions were found in cases of tuberculosis, lymphoma, and carcinoma; 15 percent were found in cardiopulmonary diseases. Occasionally they were also found in pulmonary infections in recovery stage, cirrhosis, systemic lupus erythematosus, infectious mononucleosis, and in a few cases with unknown diagnosis. Further precise information can be obtained when the smear of the sediments are evaluated qualitatively. For instance, lymphocytosis and paucity of mesothelial cells suggest tuberculosis; presence of immature and abnormal lymphoid cells suggest lymphoma, and the presence of neoplastic cells is diagnostic of neoplasm. When the cytologic findings suggest a specific diagnosis, such as tuberculosis or neoplasm, a specific biopsy is more frequently obtained than when the lymphocyte count is low or the cytologic pattern is nonspecific. Of the 88 biopsies performed in 62 patients with lymphocytic effusions, a specific diagnosis was obtained in 30 patients; while in 53 biopsies performed in 40 patients with less than 50 percent lymphocytes, a specific diagnosis was obtained in 4 patients. It is suggested that pleural lymphocytosis be given more weight and used as a guide, among other criteria, for pleural biopsy."

AWARDS AND HONORS SECTION

NAVY CROSS

Hickey, William L., HM2 USN
Mack, Francis W., HM3 USN, Posthumously
Mayton, James A., HM1 USN

PURPLE HEART

Mironas, Victor, HM3 USN

SILVER STAR

Barraud, Wesley L., HM2 USN
Carper, Loring W., Jr., HN USN (P.H.)
Cooper, David L., HN USN
Counce, Donald E., HN USN
Creed, Edward G., HM3 USN
Fredette, Bradford T., HN USN
Furman, Richard L., HM3 USN
Gunn, Daniel M., HM3 USN
Johnson, Lawrence E., HM3 USN (P.H.)
Johnson, William D., HN USN
Morris, S. A., HM3 USN
Roach, R. F., HN USN (P.H.)
Smith, B. L., HM3 USN

BRONZE STAR

Beeby, J. L., LCDR MC USN
Bird, H. A., HM3 USN
Blair, Thomas B., HM3 USN
Bodkin, Thomas R., HM1 USN
Borbee, William D., HN USN
Bratcher, Ralph E., HM3 USN
Breeze, J. D., HM3 USN
Bridges, Robert J., Jr., HM3 USN
Cannon, T. V., HN USN
Chavez, Richard P., HN USN
Clark, W. D., HM3 USN
Cole, Kermit G., HM3 USN
Enzman, James M., HM2 USN
Erickson, J. S., HM3 USN
Galbally, T. E., HM3 USN
Gallegos, E. M., HN USN
Gonlin, Albert J., HM2 USN
Hand, James E., HMC USNR
Harris, Thomas A., Jr., HM3 USN
Jackson, John L., HM2 USN
Johnson, Walter C., HM2 USN
Keenan, J. M., HM2 USN
Klapacki, John A., HM3 USN
Lane, R. O., HN USN
Layng, F. C., Jr., LT DC USNR
Leckliter, D. W., HM3 USN
Lewis, Richard L., Jr., HM2 USN (2nd Award)
Louis, William, HM1 USN
McCurdy, Donald E., HM2 USN
Molito, G. J., HN USN
Nally, Robert G., HN USN, Posthumously
Palmer, F. E., HM2 USN
Peralta, John R., HN USN
Ray, Ora D., HM3 USN
Savage, R. G., HM3 USN
Scearse, R.D., HM3 USN (2nd Award)
Schaefer, R. W., HM3 USN (2nd Award)
Seabrook, N., Jr., HN USN
Shaefer, R. D., HM3 USN (2nd Award)
Thomas, G. A., HN USN
Wooter, William H., HM2 USN
Zimmerman, C. J., Jr., HM3 USN

LEGION OF MERIT

Anderson, Gustave T., CAPT MC USN
Engle, Paul R., CAPT MC USN
Graybiel, Ashton, CAPT MC USN (2nd Award)
Hering, Alexander C., CAPT MC USN
Wilson, L. H., CAPT MC USN

AIR MEDAL

Campbell, Donald F., HM3 USN (3rd, 4th Award)
Donahue, Richard E., CDR MC USNR

NAVY AND MARINE CORPS MEDAL

Farthing, C. W., HMC USN
Heckert, Paul J., Jr., HMC USN

NAVY COMMENDATION MEDAL

Cash, Carl L., HM1 USN
Cash, Carl R., HMC USN
Chapdelaine, Jack A., CDR MSC USN
Deaner, Richard M., LT MC USN
Erickson, J. S., HM3 USN (also Bronze Star)
Hartman, Carl H., LT MSC USN

Hill, Jack C., Jr., HM2 USN
Horgrave, R. A., HM2 USN
Hunter, Robert E., HM3 USN
Kailer, Charles E., CAPT DC USN
Kjergaard, Arthur M., HM1 USN
Massey, John A., LTJG MSC USN
Pojeky, Ruth M., CDR NC USN (also Vietnamese Medal of Honor, Vol 49 No 3)
Pollard, Joseph P., CAPT MC USN (2nd Award)
Rapson, Dorothy J., LTJG NC USNR
Robinson, Jerry M., LT MC USN
Scola, Francis P., CAPT DC USN
Smith, Lloyd D., LTJG MSC USN
Sproles, Nolan E., HMC USN
Veal, Roy G., HM1 USN

NAVY COMMENDATION FOR ACHIEVEMENT

Bax, M. A., HM1 USN
Becker, David E., LCDR MSC USN
Littlefield, Fred A., HM1 USN
Mart, Russell, HM1 USN
Olliff, Benjamin C., Jr., LT MC USN
Sweany, William G., HM2 USN

JOINT SERVICE COMMENDATION MEDAL

Daye, Jesse R., HMC USN

CERTIFICATE OF COMMENDATION

Long, Charles M., HM2 USN

LETTER OF COMMENDATION

Beeker, G. M., HN USN
Churchville, Francis J., HM2 USN

ARMY COMMENDATION MEDAL

Theros, Elias G., LCDR MC USN
Youngs, Luther A., III, LCDR MC USN

COAST GUARD COMMENDATION MEDAL

Crowell, John F., HM3 USN

DENTAL SECTION

POSTDOCTORAL FELLOWSHIP PROGRAM

Description: The Postdoctoral Fellowship is a program of inservice learning principally for junior officers to obtain earlier advanced training than is possible by awaiting assignment to the Naval Dental School. It should be pointed out that enrollment in a fellowship does not preclude assignment to the Naval Dental School at a later date. The clinical specialty fellowships are similar in content but less vigorous than a first year level (residency) training in the various specialties of dentistry.

Specialties: Postdoctoral Fellowships are available in the clinical fields of periodontology, prosthodontics, endodontics, oral surgery, oral pathology and research. Attention is invited to a newly established Postdoctoral Fellowship Program in Dental Education and Preventive Dentistry at the Naval Dental Technicians School, Naval Training Center, San Diego, California. Applications submitted in accordance with article 6–130 for Fellowships will be considered by the Bureau of Medicine and Surgery Dental Training Committee.

Location: Fellowship sites are not fixed and may change from year to year depending on unique requirements and availability of preceptors. The following 21 sites have been established for 30 participants in the Postdoctoral Fellowship Program for Fiscal Year 1968:

Naval Medical Research Institute, Bethesda, Maryland.
Naval Dental School, Bethesda, Maryland
Naval Medical Research Unit No. 1, University of California, Berkeley, California
Naval Hospital, Great Lakes, Illinois
Naval Dental Research Institute, Great Lakes, Illinois
Naval Air Station, Jacksonville, Florida
Naval Dental Clinic, Camp Pendleton, California
Naval Training Center, San Diego, California
Marine Corps Recruit Depot, San Diego, California
Naval Hospital, Long Beach, California
Naval Hospital, St. Albans, New York

Naval Hospital, Jacksonville, Florida

Naval Station, Little Creek, Virginia

Naval Station, Treasure Island, California

Naval Station, Newport, Rhode Island

Naval Dental Clinic, Washington Navy Yard, Washington, D.C.

Naval Dental Clinic, Norfolk, Virginia

Marine Corps Recruit Depot, Parris Island, South Carolina

Naval Dental Technicians School, San Diego, California

Naval Hospital, Camp Pendleton, California

Objectives: The fellowship program has a twofold aim: first, to provide the junior officer an earlier opportunity for advanced study than he would have by awaiting assignment to the Graduate or Postgraduate Courses at the Naval Dental School. In this respect the fellowship program may be considered as the equivalent prerequisite for other advanced training in exceptional cases. A second aim of the fellowship program is the training of a broader base of skilled specialists for the staffing of dental clinics not requiring board certified personnel, and research facilities operated by the Naval Dental Corps.

Scope: This instruction is designed as academic periods of study, clinical training, teaching experience or research with neither prerequisites nor accreditation toward specialty board certification. In the clinical fields, two or three special cases should be prepared in detail for presentation to the staff. A thesis based either on his special cases or on research, and including a complete literature search should be encouraged but not required.

Prerequisites: Must hold a commission in the Dental Corps of the Regular Navy. Must have completed a tour of duty at sea or in areas considered foreign shore for rotational purposes. Seniority is not a consideration for assignment to a postdoctoral fellowship.

Quota Control: Applicants are considered by the Bureau of Medicine and Surgery Dental Training Committee on a competitive basis reflected by their academic and service records.

Active Duty Following Fellowship: Requires an agreement not to resign during the course and to serve in the Navy for at least 1 year after completion of the Fellowship.

Application: Dental officers intending to submit applications for enrollment in the Postdoctoral Fellowship Program during Fiscal Year 1968 for instruction commencing in the academic year 1968 are encouraged to submit at the earliest convenient date. Early submission of applications to the Bureau of Medicine and Surgery prior to the deadline of 1 December will allow for possible forwarding delays and give sufficient time for processing of records.

In conjunction with or immediately following submission of requests for assignment to the above training program, it is requested that transcripts of academic records earned during predental and dental school training be forwarded to this Bureau, Code 611, for review by the Dental Training Committee. Any charges incurred in the procurement of requested transcripts must be at the expense of the applicant.

Additionally, it is requested that a statement concerning motivation for requesting the above training program, consistent with known abilities, interests and career plan, be forwarded to this Bureau.

General Information: In outstanding cases, the Training Committee may accept the fellowship as the equivalent (MMD 6–124, 6–125 and 6–129) prerequisite for advanced training in a civilian institution. Officers may include in their application a preference site for fellowship training which would be considered by this Bureau in making the assignment following acceptance into the program.

DENTAL OFFICER SERVICE TRAINING AGREEMENT

Attention is invited to BUMED NOTICE 1520 of 3 July 1967, announcing a forthcoming change in MANMED 6–130 and BUMEDINST 1500.9A, Navy Medical Department Formal Schools Catalog, concerning Dental Officer Service Training Agreements.

The changes listed herein are effective as of 1 January 1968. Appropriate changes are being made in current directives. Applications now being submitted to the Bureau of Medicine and Surgery, Code 611, for advanced training to commence in the academic year 1968–1969 will include the following data as appropriate:

(1) Periods of training of over 1 month and less than 5 months duration will require an agreement not to resign during the course and to serve in the Navy for at least 1 year after completion of the course.

(2) Periods of training of 5 to 12 months (other than internships) will require an agreement not to resign during the course and to serve in the Navy after completion of the course as follows:

Graduate/Postgraduate Long Course (Civilian Institution)—3 years

Graduate/Postgraduate Courses, Naval Dental School—2 years

Graduate/Postgraduate Level Training (Residency Type)—2 years

Postdoctoral Fellowship Program—1 year

Discharge of more than one period of required service may not be accomplished concurrently. Time spent in advance training courses cannot be used to discharge previously required service.

APPLICATION FOR ADVANCED TRAINING

Dental officers intending to submit applications for enrollment in long courses of instruction during Fiscal Year 1968 are encouraged to submit at the earliest convenient date. This includes applications for graduate courses, postgraduate courses, first, second and third year level (residency) training, postdoctoral fellowships, and long graduate courses at civilian institutions. Early submission of applications to the Bureau of Medicine and Surgery prior to the deadline of 1 December will allow for possible forwarding delays and give sufficient time for processing of records.

The Dental Training Committee in the Bureau of Medicine and Surgery convenes in early January 1968 to consider applications for advanced training for the 1968–1969 academic year.

In conjunction with or immediately following submission of requests for assignment to any of the above training programs, it is requested that transcripts of academic records earned during predental and dental school training be forwarded to this Bureau, Code 611, for review by the Dental Training Committee. Any charges incurred in the procurement of requested transcripts must be at the expense of the applicant.

Additionally, it is requested that a statement concerning motivation for requesting any of the above training programs, consistent with known abilities, interests and career plan, be forwarded to this Bureau.

NURSE CORPS SECTION

AUTOMATION

LCDR Josephine Remas NC USN, the Central Surgical Room supervisor at the Naval Hospital, Charleston, S.C., spoke recently at the Supervisors' Conference. Her topic "Automation" was compiled from reports she heard at the Central Supply Seminar in Dallas, Texas.

LCDR Remas stressed the need to examine the total hospital organization to find ways to relieve the nurse for nursing. One way in which the nurse can be assisted in caring for her patients is through increasing the efficiency of the CSR Department. She quoted Mr. Gorden Friesen in saying that this can be partially accomplished through architectural design which includes mechanized transportation for delivering supplies, pneumatic tubes for sending messages and small articles and intercommunications systems to keep the nurse and patient in closer contact.

The nurse is also allowed more time for nursing when monitoring devices and disposable supplies are used. The new monitoring devices enable the nurse to assess the condition of her patient more readily and accurately. Disposable supplies increase efficiency and provide greater safety. Among the new disposable items which will soon be tried at the Naval Hospital, Charleston is a "disposable" thermometer. While the instrument is an electronic device, the cover of the probe is changed for each patient. It is hoped that use of the instrument will result in saving of time and money and greater accuracy of reading.

LCDR Remas concluded her talk by stating that, although the initial cost of instituting new forms of automation may appear high, the cost in dollars cannot be compared to the savings that are measured by the value of improved care of patients when nurses are allowed to spend their time nursing.—Nursing Div, BuMed.

FIRE FIGHTING

LT VaVon Lockwood and LT Monica Durupt of the Staff Education Department of the Naval Hospital, Charleston, S.C. recently arranged a program in fire fighting and emergency removal of patients. The aid of Mr. Holt, Chief, and Mr. Massalon, Inspector, of the base Fire Department was enlisted. The two class series was presented twice to enable as many members as possible of the staff to attend.

During the first class the proper procedure to follow in case of fire was presented. The techniques to utilize for the various types of fires and the proper method of using different extinguishers was practiced by members of the audience. Mr. Massalon illustrated the correct manner of suffocating flames when the clothing of a patient was in flames. This dramatic demonstration was accomplished by Mr. Massalon placing an asbestos board on his chest and igniting it so that the participants in the program could get the "feel" of putting out a fire.

The second program consisted of a movie showing the evacuation of patients from a burning hospital and practice in carrying incapacitated patients.

The staff is now more aware of fire hazards and better prepared to act in case of emergency.—Nursing Div, BuMed.

PREVENTIVE MEDICINE SECTION

ALCOHOLISM: A GROWING MEDICAL-SOCIAL PROBLEM

Metrop Life Insur Co Statist Bull 48:7–10, April 1967.

Alcoholism, long viewed by many as essentially a moral issue calling for social censure and punishment, is now increasingly being approached as a health problem requiring the combined skills of medicine, psychiatry, and sociology. It is generally defined as excessive use of alcoholic beverages to a point interfering with health or with economic and social functioning. The additional problem of physical dependence on alcohol is present in chronic alcoholism where withdrawal may produce a severe and even fatal reaction. The exact number of alcoholics in the United States is unknown, but the latest estimate by the National Council on Alcoholism places it at 6,500,000, an increase of 1.5 million within a decade.

There were nearly 11,000 deaths attributed to alcoholic disorders in the United States in 1964. Almost ¾ of these deaths were reported due to cirrhosis of the liver with alcoholism, over a fifth to alcoholism, and the remainder to alcoholic psychosis. The reported death rate from alcoholic disorders has risen steadily in recent years—from 5.5 per 100,000 population in 1950 to 8.7 in 1964, an increase of nearly 60% over the period.

Higher death rates from cirrhosis of the liver with alcoholism accounted for most of the increase, while mortality from the other disorders showed little change during the past 15 years. This situation may reflect earlier and better medical treatment in acute alcoholic episodes among persons who later suffer from chronic liver disease. Cirrhosis of the liver, a frequent complication of alcoholism, has been believed generally to result from nutritional deficiencies in the diets of alcoholics and not from the action of alcohol itself. Recent studies suggest that alcohol can produce fatty liver—the initial stage of alcoholic cirrhosis—even when diets are normal and adequate.

The upward trend in mortality from alcoholic disorders shows considerable variation by sex and race. The rise has been steeper for nonwhite persons than for white, and for women than for men. Death rates for white men age 20 and over rose almost 30% between 1950–51 and 1963–64, compared with over 90% for nonwhite men. Among women, the whites recorded a 75% increase and the nonwhites almost 150%. The increase for women was 2½ times that for men among whites, and 1½ times among nonwhites.

The sharpest increases were recorded among nonwhites in their thirties, where the rate of deaths from this cause went up almost 130% for men and nearly 160% for women. White persons registered the largest increases at later ages: about 50% for

men in their sixties and over 100% for women in their fifties and sixties.

The 1963–64 recorded death rate from this cause among nonwhite men was nearly twice that for white men. The disparity was greatest at the younger ages, but decreased with age so that at ages 60 and over the rate for white men slightly exceeded that for the nonwhite. The peak mortality among white men was in their 60's while among the nonwhite it came in the 50's. In both color groups the mortality rose regularly with advancing age until midlife but tended to decline sharply at the oldest ages—a possible consequence of the high toll in midlife with resultant smaller numbers of alcoholics surviving to old age. The recent alcoholic mortality picture for women was generally similar to that for men. The death rate of nonwhite women also was about double that of white women and their peak mortality likewise occurred at an earlier age than among whites. In both color groups the death rate from alcoholic disorders was much lower for women than for men. Among white persons, the rate for men was nearly 3 times that for women, while among nonwhites the ratio was about 2:1.

The reported mortality from alcoholic disorders does not present an accurate picture of the problem. Several studies suggest that serious understatement exists in the reporting of deaths associated with alcoholism. Because of the social stigma involved, there is reluctance to certify alcoholism as the cause of death where it is possible to assign another cause or complication; this may be a significant factor in the recorded differential between whites and nonwhites in death rates from alcoholism. Thus, it is fairly certain that a considerably greater proportion of deaths ascribed to cirrhosis of the liver are associated with alcoholism than the third so reported. Deaths in accidents involving alcohol are usually assigned to the accident and those resulting from a combination of drugs and alcohol are more likely attributed to the drug.

Alcoholics are subject to distinctly higher than average death rates. A recent insurance study indicated that persons with a history of alcoholism experienced mortality 2½ to 3 times higher than standard risks. The heaviest excess mortality was due to diseases of the digestive system, suicide, motor vehicle accidents, other accidents, and homicides.

The cost of alcoholism is not adequately reflected by morbidity and mortality statistics. The National Council on Alcoholism estimates an annual loss to industry of over $2 billion, resulting from absenteeism, lowered productivity, and accidents associated with alcoholism, as well as an immeasurable toll of disrupted family life.

Over the past 30 years there has been a considerable effort to deal with alcoholism by interested physicians, Alcoholics Anonymous, the National Council on Alcoholism, and industrial programs. Recently the Department of Health, Education, and Welfare undertook a major program of research, education, and professional training to combat alcoholism. Its objectives are to make available to alcoholics the best treatment and rehabilitation services, to improve techniques of treatment, and to find effective ways of preventing the disorder. A national center for the prevention and control of alcoholism is being established within the National Institute of Mental Health at Bethesda, Maryland. An 18-member national advisory committee on alcoholism is being set up to include representatives of medicine, social work, labor, industry, vocational rehabilitation, education, law, and civic organizations concerned with alcoholism. It is expected that these efforts will spur new and better approaches to combatting the menace of alcoholism.

STREPTOCOCCI AND RHEUMATIC FEVER

Lancet 1(7488):485, Mar 4, 1967.

The connection between steptococcal infection and rheumatic fever is now firmly established by 3 sources of evidence—epidemiological, serological, and prophylactic. The incidence of rheumatic fever both in the general population and in closed communities clearly follows the rise and fall in streptococcal infections. The serological demonstration of antibodies to various streptococcal products in almost 100% of the patients with acute rheumatic fever confirms this epidemiological relationship. Finally, the unquestionable value of antistreptococcal chemotherapy in the prevention of initial attacks of rheumatic fever after known infection with streptococci, as well as the success of the same drugs in preventing recurrences, confirms that the streptococcus is the protagonist of this disease. Since streptococci themselves are not found within the lesions of rheumatic fever, and since none of the known products of these organisms has the ability to induce these lesions by

direct action, the view which has gradually been accepted is that some immunological response, primarily directed against the streptococci, somehow or other comes to involve the host's own tissues. And this idea has lately received strong support.

The most obvious reason why an immune reaction to an exogenous agent should also affect an individual's own tissues is the existence of immunologically cross-reacting groups within the tissues and the organisms. The existence of such groups in the human myocardium cross-reacting with an antigen in several strains of group-A Beta-hemolytic streptococci was first demonstrated by Kaplan and Meyeserian, and has now been confirmed. The antigen in the heart is associated with the subsarcolemmal region of the myocardial fibres, and in the steptococci it is associated with the cell wall. Its occasional presence in organisms other than group A suggests that it is not part of the group-specific polysaccharide, and its occasional presence in strains lacking the type-specific M protein also excludes this component. The corollary to the discovery of cross-reacting antigens in the myocardium and in streptococci is the existence of corresponding cross-reacting antibodies in the blood of patients with rheumatic fever. These antibodies were also first identified by Kaplan, et. al., whose findings have since been fully confirmed by other workers.

Apart from the Aschoff body, which is widely regarded as the histological hallmark of active rheumatic carditis, the most characteristic cardiac feature of the disease is the damage to the valves. Apparently this action, like that on the myocardium, can also be attributed to cross-reacting antigens. Halpern and his colleagues in Paris now report a striking cross-reactivity between the structural glycoproteins of the heart valves, both human and bovine, and the polysaccharide responsible for the group specificity of the Beta-hemolytic streptococci. Furthermore, they say that "recently we were able to demonstrate the presence of antibodies which react with the urea extract of heart valves (valve structural glycoproteins) in the sera of patients with rheumatic disease." A word of caution is necessary: the presence of antibodies capable of specific reaction with the affected organ in any disease does not prove that the antibody causes the disease. It is conceivable, and often highly probable, that the antibodies are the outcome of the disease, not its cause—the result of the release of antigen from the damaged organ. The failure to produce convincing cardiac lesions of rheumatic type in any of the many experimental animals injected with streptococcal vaccines (despite the development of antibodies cross-reacting with myocardium and presumably with valve constituents) strongly suggests that something more than humoral antibodies is required. Even when the organisms are injected with Freund's complete adjuvant to stimulate the appearance of specifically sensitized cells in addition to antibody, rheumatic carditis does not arise. Nevertheless, in view of the unquestionable link between steptococcal infection and rheumatic fever, these recently established immunological relationships must surely be significant.

ONCHOCERCIASIS

Brit Med J 1(5531):3, Jan 7, 1967.

For some time the World Health Organization (WHO) has been concerned about the effects of onchocerciasis in developing countries. The disease attacks about 20 million people in Africa and another 200,000 in Central America. Thus in 1953 and again in 1965 the WHO organized meetings of committees of experts to advise on the clinical effects of the disease, its means of transmission, and its influence on economic development.

Onchocerciasis is an infection by a filarial worm. The adults live in the subcutaneous tissue, often encapsulated in fibrous nodules, and the microfilariae or embryos travel in the skin, whereas in most other filarial diseases they are found in the blood. In the skin the microfilariae cause pruritus and excoriated dermatitis and eventual atrophy, commonly referred to as presbydermia. Unfortunately they find their way from the skin of the face to the conjunctiva and thence to the cornea and iris, where they produce keratitis and iritis. Choroidoretinitis also occurs in patients who are heavily infected for long periods. The keratitis and iritis, when severe, may cause blindness; the fundal lesions do so much less frequently.

The infective forms of the parasite are transmitted by the bites of various species of *Simulium* flies. The larval stages of the flies can live only in water which is well oxygenated, and so the flies breed mostly in hilly country with tumbling, clear, bubbling streams. Well-watered country frequently has fertile soil and is usually densely populated, so considerable foci of

the disease occur. In Africa and Central America the endemic areas support innumerable small cultivators and also contain many large estates producing cash crops such as coffee, tea, and rubber—from which much of the countries' foreign earnings derive. The disease is endemic also in less hilly country—as, for example, the savannahs of northern Ghana and Nigeria—provided there is sufficient well-oxygenated water, and here spillways from small earth dams and irrigation channels can permit the growth of enormous numbers of the flies. Country such as this, though usually less economically productive than the hillier regions with their higher rainfall, nevertheless contains large numbers of people exposed to the infection. So this disease, with its power to impair vision and cause blindness, may affect the economic viability of whole regions, and indeed some fertile parts of northern Ghana and the Sudan have become depopulated as a result of it. On the other hand, the WHO report relates that when control operations were undertaken to protect workers building the Owen Falls dam in Uganda there was a spectacular uncapitalized development of the region and previously untenable land became cultivated for cash crops.

In addition to giving advice on the control of the disease and its vectors, the World Health Organization through its scientific groups and expert committees has arranged and provided for much survey work, particularly in West Africa. It has also organized a scheme for studying the disease in some areas of Kenya where transmission was interrupted 9 to 18 years ago. In people who, because of their age, could not have been exposed to infection for more than 15 years at the time of interruption ocular and skin lesions were not found with higher frequency than in the uninfected population. This study also provided evidence of the length of life of the adult worm. "The follow-up survey revealed that 11 years after interruption of transmission live adults and microfilariae were still found. Eighteen years after interruption no microfilariae were found and all excised nodules contained dead worms." Data of this kind are fundamental to control programs.

In its report WHO summarizes much that has been done in the past decade, the present position is outlined, and the many future research needs are discussed. This publication deserves most careful consideration by all concerned with Central Africa and Central America.

A COMMON SOURCE OUTBREAK OF CUTANEOUS LARVA MIGRANS

C. E. Fuller DVM MPH, USDHEW PHS
Public Hlth Rpts 81(2):186–190, Feb 1966.

During the construction of a new hospital at an Air Force Base, in Florida, 7 of 9 employees working in the crawl space below the building developed severe dermatitis. The skin eruptions were attributed to exposure to larva of *Ancylostoma braziliense*. The men lay on their backs or sides on the ground in a 3-foot-high crawl space to install and paint pipes suspended from the floor above.

The workers reported that they frequently saw tracks of dogs and cats in the area when they began work in the mornings. In Florida the incidence of *A. braziliense* in these animals is high, and the moist sandy loam in the shady crawl space particularly favored the development of ova. Feline feces buried at night in the soil served as a vehicle of infection when the fecal material touched the workers' arms, legs, and backs.

Ethyl chloride spray was prescribed for the dermatitis. Control measures recommended for the crawl space were the screening of entrances to prevent entry of animals, spraying the larvicide sodium borate at concentrations of 10 lbs per 100 square feet of soil, covering the earth with heavy plastic sheeting for the workmen to lie on whenever possible, and encouraging the men to wear long-sleeved shirts, secure trouser legs at the ankles, and shower immediately after work. A 6-month follow-up indicated that none of the men were reinfected after the larviciding of the crawl space soil.

HOOKWORM INFECTION AND INTESTINAL BLOOD LOSS

C. Martinez-Torres, et. al., Trans Roy Soc Trop Med & Hyg 61(3):373–383, 1967.

The study of 54 subjects infected with *Necator americanus* showed a high correlation between fecal blood loss measured with ^{51}Cr and hookworm infection estimated either by the number of parasites recovered or by oviposition. Fecal blood loss was of the order of 0.03 ml per worm daily, and 2.1 ml per 1,000 eggs per gram of feces. The number of eggs per female *Necator* was about 3,000 per day and 30 per gram of feces.

In 5 cases studied at different levels of hemoglobin concentration the fecal blood loss and oviposition did not show any consistent pattern. The red cell survival measured with ^{51}Cr was corrected after iron treatment in 4 out of 5, although they were carrying heavy infections, indicating that the toxin of the parasites does not interfere with the life span of the erythrocytes.

MEASLES FILM

USDHEW PHS Public Hlth Rpts 82(3):252, March 1967.

"Spot Prevention," a film to support and promote measles immunization, has been produced by the Public Health Service Audiovisual Facility. This fast-moving, humorous film, showing the chase and capture of measles "germ" and his "conversion" to protective vaccine, is animated, with a live introduction and closing. Geared toward children from preschool to second grade but enjoyable to adults, the film is particularly adaptable for use by schools, churches, civic organizations, and television.

The 16 mm. color film, with sound, runs 13½ minutes. It is available for free short-term loans from the Public Health Service Audiovisual Facility, Atlanta, Georgia 30333, Attention: Distribution Unit. It can be purchased from Du Art Film Laboratories, Inc., 245 West 55th Street, New York 10019.

KNOW YOUR WORLD

Did You Know?

That analysis of questionnaires from 2,606 persons sensitive to insect sting reveals an expectancy of progressively severe reactions in about 65% of persons not hyposensitized?

Following hyposensitization, reactions to subsequent stings are reduced in about 90% of treated persons. Protection may be maintained for years or may be lost in less than a year. Progressively severe reactions to stings are uncommon following recent adequate hyposensitization. Hyposensitization is recommended for persons who have had any degree of systemic sensitivity following an insect sting. Epinephrine is the primary drug of choice for treatment of an acute, immediate reaction to insect sting.[1]

That increases in the reported incidence of jungle yellow fever and plague, as well as wider geographical distribution of these diseases, were recorded in 1966 in the Americas as compared with 1965?

One case of louse-borne relapsing fever was reported, the first since 1963. Cholera was again absent, after the occurrence of two laboratory acquired cases in 1965.[2]

That louse-borne typhus persists in the mountainous regions of Mexico and the Andean regions of South America?

In 1966 outbreaks from 6 to 86 cases were reported in the villages of Bolivia, Chile, Ecuador, Mexico and Peru. Of the total 455 cases, 359 occurred in these local outbreaks.[3]

That the State of Barbados has officially become a member of WHO? [4]

That 53 cases of poliomyelitis occurred in Nicaragua from 5 Feb through the third week of May 1967?

Twenty-six of the cases occurred in the Department of Managua. Age distribution for all cases: under 1 year—11 cases; 1–4 years—37; 5–14 years —4; unknown age—1. Throughout the country, 200,000 doses of vaccine are being administered in intensified vaccination programs.[5]

That of 1,360 million cubic kilometers of water in all its forms on the surface of the Earth, only 35 million cubic kilometers consists of fresh water, and 80% of that is in the form of snow and ice?

An attempt to determine accurately the quantity of ice and snow on the Earth's surface is to be carried out under the auspices of the International Commission of Snow and Ice as part of the contribution of glaciologists to the International Hydrological Decade. Wet water often makes an important contribution to irrigation and the production of hydroelectricity.[6]

That the United States of America has recently given 5 million rupees to India to finance various projects in agriculture, health, education and community development?

Health and sanitation projects will be assigned close to 500 million rupees for programs to eradicate malaria and smallpox, to establish rural health centers, and to provide drainage, sanitation and water-supply systems.[7]

That 151 out of 210 countries or other population units, now live in areas originally at risk to malaria?

While 953 million people are free of active malaria transmission there are 638 million who live in areas where transmission of malaria still continues.[8]

That it is estimated that worldwide between 3 and 4 million persons die each year from tuberculosis and that more than 2 billion persons, if tested, would be identified as reactors to tuberculin? [9]

References

1. JAMA 193(2): 115-120, Jul 12, 1965.
2. PANAMSANBU WHO Wkly Epid Rpt XXXIX (16): 91, Apr 19, 1967.
3. PANAMSANBU WHO Wkly Epid Rpt XXXIX (16): 92, Apr 19, 1967.
4. JAMA 200(12): 218, Jun 19, 1967.
5. PANAMSANBU WHO Wkly Epid Rpt XXXIX (23): 132, Jun 7, 1967.
6. NATURE 214(5093): 1073-1074, Jun 10, 1967.
7. JAMA 200(7): 179, May 15, 1967.
8. WHO Expert Committee on Malaria, WHO Technical Rpt Series No. 357, p 6, 1967.
9. Dis Chest 51(6): 668, Jun 1967 (Davey, W. N., Bol Ofic Saint Panamer 61:144, 1966).

EDITOR'S SECTION

ANNIVERSARY MESSAGE FROM SECNAV TO MSC OFFICERS

On this 20th Anniversary of the Medical Service Corps of the United States Navy, I extend my personal greetings and best wishes to each of you.

Your record of accomplishments in the two decades since the establishment of the Medical Service Corps is enviable and fully justifies the pride that is so evident among all the officers of your Corps. I have every confidence that you will continue to discharge your responsibilities and accept new challenges with the same determination you have so ably demonstrated in the past.

To all officers of the Medical Service Corps—HAPPY BIRTHDAY!

s/Robert H. B. Baldwin
Acting

ANNIVERSARY MESSAGE FROM THE SURGEON GENERAL TO MSC OFFICERS

It is with pride and pleasure that I extend to you my most hearty congratulations on the 20th Anniversary of the Medical Service Corps and I assure you that all members of the Medical Department join me in these felicitations.

The men and women who comprise your Corps have acquired a well deserved reputation for their abilities, devotion to duty, and spirit of service without which our efforts to accomplish our mission would be sorely ham-

pered. I have every confidence that you will continue your fine accomplishments and accept new responsibilities and challenges with the same willingness and loyalty to duty you have demonstrated so well during the past 20 years.

To each and every one of you, wherever you may be serving, I extend my warmest regards and wish you a HAPPY BIRTHDAY!

R. B. BROWN
Vice Admiral MC USN
Surgeon General

POSTGRADUATE SHORT COURSES FOR MEDICAL DEPARTMENT OFFICERS

Sponsored by the Department of the Army and Air Force During Fiscal Year 1968.

The following postgraduate professional short courses will be conducted by the Army and Air Force Medical Service during Fiscal Year 1968. Eligible Medical Corps, Dental Corps and Nurse Corps officers, are those who meet the criteria prescribed by BUMED INSTRUCTION 1520.8 Series; Manual of the Medical Department 6–130; and BUMED INSTRUCTION 1520.14 Series, respectively. Eligible Medical Service Corps officers are those who are currently assigned to billets with a direct relationship to the courses listed and should apply in accordance with BUMED INSTRUCTION 1520.12 Series. Officers desiring to attend should submit their requests in ample time to reach the Bureau at least 8 weeks prior to the convening date of the course desired. This lead time is necessary in order to comply with the Army and Air Force requests to return unused quotas 6 weeks in advance of the convening dates of the courses listed.

Army Courses

Courses	*Installation*	*Date*
Advanced Course in Aerospace Pathology	Armed Forces Institute of Pathology	15–17 Nov 1967 MC
Advanced Pathology of the Oral Regions	Army Institute of Dental Research, Walter Reed Army Medical Center	11–15 Mar 1968 DC
Application of Histochemistry to Pathology	Armed Forces Institute of Pathology	15–19 Jan 1968 MC
Current Problems and Trends in Medical Plans and Operations	Fitzsimons General Hospital, Denver, Colorado	4–8 Dec 1967 MC

Courses	*Installation*	*Date*
Eighth Annual Armed Forces Institute of Pathology Lectures	Armed Forces Institute of Pathology	18–22 Mar 1968 MC
Fifteenth Annual Armed Forces Institute of Pathology Course in Oral Pathology	Armed Forces Institute of Pathology	4–8 Mar 1968 DC
Forensic Pathology	Armed Forces Institute of Pathology	1–5 Apr 1968 MC
Genitourinary Pathology	Armed Forces Institute of Pathology	13–17 Nov 1967 MC
Geographic Pathology of Infectious Diseases	Armed Forces Institute of Pathology	8–12 Apr 1968 MC
Global Medicine	Walter Reed Army Institute of Research	15 Aug–22 Dec 1967 13 Feb–21 Jun 1968 MC
Introduction to Electron Microscopy	Armed Forces Institute of Pathology	27 Nov–1 Dec 1967 MC
James C. Kimbrough Urological Seminar	Brooke General Hospital BAMC, Fort Sam Houston, Texas	30 Oct–2 Nov 1967 MC
Maternal and Child Health Nursing	William Beaumont General Hospital, El Paso, Texas	6–15 Jun 1968 NC
Neuropathology	Armed Forces Institute of Pathology	22–26 Jan 1968 MC
Nutrition Review for Therapeutic and Research Dietitians	Walter Reed Army Institute of Research	18–29 Mar 1968 MSC
Occupational Therapy Educational Supervisors Course	Walter Reed Army Institute of Research	10–12 Jun 1968 MSC
Ophthalmic Pathology	Armed Forces Institute of Pathology	11–15 Sept 1967 5–9 Feb 1968 MC
Oral Diagnosis and Therapeutics	Army Institute of Dental Research, Walter Reed Army Medical Center	22–26 Apr 1968 DC
Oral Surgery	Army Institute of Dental Research, Walter Reed Army Medical Center	15–19 Jan 1968 DC

Courses	*Installation*	*Date*
Orthopedic Pathology	Armed Forces Institute of Pathology	25 Sept–3 Nov 1967 MC
Otolaryngology Basic Science Course	Armed Forces Institute of Pathology	15 Apr–24 May 1968 MC
Pathology of the Larynx	Armed Forces Institute of Pathology	6–8 Sept 1967 MC
Periodontics	Army Institute of Dental Research, Walter Reed Army Medical Center	12–16 Feb 1968 DC
Postgraduate Course in Military Ophthalmology	Walter Reed General Hospital, Walter Reed Army Medical Center	8–11 Apr 1968 MC
Postgraduate Course in Oral Surgery	Letterman General Hospital, San Francisco, California	8–12 Apr 1968 DC
Postgraduate Course in Prosthodontics	Letterman General Hospital, San Francisco, California	9–13 Oct 1967 DC
Postgraduate Course in Restorative Dentistry	Letterman General Hospital, San Francisco, California	4–8 Dec 1967 DC
Present Concepts in Internal Medicine	Letterman General Hospital, San Francisco, California	7–10 Nov 1967 MC
Preventive Dentistry	Army Institute of Dental Research, Walter Reed Army Medical Center	9–13 Oct 1967 DC
Principles of Military Dental Research	Army Institute of Dental Research, Walter Reed Army Medical Center	13–17 May 1968 DC
Prosthodontics	Army Institute of Dental Research, Walter Reed Army Medical Center	4–8 Dec 1967 DC
Special Environmental Pathology-Southeast Asia	Armed Forces Institute of Pathology	20–24 May 1968 MC
Surgical and Orthopedic Aspects of Trauma	Brooke General Hospital, BAMC, Fort Sam Houston, Texas	4–7 Mar 1968 MC
Symposium on Current Surgical Practices	Walter Reed General Hospital, Walter Reed Army Medical Center	1–3 Apr 1968 MC–NC

Courses	*Installation*	*Date*
The Ogden C. Bruton Pediatric Seminar	Walter Reed General Hospital, Walter Reed Army Medical Center	6–8 Mar 1968 MC
The Science of Materials in Dentistry	Army Institute of Dental Research, Walter Reed Army Medical Center	11–15 Sept 1967 DC
Tri-Service Orthopedic Seminar	Fitzsimons General Hospital, Denver, Colorado	18–21 Sept 1967 MC
Twentieth Annual Symposium on Pulmonary Diseases	Fitzsimons General Hospital, Denver, Colorado	11–15 Sept 1967 MC

Air Force Courses

Courses	*Installation*	*Date*
Global Medicine OZY 9336—Class 68–A	Air Force School of Aerospace Medicine, Brooks AFB, Texas	22 Jan–2 Feb 1968 MC
Lectures in Aerospace Medicine OZY 9356–2—Class 68–A	Air Force School of Aerospace Medicine, Brooks AFB, Texas	5–8 Feb 1968 MC
Medical Aspects of Advanced Warfare OZR 9300–2—Class 67–B	Medical Service School USAF, Sheppard AFB, Texas	6–10 Nov 1967 MC
Medical Aspects of Advanced Warfare OZR 9300–2—Class 68–A	Medical Service School USAF, Sheppard AFB, Texas	5–9 Feb 1968 MC

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