NAVMED P-5088

UNITED STATES NAVY
Medical News Letter

Vol. 50 Friday, 3 November 1967 No. 9

Surgeons General of the Past

(The twenty-third in a series of brief biographies)

Ross T McIntire was born in Salem, Oregon on 11 August 1889, graduated from the Willamette University Medical School in 1912, and was appointed Assistant Surgeon in the Navy 4 April 1917. He served aboard the New Orleans which played a part in supporting Admiral Kolchak's counterrevolutionary drive against the Bolsheviks, and then at the Canacao (P.I.) naval hospital, the Naval Medical School, and at the San Diego Hospital as Head of the EENT Department. In the latter capacity he also served twice on the hospital ship Relief. In 1931 Doctor McIntire was assigned to the Washington Naval Hospital with additional duty as Instructor in Ophthalmology and Otolaryngology at the Naval Medical School. In 1933 he became physician at the White House and in 1938 was appointed Surgeon General. While serving as President Roosevelt's personal physician and accompanying the President on many World War II diplomatic missions and military inspection trips, he directed the largest water-borne medical department in history. Prefabricated and transportable hospitals of several types were developed to support combat installations, and large hospital ships (as well as many smaller ships used also for attack purposes) were utilized to provide medical care and evacuate patients from combat areas. Logistic support was furnished by over 32 medical supply facilities, depots, warehouses and supply barges. Vice Admiral McIntire acted primarily as a policy maker and led over 175,000 doctors, dentists, nurses and corpsmen engaged in far flung activities related to surgery, preventive medicine, research, training, personnel recruitment, new drugs, and submarine and aviation medicine. After a quite active retirement period Admiral McIntire died in Chicago 7 December 1959.

*United States Navy*
*MEDICAL NEWS LETTER*

Vol. 50 Friday, 3 November 1967 No. 9



*Policy*

The U.S. Navy Medical News Letter is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article, in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

*Change of Address*

Please forward changes of address for the News Letter to Editor: Bureau of Medicine and Surgery, Department of the Navy, Washington, D.C. 20390 (Code 18), giving full name, rank, corps, old and new addresses, and zip code.

## CONTENTS



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

# EMERGENCY MANAGEMENT OF UNEXPECTED DEFECTS OF HEMOSTATIC FUNCTION IN SURGICAL PATIENTS

*William B. Wilson MD, JAMA 201(2):123–126, July 10, 1967.*

From time to time the surgeon is faced with the problem of an apparent defect of hemostatic function in a patient during surgery or postoperatively. This usually requires prompt and rational management, if undesirable complications are to be avoided.

*Preoperative Screening.*—Although this paper deals primarily with operative and postoperative management of unexpected defective hemostasis, it is appropriate to mention first the preoperative screening of patients for bleeding tendency, since this is by far the most important approach to the problem. It should go without saying that surgery should never be undertaken lightly in patients with a known or suspected true defect of coagulation, no matter how mild the defect is supposed to be. Elective operation on the so-called mild hemophiliac, without proper preparation, can especially give cause for regret, since the postoperative management of these patients is always complicated and costly.

A brief but appropriate history is the easiest and best screening device for detecting a bleeding tendency. Symptoms of bleeding tendency are usually easier to elicit than to evaluate. Bleeding of truly pathologic significance usually lasts more than 24 hours. Bleeding with tonsillectomy and oozing after tooth extraction, unless quite prolonged, are so nonspecific as to be of little diagnostic value. "Easy bruising" is nonspecific and usually can be disregarded unless it is of recent onset, or unless it has been particularly severe, with repeated hematoma formation. In these cases it requires thorough investigation. One should always inquire about current anticoagulant therapy. A patient with a family history suggestive of bleeding tendency should always be investigated by the laboratory.

After obtaining the history, if a hemorrhagic diathesis is suspected, thorough laboratory investigation of hemostatic function should be done before surgery, if at all possible. This should begin with a properly performed determination of Ivy bleeding time, platelet count, prothrombin time, partial thromboplastin time, and tourniquet test, and blood smear for evaluation of leukocytes and platelet morphology and aggregation. Further tests which may be of value are the prothrombin consumption, platelet adhesiveness, clot retraction, clot stability, fibrinogen assay, and fibrinolysin tests. All these tests are within the capabilities of well-trained technologists. Should any results be positive or borderline, surgery should be deferred pending hematologic consultation.

*Initial Management.*—A true coagulation defect which becomes first apparent during surgery is generally due to a limited number of causes, which include the following: (1) hemostatic defect of multiple transfusions; (2) acute fibrinogenopenia; (3) unsuspected mild hemophilia; (4) unsuspected prothrombin deficiency (usually due to anticoagulant therapy); (5) unsuspected thrombocytopenia, thrombocytopathy, or von Willebrand's disease; (6) circulating anticoagulants; and (7) shock and/or hypoxemia (if not a primary cause, hypoxemia is at least contributory).

When the surgeon is faced with a previously unsuspected hemostatic defect in a patient on the operating table, he should first be satisfied in his own mind that such a defect does exist, as evidenced by uncontrollable oozing at the operative site or possibly ecchymoses or mucous membrane hemorrhages elsewhere. Uncontrollable oozing suggests a true coagulation defect such as acute fibrinogenopenia, unsuspected prothrombin deficiency, or hemophilia. Petechiae and ecchymoses suggest platelet deficiency, but may be seen with acute fibrinogenopenia. If the surgeon feels a true defect to be present, he must call for immediate help from the laboratory. A drop of blood is smeared for estimation of platelet numerical adequacy, and venous blood is obtained in anticoagulant for determination of prothrombin time, and for a rapid screening test for fibrinogen (thrombin [Fibrindex] is fast, easy, and reasonably accurate, within the limitation to be discussed below). These tests can all be done in a few minutes, and, if, positive,

From the Department of Pathology, Mississippi Baptist Hospital, Jackson.

provide a basis for rational, effective therapy. If results are negative, a test of partial thromboplastin time is ordered. Every effort must be made to prevent shock and hypoxemia, as these may have an adverse effect on hemostasis.

*Hemostatic Defect of Multiple Transfusion Therapy.*—This syndrome possibly represents a more complex defect than is implied in the popular term, "platelet washout." It is not uncommon and is often fatal. It usually presents as a defect of hemostasis after at least 6 to 10 units of stored blood are given within a few hours' time. In the severe or fatal cases, thrombocytopenia is invariably present and fibrinogen is usually at subnormal or borderline levels. Generally, it is seen at operations in which large amounts of stored blood are transfused, but it may occur when multiple transfusions are required because of trauma or gastrointestinal bleeding; it may be first suspected only after such a patient gets to surgery. Hemostatic defect of multiple transfusion therapy can be rapidly diagnosed by examining the blood smear for decreased platelets.

Therapy is best accomplished by prevention; ie, a unit of fresh blood should routinely be given after 6 to 8 transfusions, and thereafter every third or fourth unit transfused should be fresh blood. If prevention is not obtained, therapy consists of (*a*) immediate administration of the freshest blood available; (*b*) simultaneous administration of fibrinogen, 1 gm initially, with subsequent infusions monitored by rapid fibrinogen assay of the patient's blood; and (*c*) administration of 500 mg of hydrocortisone intravenously. The blood bank should be ordered immediately to begin bleeding donors for further transfusion of fresh or platelet-rich plasma. The hemostatic defect of multiple transfusions is a grave emergency, requiring prompt and vigorous therapy.

For the preservation of platelets, the relative values of plain glass bottles, siliconized bottles, and plastic bags are probably about equal. Plastic bags are preferred by many blood banks. Regardless of what container is used, it is well established that storage of blood rapidly decreases the yield of biologically effective platelets. Ideally, platelets should be transfused within six hours after collection. In an emergency, blood up to 48 hours old may be used.

*Acute Fibrinogenopenia.*—This may be due to depletion of circulating fibrinogen by diffuse intravascular thrombosis (intravascular defibrination syndrome), or it may be due to increased fibrinolytic activity of the blood; usually, both mechanisms are operative. Acute fibrinogenopenia occurs in a number of well-recognized situations: (1) physical trauma; (2) hemorrhagic shock; (3) extensive burns; (4) disseminated carcinoma; (5) hemorrhagic defect of multiple transfusion therapy; (6) severe hemolytic transfusion reactions; (7) acquired hemolytic anemia; (8) extensive liver disease (even if prothrombin level is normal); (9) polycythemia, especially that of congenital heart disease; (10) during surgery of the lungs, pancreas, uterus, or prostate; and (11) any of the several well-defined obstetrical complications (which will not be considered here).

Development of a hemostatic defect at surgery in any of the above clinical settings, especially if preoperative history and laboratory tests indicated normal hemostatic function, should suggest acute fibrinogenopenia. The laboratory personnel can confirm this immediately by doing a rapid fibrinogen assay on a sample of blood treated with anticoagulant. If the results of this test are positive or even equivocal, or if such an assay is not immediately available, fibrinogen is given intravenously without delay. Dosage is judged adequate when the bleeding stops, as it usually does promptly. Prevention of shock and hypoxemia is most important, as they have a distinctly adverse affect on the hemostatic mechanism in acute fibrinogenopenia. Because of this, and because platelets and other factors may be acutely depleted by intravascular defibrination, transfusions of blood or plasma should be given freely, with fresh blood or plasma when possible.

The rapid tests for fibrinogen may not be reliable in the presence of active fibrinolysis, because fibrinolysis may produce circulating protein fragments ("split products") of fibrinogen which may, in turn, react in vitro in much the same manner as fibrinogen. Thus, in the thrombin-clot test for fibrinogen (Fibrindex), split products may interfere with the thrombin-fibrinogen reaction and prolong the clotting time, giving a result suggestive of more severe fibrinogenopenia than is actually present. On the other hand, in the agglutination test for fibrinogen (Fi-Test), the split products may react with the antibody-coated latex particles and cause agglutination, thus suggesting a normal or nearly normal level of fibrinogen. It would appear that when fibrinolysis is present, either as a primary process or secondary to activation by intravascular defibrination, the thrombin-clot test would be less likely to cause confusion. Confirmation of results by a more specific assay of fibrinogen should always follow a rapid determination.

In acute fibrinogenopenia, a laboratory differentiation between intravascular defibrination and fibrinolysis should be attempted. This is complicated by the fact that intravascular defibrination appears to initiate excessive activity of the fibrinolytic system. Thus, in either situation, laboratory tests may show both fibrinolysis and fibrinogenopenia. Fortunately, the immediate therapy can be logically directed toward replacement of the depleted fibrinogen, regardless of whether intravascular defibrination or fibrinolysis, or both, is the cause. For this reason, when fibrinogenopenia is demonstrated in an actively bleeding patient, the first line of defense is the intravenous infusion of fibrinogen.

More precise therapy may be accomplished by the use of aminocaproic acid, which may dramatically inhibit fibrinolysis; or by the use of sodium heparin, which inhibits intravascular defibrination. These drugs should only be used under the direct supervision of one well acquainted with their action and dangers. It has been suggested in a recent review that both drugs be used together—aminocaproic acid to inhibit excessive fibrinolysis, and heparin, in small doses, to inhibit excessive intravascular thrombosis.

There is considerable evidence that fibrinogen preparations carry the virus of homologous serum hepatitis, with the incidence having been reported to vary from 6 percent to 35 percent. The use of $\gamma$-globulin may offer some prophylaxis against post-transfusion hepatitis. Giving the first dose a few days after exposure may be of more benefit than giving it immediately.

*Hypoprothrombinemia and Unsuspected Anticoagulant Therapy.*—Emergency surgery is sometimes necessary on patients on whom no history whatever is available. Routine preoperative determination of prothrombin time in these cases will prevent surgery on patients with hypoprothrombinemia (usually due to unsuspected anticoagulant therapy). When surgery is done on patients treated with anticoagulants, the bleeding tendency is evident from the outset. Colloidal phytonadione (vitamin $K_1$) administered intravenously furnishes the most satisfactory correction of the prothrombin time, provided liver function is adequate, but requires 6 to 12 hours for effect. If more rapid action is needed, or if liver function is poor, blood or plasma not more than a few days old can be given to replace the missing clotting factors. The dosage of phytonadione will vary with the duration of the prothrombin time. Excessive doses may, paradoxically, further prolong the prothrombin time, especially when liver disease is present. Vitamin K analogs do not antagonize coumarin-type drugs, and may be toxic in those with liver disease.

*Unsuspected Hemophilia.*—Cases of mild hemophilia or "subhemophilia" may be clinically unsuspected until the appearance of a coagulation defect at surgery, although this catastrophe is more commonly feared than encountered. Only the most sensitive laboratory tests (factor VIII assay, thromboplastin generation test) can detect the mildest forms of the disease, and the technical difficulty of these tests prevents their widespread application. The partial thromboplastin time test is almost as good as the above tests; it should not be substituted for a careful history as a routine preoperative screen, but it is easy to do and should be available for selected cases. The prothrombin consumption test is easier and cheaper still, but is not as sensitive as the partial thromboplastin time and takes longer. The Lee-White clotting time and capillary-tube clotting time tests are only capable of detecting moderate to severe hemophilia, which should be clinically obvious by history, except in infants.

In considering cases of suspected hemophilia at surgery, one is concerned mainly with classical hemophilia (hemophilia A, factor VIII or antihemophilic factor [AHF] deficiency), since its management is much more difficult than that of the other hemophilia type or stage I deficiencies (these include factor IX deficiency [hemophilia B, Christmas disease], factor XI [plasma thromboplastin, antecedent] deficiency, and Carr factor deficiency, all of which are corrected with stored blood or plasma; and thrombocytopathy and thrombocytasthenia, which are correctable with only 1 or 2 units of fresh blood or platelet-rich plasma).

Classical hemophilia at surgery is a formidable problem, and it should never be underestimated, even in so-called mild cases. The diagnosis is considered if the patient is male (especially a child), and if platelet, fibrinogen, and prothrombin deficiencies have been excluded by the laboratory. Usually, but not always, the bleeding tendency is evident from the beginning of the operation. If these circumstances obtain, a presumptive diagnosis of hemophilia A is justified, and therapy is immediately started. (Blood should first be drawn for a stat partial thromboplastin time or prothrombin consumption test, or both; these take 30 to 60 minutes, and therapy cannot await the results.) The operation should be terminated as soon as feasible.

Initial therapy depends on what is immediately available. If antihemophilic globulin (concentrated factor VIII) is available, it should be given

generously. The recently developed cryoprecipitate is the most satisfactory form of concentrated factor VIII. If fresh-frozen plasma is available, 2 to 4 units of it may be given. The agent of third choice is factor VIII-rich fibrinogen (Fibro-AHF), which is available in most hospital pharmacies. Not all fibrinogen preparations are factor VIII rich, and the potency of different lots varies considerably. Effective levels of factor VIII should be achieved rapidly.

If the results of the stat partial thromboplastin time indicate that factor IX deficiency, or one of the other stage I deficiencies, is present instead of factor VIII deficiency, therapy with the above-mentioned preparation is discontinued, and stored blood or plasma is substituted.

In most cases of factor VIII or IX deficiency, levels of the missing factors must be maintained carefully for two to three weeks postoperatively. Even slight bleeding into the wound greatly complicates the problem of hemostasis and wound healing. The factor VIII or IX level should be monitored at least once daily by direct assay, determination of partial thromboplastin time, or prothrombin consumption test, in decreasing order of choice. Postoperative management of these patients requires close cooperation between hematologist, surgeon, and blood bank.

*Undefined Bleeding Tendency.*—Unfortunately, cases of apparent bleeding tendency are encountered at surgery which do not fit into a well-defined diagnostic category, at least on preliminary, emergency evaluation. These may include occasional cases of capillary defects, thrombocytopathy, thrombasthenia, von Willebrand's disease, deficient fibrin-stabilizing factor, acute factor V deficiency, or circulating anticoagulants. Management of these cases in an emergency will necessarily be somewhat empirical, since the diagnosis may be difficult to establish immediately. As in all the situations described, every effort should be made to avoid shock and hypoxemia. Blood transfusions should be given to maintain circulatory volume, and good oxygen exchange should be maintained. These measures are preferable to the use of pharmacologic agents for the maintenance of blood pressure, in most situations.

In the presence of undiagnosed, uncontrollable bleeding at the operating table, the following succession of therapeutic agents should be tried, based on safety, availability, rapidity of action, and likelihood of effectiveness: (1) fresh platelet-rich blood (1 to 3 units, as fresh as possible); (2) factor VIII-rich fibrinogen, 2 to 6 gm; (3) cryoprecipitated factor VIII, 2 to 4 units; (4) protamine sulfate, 1 mg/minute administered by intravenous drip; (5) hydrocortisone, 500 mg administered intravenously, or corticotropin (ACTH) drip, 5 units per hour administered intravenously; and (6) phytonadione; 25 mg administered intravenously. Although giving AHF-rich fibrinogen empirically is objectionable from the standpoint of transmitting homologous serum hepatitis, it nonetheless has the definite advantages of being rapidly and specifically corrective, if the patient is deficient in either of these factors; furthermore, as Stefanini and Dameshek state, "For reasons which are not clear, administration of fibrinogen seems to have a beneficial effect on many non-related types of purpura and hemorrhage." The beneficial effect, if any, of the fresh blood or AHF-rich fibrinogen should be clinically evident soon after their administration; the other agents require longer for effect.

*Postoperative Oozing of the Wound.*—Frequently, defects of hemostatic function are mild enough that they are not suspected at surgery, but become evident only as prolonged oozing of blood from the wound postoperatively. While less alarming than hemorrhage on the operating table, postoperative oozing requires effective management, since hematoma formation in the wound not only impairs healing and predisposes to infection, but complicates achievement of good hemostasis by specific therapeutic measures, especially in hemophiliacs. If the possibility of a defective ligature is excluded, and if a complete laboratory investigation, as previously described, fails to disclose a well-defined hemostatic defect which can be specifically corrected, one is forced to assume the presence of less well-defined entities, such as capillary defects, qualitative platelet defects, or circulating anticoagulants. The following empirically selected agents should be tried initially: (1) bed rest; (2) corticotropin drip, 50 units given over ten hours on successive days (or large doses of adrenal corticosteroid); (3) ascorbic acid; (4) phytonadione; and (5) thrombin administered topically on a gelatin sponge. Every effort should be made to prevent wound infection. If the above measures fail after 48 hours' trial, and the bleeding is a serious problem, infusion of 1 to 2 units daily of platelet-rich, fresh, blood or plasma is tried for several days, while continuing the previous measures.

## Summary

The emergency management of unanticipated defects of hemostatic function at surgery requires

immediate help from the laboratory. The tests required are not difficult to do, but should be readily available. Fresh blood or plasma with viable platelets fibrinogen, and cryoprecipitated factor VIII are cornerstones of specific therapy. Nonspecific therapy and ancillary measures also may be necessary.

Generic and Trade Names of Drugs

Hydrocortisone—*Cortef, Cortifan, Cortril, Hycortole, Hydrocortone.*
Aminocaproic acid—*Amicar.*
Phytonadione—*Aquamephyton, Konakion, Mephyton, Mono-Kay.*

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

# RECOGNITION AND MANAGEMENT OF SMOKE INHALATION

*James R. Webster MD, Margaret M. McCabe MD, and Mary Karp MD, JAMA 201(5):287/71–290/74, July 31, 1967.*

In spite of its significance, smoke inhalation has received little attention in the medical literature. Three representative case histories demonstrate the variable course of this entity and the need for individualized care of the victims. Of particular importance is recognition of the 6- to 48-hour latent period which may ensue before complications of acute bronchial obstruction, pneumonia, pulmonary edema, and eventual cardiopulmonary failure develop. Management may require tracheostomy, prolonged intermittent positive-pressure breathing with appropriate concentrations of oxygen and high humidity, and, when indicated, administration of systemic antibiotics and steroids. Frequent arterial blood gas measurements are essential for proper evaluation in these cases, both to delineate the status of the patients and to guide and determine the effectiveness of therapy. If victims of smoke inhalation can be managed through the acute phases of their illness, they often make a complete recovery.

Up to 1,200 deaths per year in the United States are attributed to smoke inhalation, and yet both current medical literature and recent textbooks dealing with emergency care of inhalation therapy make scant mention of this entity. As pointed out by Bates and Christie, this paucity of description is due primarily to lack of data rather than to lack of importance of the problem. Over a three-year period, in a 750-bed acute-care general hospital, 14 patients were admitted with this diagnosis, and an additional two patients were dead on arrival at the emergency ward. Ten of these 16 people had been exposed as a result of occupation (industrial or city firemen), and six as a result of other fires (five of these from cigarette smoking in bed). All were cared for by the authors. There was one death among the 14 hospitalized individuals. The representative cases described below emphasize the need for individualized care.

From the departments of medicine and anesthesiology Northwestern University Medical School and Chicago Wesley Memorial Hospital, Chicago.

## Report of Cases

Case 1 (Mild Disability).—A 34-year-old white fireman experienced an episode of syncope while fighting an industrial fire which produced large amounts of acrid smoke. Two hours later, he complained of tightness in the chest and dyspnea at rest. He had no prior pulmonary symptoms.

Examination revealed mild cyanosis; temperature, 99.6 F (37.6 C); pulse rate, 100 beats per minute; respirations, 24 per minute; and blood pressure of 150/100 mm Hg. The throat was diffusely reddened. There were expiratory wheezes and rhonchi throughout the chest. Findings from examination of the heart were unremarkable except that the second sound in the pulmonic area was accentuated. There were no peripheral signs of cardiovascular or pulmonary disease.

*Laboratory Findings.*—On admission, the hematocrit reading was 47 percent; white blood cell count (WBC) 12,100/cu mm, with 51 percent neutrophils; and venous carbon dioxide level, 27 mEq/liter. The electrocardiogram was normal, and the chest roentgenogram revealed mild accentuation of the vascular markings. The forced expiratory volume (FEV) was 2.9 liters, with a 1-second FEV of 1.9 liters.

*Hospital Course.*—Therapy included intermittent positive-pressure breathing (IPPB) for 15 minutes each hour, for which a pressure-regulated ventilator with air dilution was used, at a mask pressure of 30 cm $H_2O$, with a nebulizer solution of saline and isoproterenol (Isuprel) hydrochloride. Tracheal aspiration and antibiotics were also included in the therapy. Cyanosis subsided within four hours. Over the next two days, his fever declined, and the sputum decreased in volume and became white. By the fourth hospital day, the chest was clear upon physical examination, and the x-ray film of the chest had also cleared. The FEV was 4.9 liters, with a 1-second FEV of 4 liters. He was discharged to duty on the eighth hospital day.

This is a typical example of mild, temporary disability in a previously healthy individual. The manifestations were primarily those of acute, inflammatory bronchial obstruction and responded well to limited therapeutic measures.

Case 2 (Moderately Severe Disability).—A 52-year-old executive was hospitalized following his rescue by police from a burning hotel room. No other history was then available, but it was subsequently learned that he had been observed to have "asthma" and was a "heavy drinker."

On admission, he was stuporous with a temperature of 99 F (37.2 C); pulse rate, 80 beats per minute; respirations, 30 per minute; and blood pressure of 120/80 mm Hg. There was questionable cyanosis, and the mucous membranes of the nose and mouth were blackened. The neck veins were flat. There were diffuse inspiratory and expiratory wheezes and coarse breath sounds. The findings from the cardiac examination were unremarkable, with aortic and pulmonic second sounds of equal intensity. There were no localizing neurological signs.

*Laboratory Findings.*—On admission, the WBC was 15,310/cu mm, with 85 percent segmented and 5 percent unsegmented neutrophils. The levels of fasting blood glucose, blood urea nitrogen (BUN), and serum electrolytes were normal. Results of liver function tests were normal. The arterial pH was 7.51; arterial carbon dioxide pressure ($Paco_2$), 31 mm Hg; and arterial oxygen pressure ($Pao_2$), 81 mm Hg, while the patient was receiving oxygen by nasal catheter. The x-ray film of the chest was normal, and the ECG showed only nonspecific S–T segment changes.

*Hospital Course.*—He was placed on a regimen of continuous oxygen by nasal catheter at 5 liters/min, intravenous fluids, antibiotics, and IPPB for 15 minutes every two hours at a mask pressure of 25 cm $H_2O$, with a nebulizer solution of isoproterenol and saline. For the first 24 hours, his condition was stable, but on the evening of the second hospital day, an abrupt increase in dyspnea with cyanosis developed, together with a temperature of 102 F (38.9 C), and a respiratory rate of 45 per minute. On physical examination, moist rales were audible over the entire chest. The peripheral venous pressure was 230 mm $H_2O$, and he appeared moribund. The intravenous administration of steroids and aminophylline and continuous IPPB by mask with 100 percent oxygen, at a mask pressure of 35 mm $H_2O$, and digitalis administration resulted in some improvement. A chest roentgenogram showed extensive bilateral basal infiltrates. A tracheostomy was performed, and voluminous secretions were removed. Continuous respiratory assistance was maintained with the respirator set at a rate of 18 per minute, with a tracheal pressure of 25 cm $H_2O$. The inspired oxygen concentration was reduced to 45 percent, and the $Pao_2$ was maintained between 75 and 110 mm Hg, as measured by repeated arterial punctures. The patient slowly improved, although an acute organic brain syndrome complicated his course.

His respiratory status eventually improved so that the continuous ventilatory support could be gradually withdrawn. The IPPB treatments were administered for 15 minutes every one to four hours. When tracheal aspiration was no longer required and the vital capacity was more than three times the tidal volume, the tracheostomy tube was removed, and the IPPB treatments were given with a face mask, on an interrupted schedule.

This patient demonstrates the often-observed latent period following smoke exposure. He seemed relatively well until 36 hours after admission. Tracheostomy and IPPB appeared to be lifesaving. His acute toxic delusional state was probably due in part to his alcoholism.

Case 3 (Severe Exposure and Disability).—A 49-year-old unemployed executive was hospitalized after being found unconscious in his hotel room. The mattress was on fire, and he recalled smoking in bed following the ingestion of alcohol and sedatives. He had a long history of productive cough and moderate dyspnea on exertion. Six months previously, he had been hospitalized for pneumonia.

Examination revealed a disoriented, mildly cyanotic white man in moderate distress, with a respiratory rate of 30 per minute and a moist cough. The blood pressure was 130/80 mm Hg; pulse rate, 120

beats per minute; and temperature, 101 F (38.3 C). The hair was singed, and the oronasal mucosa was slightly reddened. The neck veins were flat. Breath sounds were distant, with expiratory wheezes and fine rales throughout the entire chest. All heart tones were distant, except for a loud second pulmonic second sound. The liver was palpable 3 cm below the right costal margin, and multiple spider angiomas were present over the chest.

*Laboratory Findings.*—On admission, WBC was 10,350/cu mm, with 27 percent unsegmented and 53 percent segmented neutrophils. Results of urinalysis and levels of BUN, fasting blood glucose, and serum sodium and potassium were normal. The venous carbon dioxide level was 32 mEq/liter, and the chloride, 89 mEq/liter. The sulfobromophthalein (Bromsulphalein) test showed 25 percent retention in 45 minutes. The arterial pH was 7.46, with a $Pao_2$ of 46 mm Hg and a $Paco_2$ of 33 mm Hg, while the patient was breathing room air. The ECG showed prominent P waves and sinus tachycardia. A chest roentgenogram was within normal limits.

*Hospital Course.*—During the first 24 hours in the hospital, increasing restlessness and cyanosis developed. The wheezing, coughing, and tachypnea increased, and copious secretions could be removed incompletely by oropharyngeal suctioning. He received procaine penicillin G; streptomycin; oxygen by nasal catheter at 5 liter/min; IPPB for 15 minutes every four hours with a ventilator on air dilution, at a mask pressure of 30 cm $H_2O$; aminophylline; and digitalis, with only minimal improvement.

On the second hospital day, his condition deteriorated further. The peripheral venous pressure was 245 mm $H_2O$, the $Paco_2$ had risen to 55 mm Hg, and the secretions were thicker. A tracheostomy was performed, and a volume-regulated respirator was used, at a tidal volume of 800 cc, a rate of 20 per minute, and an inspired oxygen concentration of 50 percent. There was an immediate decrease in cyanosis and in the temperature elevation. The confusion and hallucinations persisted, and the tracheal secretions were abundant. Nevertheless, the arterial blood gas measurements showed improvement by the third hospital day—pH, 7.46; $Paco_2$, 34 mm Hg, and $Pao_2$, 190 mm Hg, with the respirator at an inspired oxygen concentration of 30 percent. By this time, he had become oliguric, and the BUN level was 50 mg/100 ml. Thereafter, he showed slow but gradual improvement, and three weeks later his chest and mental state were almost clear. The tracheostomy tube was removed, and he was subsequently discharged without pulmonary symptoms, with an FEV of 4.1 liters and a 1-second FEV of 2.6 liters.

It is likely that this patient's underlying obstructive lung disease contributed to his turbulent course. In spite of the fact that he had received considerable exposure, the signs of thermal injury in the oropharynx were minimal. It has been pointed out that there may be little correlation between surface burns and the severity of the pulmonary damage due to inhaled smoke and fumes. The previous patient (case 2) had considerably more severe damage to his visible mucous membranes but had less severe respiratory complications.

In retrospect, the tracheostomy probably should have been performed shortly after admission in both cases. Certainly both patients would have died without the continuous ventilatory assistance and extensive tracheobronchial cleansing permitted by the tracheostoma. The arterial blood gas measurements were most helpful in following their progress and in guiding therapy.

## Comment

The above cases illustrate the variable clinical spectrum of smoke inhalation and emphasize the multiphasic natural history of the disorder:

1. There is a deceptively mild initial period in which the patient may be asymptomatic or have only mild bronchial obstruction and irritation.

2. Six to 48 hours later there may be a second phase, consisting of a sudden onset of bronchiolitis and pulmonary edema, with ineffective cough and severe airway obstruction. Retention of secretions, atelectasis, and diffuse bronchopneumonia result. Some of these changes may be secondary to impaired surfactant formation which follows damage to or hypoxia of the cells lining the alveoli. Necrosis of the bronchial mucosa, hyaline membrane formation, and interstitial edema are present in varying degrees, and the resultant low pulmonary compliance makes adequate ventilation difficult.

3. Acute right ventricular decompensation may also complicate the clinical condition at this time. If the patient survives, repair usually begins about two weeks following injury. In most instances, there is eventual return to an essentially normal functional level, without residual alveolar, airways, or vascular damage. On the other hand, chronic obstructive pulmonary changes occasionally result. It is of extreme importance that treatment be aggressive during the acute stage in order to maintain

adequate ventilation, cardiovascular compensation, and gas transfer, so that the greatest possible amount of clearing and healing can be established.

With acute chemical-smoke injury to the lungs, there is a potentially high mortality (up to 50 percent) as a result of the marked physiologic derangements. In particular, firemen (case 1) are likely to be exposed to all types of noxious fumes, and if the smoke contains oxides of nitrogen, cadmium, sulfur, or ozone, impairment may be quite extensive and persistent. Industrial exposures to toxic gases also occur in other occupational groups, such as welders and chemists, resulting in an acute pulmonary edema of chemical origin, which is in all respects similar to the syndrome which results from smoke inhalation.

Connor et al and Kleinfeld have outlined treatment programs which we utilized and which are similar to our recommendations discussed below.

Tracheostomy with a cuffed tracheotomy tube of adequate size is of great value in reducing morbidity and mortality, since it allows improved control of ventilation as well as frequent, convenient removal of secretions and maximum humidification. Although criteria for deciding when and if this should be done differ for each case, procrastination and temporizing should be avoided. Recent studies suggest that orotracheal intubation may be preferable to tracheostomy for some of these patients, especially for short-term management in the presence of head, chest, or neck burns.

Intermittent positive-pressure breathing, either continuously or on an interrupted schedule, is of tremendous importance. Pressures as high as 40 cm $H_2O$ may be required, due to the low pulmonary compliance, patchy atelectasis, and high airway resistance. These high inspiratory pressures may impair ventricular filling, and, if cardiac decompensation is imminent, further impede a decreased cardiac output. Inspiratory flow rates, therefore, should be high to keep the mean endothoracic pressure as low as possible. Close monitoring of vital signs for early evidence of impaired cardiac function is essential, and a catheter for measuring central venous pressure may be required.

High concentrations of oxygen may be needed initially for these patients if hypoxia is a major problem, but prolonged administration of high concentrations of oxygen may be damaging to the alveolar lining membranes and should be avoided, since oxygen toxicity by itself may produce extensive pulmonary damage, possibly by contributing to the previously described surfactant deficiencies. Frequent arterial blood gas measurements are required to follow these patients and are most useful for predicting complications and evaluating therapy. Specifically, the inspired oxygen concentrations should be such that the $PaO_2$ is 80 to 120 mm Hg, so that a normal oxygen saturation is maintained.

Humidification is of great importance in order to maintain good tracheal drainage. In our experience, aerosolized detergent solutions have not been superior to humidification by aerosolized normal saline, and in view of the potential hazards of nebulized detergents, enzymes, and alcohol solutions, there seem to be no clear indications for their use. We have found it useful to irrigate the tracheostomy tube with 2 to 8 cc of normal saline every one to four hours, because with such a routine, crusting and drying in the larger bronchi are prevented and secondary obstruction of the smaller conducting airways is uncommon. We have found the techniques of Bendixen et al to be most helpful in the management of the specific complications associated with long-term respiratory care. Nebulized bronchodilators may be of value, especially early in the course of the illness, but they should not be given with *continuous* assisted ventilation, because of the risk of cumulative drug side-effects.

The origin of the acute cardiac failure that these patients manifest is unclear. It responds poorly to the usual measures for pulmonary edema, such as administration of digitalis and morphine, and in the patients we have observed, the signs were those of biventricular decompensation. Oxygenation and optimum improvement in the pulmonary problem seem to be of primary importance in the treatment of this complication.

We believe that steroids reduce the acute tissue inflammation with its vasculitis, necrosis, and edema, and should be used early in patients with a history of extensive exposure to smoke or antecedent pulmonary problems. The indications for the use of antibiotics in these patients are unclear. Studies indicate that in other somewhat similar circumstances their prophylactic administration is unwarranted. Frequent sputum cultures should certainly be obtained, and changes in the predominant organisms may be useful in decisions regarding starting and/or changing antibiotic therapy. Postural drainage associated with chest percussion, frequent changes in position, and adequate humidifi-

cation are of value in accelerating bronchial clearing.

As with all clinical problems, individualization of treatment and soundly determined judgments are the crucial factors in the management of these patients.

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

# ACUTE PANCREATITIS AFFECTING THE TRANSVERSE COLON

## REPORT OF A CASE

*CDR William M. Lukash, MC USN, and LCDR Robert P. Bishop, MC USN, Amer J Dig Dis 12(7):734–736, July 1967.*

Release of activated enzymes in acute pancreatitis results in inflammatory and suppurative lesions on continguous structures, usually with associated fat necrosis. Local complications of abscess and pseudocyst are well known, but less evident are the infrequent peripancreatic inflammatory reactions causing massive ascites, splenic rupture, and colonic lesions. To exemplify the complication of an extrinsic lesion of the transverse colon which simulated both segmental ulcerative colitis and carcinoma, the following case is reported.

### Case Report

A 29-year-old white man was admitted to a naval hospital because of severe epigastric pain and vomiting of 24-hr. duration. The episode was precipitated by an excessive consumption of alcohol. He denied any hematemesis or melena, and there was no history of previous epigastric discomfort.

The histories of the patient and his family, as well as a review of his personal habits (other than moderate consumption of alcohol) were unremarkable.

*Physical Examination.*—At the time of admission, the patient appeared acutely ill, anxious, and diaphoretic. He was moderately obese for his height—weighing 215 lb. His oral temperature was 101.6° F., pulse 130, and blood pressure 166/110. The pertinent physical findings were limited to the abdomen. Marked guarding and slight abdominal distention were evidenced. Tenderness was most severe in the right upper quadrant and rebound was manifested. Bowel sounds were present, but hypoactive.

*Laboratory Studies.*—The serum amylase and lipase were markedly elevated. The initial hemoglobin was 17 gm./100 ml.; the hematocrit was 51%. The white blood count was 21,000 with 92% neutrophils. An initial blood sugar was elevated. Findings of liver-function tests were normal except for a slight, transitory elevation of the serum glutamic oxalacetic transaminase (SGOT) -92 U. On the initial chest and flat-plate abdominal radiographs there was blunting of the left costophrenic angle and a localized ileus of the duodenal loop.

*Clinical Course.*—On the basis of the clinical evidence and findings of elevated serum amylase, the patient was considered to have acute pancreatitis. He was treated with a rest program which utilized nasogastric suction and intravenous fluids including salt-free albumin, antibiotics, and anticholinergic agents. The patient responded to this regimen in 48 hr. and remained asymptomatic.

Upper-gastrointestinal radiographic study revealed "padding" with pressure defects along the medial aspect of the duodenal loop. The associated duodenal mucosa was distorted and the loop was expanded. This was consistent with the clinical diagnosis of acute pancreatitis. On cholecystogram there was a well-opacified, normal-appearing gall bladder. Radiographic examination of the colon, performed 2 weeks after the onset of the patient's illness, revealed an area of narrowing with associated mucosal irregularity in the hepatic flexure. Follow-up X-rays 2 weeks later demonstrated a persistent filling defect in the area. Further studies regarding this colonic lesion, including repeat liver function tests and results of a radioisotopic $^{198}Au$ liver scan, yielded normal findings.

From the Gastroenterology Branch, Department of Internal Medicine, Naval Hospital, National Naval Medical Center Bethesda, Md., and the Gastroenterology Branch, Department of Internal Medicine, Naval Hospital, Philadelphia Pa.



The authors wish to thank Frances H. Atkinson, B.A., R.R.L., for technical editing.

Because of the persistent defect in the hepatic flexure noted during the barium-enema examination, an exploratory laparotomy was performed. A firm inflammatory mass was found adherent to the wall of the hepatic flexure and the liver. Upon incision, it was found to contain yellow necrotic material resembling fat necrosis. The pancreas was palpated and, though it was somewhat firm, it was considered to be normal. No other areas of fat necrosis were noted. A follow-up barium-enema examination 6 weeks later showed entirely normal findings, and the patient remained asymptomatic throughout the remainder of his hospital stay.

## Discussion

This case is an example of a disease of pancreatic origin involving the transverse colon. The first barium-enema examination suggested an area of segmental ulcerative colitis with narrowing and mucosal irregularity. A second barium enema, however, showed a filling defect with the typical shelving aspect of a primary carcinoma. At laparotomy a residual inflammatory process with fat necrosis was seen; it produced an extrinsic defect in the hepatic flexure of the colon.

An invasion by the pancreatic inflammatory process into the mesocolon resulted in collapse of a continguous localized segment of transverse colon from encroaching edema. Had a plain flat-plate radiograph of the abdomen been obtained at this stage of the disease, radiolucent air on both sides of the collapsed colonic segment would appear as the "cut-off sign" of acute pancreatitis (as described by Stuart[1]).

A constricting colonic lesion greater than 2 in. in length is more likely to be extrinsic, resulting from pressure from inflammatory reactions or metastatic masses, rather than a primary carcinoma.[2] Mucosal destruction and a shelving defect, on the other hand, suggested a primary neoplasm. However, the area of collapsed transverse colon expanded and the observed filling defect was produced by the residual mass resulting from the pancreatitis and fat necrosis.

## Summary

A case is reported in which a lesion of the transverse colon was caused by acute pancreatitis. A deformity in the hepatic flexure of the transverse colon was noted from barium-enema examination and it simulated a segmental ulcerative colitis or a carcinoma. This deformity was attributed to an inflammatory mass with fat necrosis, which resulted from corrosive enzymes released from the pancreas.

U.S. Naval Hospital
National Naval Medical Center
Bethesda, Md. 20014

### References

1. Stuart, C. Acute pancreatitis; preliminary investigation of a new radiodiagnostic sign. J Fac Radiol (Lond.) 8:50, 1956.
2. Kent, K. H. Extrinsic lesions affecting the transverse colon. Amer J Roentgen 8:779, 1963.

# DIAGNOSIS AND TREATMENT OF SCABIES

*Alan Lyell,* MD, FRCPE, MRCPG, Brit Med J 2(5546):223–225, April 22, 1967. Reprinted from the British Medical Journal, by permission of the Author, Editor and Publishers, B.M.A. House, Tavistock Square, London, W.C. 1.*

The diagnosis of scabies depends on finding a burrow or one stage of the parasite. The search is prompted by clinical suspicion and informed by knowledge of the natural history and habits of the human *Acarus scabiei*. The best source of this information is the monograph by K. Mellanby.[1]

Scabies should be considered in every patient who complains of persistent itch. In typical cases the diagnosis is easy, but when the visible changes are unusual or slight, scabies may not be suspected. It is important to think of the itching patient in relation to his environment, and if another member of the household is itching too there is a prima facie case for suspecting an infestation.

## Natural History of the Acarus

The clinical manifestations of scabies are the eventual result of an impregnated female acarus establishing her home in the skin. The host usually picks her up by close physical contact with a case of scabies, but infected bedding and clothing can sometimes transmit the disease. She chooses the site

* Consultant Dermatologist, Royal Infirmary, Glasgow.

for her home and then she elbows, bites, and digs her way into the skin, tunnelling within the stratum corneum, but not penetrating any deeper. Unless the patient has had scabies previously he does not itch at this stage, and the burrow is inconspicuous. The acarus tunnels on slowly (0.5 to 5 mm. per day) leaving a trail of eggs and faeces behind her in the burrow. The eggs hatch into larvae, which develop first into nymphs and then into adults, the process taking about two weeks altogether.[1] The immature forms leave the burrow and take shelter in the hair follicles. The mature males are said to make short burrows, but they are rarely seen and probably spend a restless life in search of love. The proof of their successful activity is the new generation of impregnated females which start burrows of their own. About one month after the establishment of the first burrow the patient begins to itch, probably because he has become sensitized to acarine products. The patient now realizes that something is wrong with his skin, he begins to scratch, and, according to Mellanby, the scratching limits the population of acari by depriving them of a roof over their heads, and perhaps by crushing them as well. In experimentally induced infections Mellanby found that the population reached a maximum within three months, and varied from 20 to 400 egg-laying females. Examination of nearly 900 men with scabies showed a mean population of 11.3 adult females, and that more than half the patients harboured five adult female acari or fewer. It is generally believed that infestations do not clear spontaneously, but Mellanby hints that they may do so rarely.

## Clinical Features

The patient comes to the doctor complaining of severe generalized irritation, made worse on getting warm, particularly on going to bed. Examination reveals an eruption which has two aspects: the burrows, which have to be searched for, and changes resulting from sensitization and secondary infection, which are obvious. There is a widespread rash of follicular papules, usually well marked on the abdomen, buttocks, the inner sides of the thighs, and the axillary folds. Scratch marks, crusted papules, and pustulation demonstrate the effects of scratching. The burrows are found on the sides and webs of the fingers, on the ulnar border of the hands, on the volar aspect of the wrists, on the points of the elbows, on the axillary folds, on the areolae of the nipples in women and on the external genitalia in men, on the buttocks, and on the margins or soles of the feet. Mellanby found the hands and wrists the favourite site (63 percent of the acari recovered came thence), the extensor aspect of the elbows being next (10.9 percent). The undisturbed burrow is a thin, wavy, dark hairline about 1 cm. long. It begins at a funnel-shaped opening bordered by ragged edges of the stratum corneum, and ends in a white, shiny dot, quite perceptible to keen naked eye observation, which is the acarus. A vesicle often develops just behind the acarus in the floor of the burrow. The sites of old burrows are marked by ragged edges of the stratum corneum that have formed their walls, and a crust representing the dried vesicle. An infective eczema is apt to develop on the fingers and wrists and at many other sites, including the nipples and the glans penis. Although it can mask the burrows its presence suggests the possibility of scabies to the trained eye. Patients who are unable to scratch do not develop septic lesions or eczema.

## Diagnosis

The clinical picture, comprising an itchy patient, papules, vesicles, and pustules on the fingers and wrists, and follicular papules on the trunk, should be regarded as due to scabies unless proved otherwise. The diagnosis is probable if other members of the household are affected, and is put beyond doubt if a burrow can be found or acarine material can be identified. The best method is to scrape an entire burrow along its length with the edge of a scalpel blade held at right angles to the skin. The material is transferred to a microscope slide, mounted in a drop of liquor potassae, warmed gently, and examined under the low power of the microscope. The acarus has a body shaped like a tortoise, with two pairs of legs in front and two pairs behind. The fore-legs end in "suckers" and the hind-legs in long bristles. She is about 400 microns long, and coated with short bristles. If you find her you will confirm the diagnosis, and if you let the patient look down the microscope you will ensure his co-operation. If you fail to find the acarus you may find eggs or immature forms. It may be necessary to examine more than one burrow.

The typical picture is subject to a number of variations. Infants develop burrows on the head as well as on the hands and feet, which will have implications for treatment. Cleanly people usually develop few lesions, which is one reason why the disease is apt to be missed in nurses. A heavy, helpless patient with an undetected infestation, who requires a lot of lifting, can start an epidemic among the nursing staff before anyone realizes

what has happened. It is amazing how scabies can remain undetected under such aliases as "reaction to irradiation," "drug eruption," and "neurodermatities." The blisters that develop in the floor of the burrows can mimic cheiropompholyx, and the infective eczema that has been mentioned already can dominate the picture. The eczema may sometimes be limited to the nipple area or glans penis and form the presentation of the disease. Sometimes the infective element predominates and produces folliculitis, boils, cellulitis, or lymphangitis. The amount of itch varies from torment to little or none. Absence of irritation is believed to account for the thousands of acari that abound in Norwegian scabies. The manifestations of this rare form resemble an exfoliative dermatitis. The diagnosis usually has to await an epidemic of scabies being traced to the patient, but on one occasion it was made by post when a skin biopsy from a puzzling case was received by a distant laboratory, and sections showed the stratum corneum almost as full of mites as the rind of a Stilton cheese.

Differential Diagnosis

The disease most commonly confused with scabies is *eczema,* particularly atopic eczema (Besnier's prurigo). The itch can be as intense, it has the same quality of being aggravated by heat (a feature that occurs in most forms of pruritus), and appropriate lesions may be seen on expected areas such as fingers, wrists, nipples, and trunk. Burrows will not be seen, but a linear arrangement of eczema vesicles can give an impression of a burrow. Other members of the family may suffer from atopic eczema. Occasionally one needs to observe the patient for a time and to try the effect of treatment before being certain that scabies is not present. The infective eczema that is a part of scabies is betrayed by the distribution of the lesions.

*Papular urticaria* causes intense irritation in young children. The child finds relief in scratching the papules that occur on the trunk and limbs, causing them to ooze or bleed. The exudate dries into crusts and secondary infection often ensues. No burrows occur and urticarial lesions predominate, but scabies can be imitated closely in other respects, including the occurrence of more than one case in the family. Many cases of papular urticaria can be traced to the bites of insects, among which fleas, bedbugs, and the parasites of birds, cats, and dogs are important. The patient gets better in hospital because the parasites are left at home, whereas a scabetic child brings its own parasites with it and the change of environment does not influence the eruption.

Human disease resulting from contact with *animal scabies* is a special example of papular urticaria. Most animals harbour their own brand of acarus, which is indistinguishable morphologically from the human acarus, but is incapable of burrowing and breeding in any species but the one for which it has become adapted. The dog is no exception, and his acari cause an itchy papulovesicular eruption in master and his household. Dog scabies is called sarcoptic mange. This interesting and often overlooked cause of skin trouble has been reviewed recently by Smith and Claypoole.[2] The mangy dog, like the patient with Norwegian scabies, harbours thousands of acari, which explains the liberality with which he scatters them in his environment. *Cheese mites* can be acquired in much the same way by those who hump cheeses on their shoulders or handle them, and they cause local irritation at the sites of contact. Many other commodities such as *grain*, *straw*, and *copra* can harbour parasites that irritate the people handling them, and the part played by vegetation in providing a home for the *harvest bug* is well known. No burrows are found in any of these diseases.

*Pediculosis corporis* produces intense itch and pigmentation of the trunk and proximal parts of the limbs. The lice and their eggs should be sought in the underclothes. *Pubic lice* can be overlooked or mistaken for small blood crusts. The lice and the eggs are attached to body hair. *Parasitophobia* is a delusion of infestation. The patient describes feeling insects crawling beneath the skin. Examination reveals traumatic lesions, some having small darkish crusts, the result of excavating with the finger nails or other digging implement. Often a small crumpled piece of paper is produced for the doctor's inspection containing "the parasites"; it holds a collection of heterogeneous debris such as scales, crusts, bits of hair, crumbs, nail clippings, sugar grains. The proffering of such pathetic "evidence" of infestation is not a sufficient reason for excluding an infestation. Compulsive picking, pinching, or scratching of the skin occurs without delusions of infestation and gives rise to difficulty in the physician's mind because there is no proper name for it. Neither *neurodermatitis* nor *dermatitis artefacta* is really apt. It is easy to invoke emotional factors as a cause of itch, but before doing so it is wise to make sure that the patient is under an appreciable emotional strain, and also that physical causes such as *lymphadenoma, drug reactions, occult neoplasms*, and *renal disease* can be excluded.

Most cases of *dermatitis herpetiformis* that come to our clinic have been treated for scabies. Der-

matitis herpetiformis produces groups of lesions on favourite sites such as scalp, shoulders, buttocks, hips, and elbows, but it usually spares the hands. The histological picture of an early papulo-vesicle is characteristic, and a response to sulphapyridine or dapsone can be anticipated.

*Biliary cirrhosis,* pruritic eruptions of *pregnancy,* a toxic reaction to the *contraceptive pill,* and *onchocerciasis* are rarer causes of persistent severe itch and papules that can be confused with scabies.

## Treatment

The three essential things to do are to treat all the members of the household simultaneously, to use an application that will kill the parasites, and to apply it to the whole skin area, not merely the spots. Failure can be traced to neglect of these injunctions.

Simultaneous treatment is important, because unless the infestation can be stamped out in the household at one blow, the untreated members will reinfect the treated ones sooner or later. "The household" includes everyone living in the house and extends to outsiders who are involved in regular close physical contact with members of the household, such as the relative or the neighbour who pops in every day.

Sulphur ointment *B.P.* and benzyl benzoate application *B.N.F.* will kill the parasites. They are used half strength for older children and quarter strength for babies. Sulphur has a reputation for being more certain and less likely to cause irritation. Benzyl benzoate is pleasanter to use and is kinder to the clothing. Mellanby[1] believed them equally effective. The medicament is applied to the whole skin surface excluding the scalp and face (except in babies, whose heads must be treated) particular care being taken to see that no fold or crease is missed, and that the soles of the feet are included. Applications are made on three successive nights. A bath before the first application and another on the night after the last application is desirable. Wherever possible clean underclothes and clean sheets should be used after the last bath. The treatment seldom irritates the skin provided that it is limited to three applications. Patients should be warned that it is natural for the itch to persist for some weeks after treatment. Local treatment at this stage should be confined to simple applications such as calamine lotion. Many "failures of treatment" that we see are no longer infested, but have a sulphur or benzyl benzoate dermatitis often brought on by the patients continuing these medicaments contrary to instructions, in the belief that because they are still itching they are still infested. Unless a live acarus is found it is impossible to decide whether the patient is still infested in the weeks succeeding treatment. Failure of thoroughly applied treatment is equivalent to reinfection, and in this event one must search for the member of the household who has been overlooked. The anti-scabetic treatment takes precedence over local treatment of any sepsis. Oral antibiotics should be used concurrently to treat severe pyogenic infection or lymphangitis. When the mites have been dealt with, any residual infection can be treated, for example with an antibiotic-corticosteroid local application. Attempts to deal with the sepsis before tackling the infestation will not succeed. Scabies can be transmitted by sexual contact, and it is advisable to bear in mind the possibility of associated venereal disease.

The efficient treatment of scabies in a community is a matter of knowledge and organization. It should be possible to eradicate such an easily treatable infestation, and its continued existence in Britain should be a cause for concern. The provision of treatment centres would help the general practitioner, who is the person in the best position to deal with the problem. The advantages of treatment centres would be that a routine of thorough treatment could be provided which would virtually guarantee that the person treated would be cured of his infestation; furthermore, such a centre would be a means of treating the rest of the household, an operation which is difficult at present. As long as the success or failure of treatment continues to lie so largely in the hands of the patient, so long will scabies continue.

## Summary

The diagnosis of scabies is a matter of suspicion backed up by knowledge. Suspect every patient with a persistent itch, particularly if other members of the family are affected. Accurate diagnosis requires the discovery of a burrow or acarine products. Benzyl benzoate application or sulphur ointment should be applied to the whole skin surface below the chin on three occasions only. Every member of the household is treated simultaneously. Without treatment centres this preventable disease is likely to remain endemic.

The author acknowledges gratefully the helpful criticism of Drs. J. O'D. Alexander, T. Cochrane, and T. A. Pasieczny.

### References

1. Mellanby, K., *Scabies,* 1943, London.
2. Smith, E. B. and Claypoole, T. F., *J. Amer. Med. Ass.,* 1967, 199, 59.

# HOSPITAL SHIP PSYCHIATRY IN A WAR ZONE

*LCDR Robert E. Strange, MC USN, and CDR Ransom J. Arthur, MC USN,*
*Reprinted from the American Journal of Psychiatry, volume 124, pages 281–286, 1967.* 

*The hospital ship Repose has provided combat-supportive medical services for U.S. forces in Viet Nam, receiving casualties evacuated directly from the field medical units as well as patients referred from the major hospitals ashore. Although the psychiatric patient population aboard was similar in many respects to that at any other military hospital, the function of the ship as an intermediate echelon of psychiatric treatment in the war zone helped to maintain a strong back-to-duty orientation, and 50 percent of all psychiatric patients taken aboard were returned to full duty.*

In February 1966 the U.S. Navy hospital ship *Repose* arrived off the coast of Viet Nam and began to furnish medical services for Marine and Navy forces in the I Corps area. This vessel was the first and until recently the only hospital ship to function in a combat support role since the Korean War, and her unique situation furnished opportunity for a specialized study of psychiatric problems in the U.S. military population involved in the Southeast Asian conflict. This report details the results of a study of demographic variables and clinical features of psychiatric patients hospitalized aboard *Repose* during the initial seven months of her operations off Viet Nam.

## Clinical Facilities and Personnel

The psychiatric unit aboard the *Repose* was a single ward in two adjoining compartments, containing a total of 48 functioning beds. The staff consisted of one psychiatrist, one psychiatric nurse, and nine hospital corpsmen. This was an open, unlocked unit, and the patients were allowed freedom of movement about the ship commensurate with their degree of illness and responsibility. Treatment methods included individual and group psychotherapy and medications. No somatic therapy was done, and psychotherapy was generally of a short-term type.

The patient census fluctuated greatly. Usually there were 12 to 15 patients on the ward, although on occasion the census rose to as high as 35. Length of hospitalization was also quite variable, ranging between extremes of overnight and 60 days. Over a representative two-month period the mean length of inpatient care was 13.5 days.

## Operational Patterns and Intake Sources

During the seven-month period under consideration the *Repose* served two medical functions. Much of the time she steamed in a scheduled pattern and received patients sent from the major hospitals ashore. These patients were referred by specialists at those facilities, who had initially received them from the field medical units. The ship then functioned as a third echelon of treatment. Frequently, however, the ship furnished direct combat support of Marine operations, and casualties were evacuated directly from the medical units in the field with no previous specialty evaluation, thereby placing the *Repose* in the role of second echelon treatment.

Of the 143 psychiatric patients in this study, 77 (54 percent) were referred for hospitalization by psychiatrists stationed ashore with the Marines. Sixty-six (46 percent) arrived aboard with no initial psychiatric contact elsewhere. One hundred and five Marine and 38 Navy personnel were psychiatric inpatients during this period, the Navy patients coming from both units ashore and ships operating in the area. Repair and upkeep of the ship necessitated several lengthy departures from the war zone during this initial period of operations. Because of all these factors there was great fluctuation in rate of patient intake, but while on station there was a mean admission rate of 1.7 per day.

## Method

For purposes of study demographic variables were extracted and compiled on the initial group of 143 hospitalized patients, particularly in relationship to diagnostic categories. Three basic categories were utilized: character and behavior disorder, psychoneurotic reaction, and psychotic illness. In the total patient population 67 percent were classified as character and behavior dis-

LCDR Strange is Assistant Chief of Psychiatry, Naval Hospital, Philadelphia, Pa. 19145, and CDR Arthur is Officer in Charge, Navy Medical Neuropsychiatric Research Unit, San Diego, Calif. 92152.

TABLE 1. *Distribution by Age and Diagnostic Categories of 143 Psychiatric Patients Admitted Aboard USS Repose*

| AGE (Years) | CHARACTER AND BEHAVIOR DISORDER | | PSYCHO-NEUROTIC DISORDER | | PSYCHOTIC DISORDER | | TOTAL | |
|---|---|---|---|---|---|---|---|---|
| | Number | Percent | Number | Percent | Number | Percent | Number | Percent |
| 46-50 | 1 | 1.0 | | | | | 1 | .7 |
| 41-45 | | | 1 | 3.6 | | | 1 | .7 |
| 36-40 | 1 | 1.0 | 2 | 7.1 | | | 3 | 2.1 |
| 31-35 | 1 | 1.0 | 3 | 10.7 | 2 | 10.5 | 6 | 4.2 |
| 26-30 | 5 | 5.2 | 4 | 14.3 | 2 | 10.5 | 11 | 7.7 |
| 21-25 | 36 | 37.5 | 7 | 25.0 | 10 | 52.6 | 53 | 37.1 |
| 17-20 | 52 | 54.2 | 11 | 39.3 | 5 | 26.3 | 68 | 47.5 |
| Total | 96 | 100.0 | 28 | 100.0 | 19 | 100.0 | 143 | 100.0 |
| Mean age | 21.4 | | 25.0 | | 22.6 | | | |

order, 20 percent as psychoneurotic, and 13 percent as psychotic (Table 1).

## Results

*Character and behavior disorder.* These patients had a mean age of 21.4 years. A disproportionate number were in the pay grade of E–2 (private first class or seaman apprentice). The sample was characterized by short length of service. Sixty-five percent were unmarried. Excited, agitated, or violent behavior was noted in 45 percent of these cases. A history of civilian and military disciplinary problems tended to be characteristic of this group. Sixty-three percent of these men had been in active combat, and in 49 percent combat was judged to be a major factor in precipitating hospitalization.

*Psychoneurotic disorder.* These patients had a mean age of 25 years and were characteristically in the pay grade of E–4 and above with more than three years of service. Forty-eight percent were unmarried. Sixty-one percent reported somatic complaints, and 54 percent had significant depressive symptoms. Only 18 percent had a history of agitation or violence. Seventy-nine percent had been in combat, and this stress was felt to be a major precipitating factor in the hospitalization of 47 percent of this group.

*Psychotic disorder.* These patients had a mean age of 22.6; a relatively high proportion were in the pay grades of E–3 and E–4. Seventy-seven percent were single, an apparently disproportionate number after age differences were taken into account. Ninety percent manifested overt thought disorders, and 63 percent had perceptual disturbances to the point of hallucinations. Thirty-two percent had paranoid ideation; 53 percent showed hostility by suspiciousness, irritability, or menacing behavior; and 18 percent had a history of excited or violent behavior. Sixty-three percent were apathetic and withdrawn.

Of this group only 32 percent had been in combat, and combat was judged to be a significant precipitating stress in only 16 percent.

*Other variables.* Suicidal attempts and threats were relatively infrequent in this sample (eight percent), as were homosexual problems or concerns (four percent). Only six patients reported homosexual preoccupations, and three of these were psychotic.

In comparing the three diagnostic categories there were no significant statistical differences in region of birth, previous psychiatric contacts, or educational level. Forty-three percent of the total sample had completed high school. There were no differences in length of time in the war zone and no differences in proportions of Navy and Marine Corps personnel by diagnosis.

Thirty percent of the total sample reported significant marital problems with no diagnostic differentiation. Combat wounds were judged to be a major precipitating factor in six percent, and these occurred mostly in the character and behavior disorder group.

## Treatment and Disposition

Of the character and behavior disorder group 54 percent received medications during the course of their hospitalization. Fifty-two percent were eventually returned to full duty, 37 percent were evacuated for medical administrative action, and 11 percent had some other form of disposition (Table 2).

Eighty-two percent of the psychoneurotic patients were treated with drugs. Seventy-five percent were returned to full duty and 25 percent were evacuated out of the combat zone.

Of the psychotic patients, 90 percent were treated with medications and all were evacuated

TABLE 2. *Distributions for Disposition and Diagnostic Categories of 143 Psychiatric Patients Admitted Aboard USS Repose*

| Disposition | Character and Behavior Disorder | | Psycho-Neurotic Disorder | | Psychotic Disorder | | Total | |
|---|---|---|---|---|---|---|---|---|
| | Number | Percent | Number | Percent | Number | Percent | Number | Percent |
| Full duty | 50 | 52.1 | 21 | 75.0 | | | 71 | 49.6 |
| To duty recommending administrative action or discharge | 4 | 4.2 | | | | | 4 | 2.8 |
| Transfer to naval hospital | 35 | 36.5 | 7 | 25.0 | 19 | 100.0 | 61 | 42.7 |
| Return to limited duty | 1 | 1.0 | | | | | 1 | .7 |
| Desertion | 1 | 1.0 | | | | | 1 | .7 |
| Other | 5 | 5.2 | | | | | 5 | 3.5 |
| Total | 96 | 100.0 | 28 | 100.0 | 19 | 100.0 | 143 | 100.0 |

to hospitals in the United States for additional treatment and disposition.

## Discussion

The above figures indicate that the majority of psychiatric patients treated aboard the *Repose* in the war area were similar to those in other military hospitals. For example, the proportions of major diagnostic categories among 470 patients at a large naval hospital in the United States were: character and behavior disorders, 69 percent; psychoneurotic, 23 percent; and psychotic, eight percent. Character and behavior disorders predominated, as is true of the general military psychiatric population. For purposes of this initial study the personality disorder category included those with the diagnosis of situational reaction.

This sample of combat zone patients presented certain unique characteristics, however. Combat stress was judged to be a major factor in precipitating symptoms in 47 percent of the psychoneurotics and in 49 percent of the character and behavior disorders. It appeared that many of these patients might have avoided hospitalization a higher rate than that of hospitals geographically removed from the war area.

It is well known that the farther a psychiatric casualty is removed from combat, the more difficult it is to return him to duty. The position of the hospital ship psychiatric facility is that of an intermediate echelon of treatment between the field and the out-of-theater hospital. Because of geographical and psychological involvement in the combat zone a number of patients who require longer care than can be given ashore can be treated on the ship and returned to their units, whereas if they had been evacuated out-of-country the likelihood of return to duty is much less. Such salvage of combat manpower is, of course, a primary mission of military psychiatry.

A strong back-to-duty orientation was maintained on the ward. Those patients, primarily psychotic, who obviously were going to have to be evacuated were usually transferred after four or five days of treatment designed to control their florid psychosis. An attempt was made to have patients who were to return to duty in the majority on the ward at any one time.

Group therapy sessions, frequently led by hospital corpsmen who are neuropsychiatric technicians, were held daily. In both individual and group therapy, discussion of combat of course predominated and it was difficult to get beyond this topic in therapeutic work. Ventilation, discussion, suggestion, persuasion, and support were the major therapeutic devices employed. All the discussions were strongly reality oriented. War is all too real and one cannot escape from reality—this point was brought home repeatedly.

Drug therapy was also employed with good success. This is the first war since the introduction of phenothiazine drugs, and they proved to be very useful indeed. Any acute combat syndrome—almost regardless of symptoms, including acute agitated depression, anxiety reaction, hysterical episodes, and psychosomatic problems—seemed to be largely ameliorated within 48 hours by the use of very heavy doses of chlorpromazine coupled with nighttime sodium amobarbital sedation. After 48 hours, medication could be drastically reduced or even stopped and psychotherapy begun without relapse.

A major problem in ward management, not encountered to so great a degree in ordinary hos-

pital practice, was the extreme difficulty in finding useful tasks to keep the patients occupied after the initial stage of hospitalization had passed. The patients frequently had too much time to brood, and this idleness seemed to increase their anxiety.

The general ward atmosphere was different in several ways from that prevailing in Navy hospitals within the United States. In the first place, antisocial or acting out behavior, so common in a peacetime ward with character disorder patients, was virtually absent on the hospital ship. Secondly, the general tone of the ward was that of marked depression, much more so than in an ordinary Navy psychiatric ward. This depressed ambience seemed to be due to the large number of depressed patients (a characteristic Viet Nam psychiatric syndrome) and to the pervading sense of returning to combat and possible death or mutilation. Underneath the depression was a strong undercurrent of hostility, which taxed severely the psychiatrist's own emotional resources as well as those of his staff.

A common patient type encountered on the wards was a squad leader, particularly one in the grade of corporal. It seemed difficult for some of these young men, still in late adolescence, to handle the grave challenge of being responsible for other men's lives. Unlike the older officers and more senior noncommissioned officers, their own maturation had not progressed far enough to make the burden of leadership tolerable.

There seemed to be two peaks for psychiatric disability: one after two or three months, when the immature personalities or character and behavior disorder individuals collapsed, and the other at approximately 10–11 months, when the anxious, neurotic but highly conscientious Marine might develop incapacitating symptoms. The closer he approached to the 13-month rotation date the more obsessively convinced the individual became of the imminence of his death. In some individuals, particularly corpsmen, the fear of gross mutilation was greater than that of death itself.

However, in spite of all the stress in the war zone, the rate of psychiatric disability in Viet Nam has been remarkably low for all the armed forces, less than in either World War II or Korea. Many reasons have been advanced to explain this; among the most cogent are a limited, finite tour of duty, intermittent rather than continuous combat exposure, and a high sense of purpose and commitment on the part of the individuals facing combat.

Finally, it must be said that the military psychiatric lessons of 1918, 1943, and the Korean War have been well learned by the young psychiatrists in the Viet Nam theater. They know full well how medical intervention and facilities in the past often encouraged regression and invalidism, and they are imbued with a sense of therapeutic zeal and optimism which is a potent force in the prevention of chronic psychiatric disability.


### Summary

A report of psychiatric experience aboard the U.S. Navy hospital ship *Repose* has been presented. A survey of 143 psychiatric cases admitted during the ship's initial operations in the Viet Nam combat zone from February through August 1966 was recorded. Sixty-seven percent of these patients were classified as character and behavior disorders, 20 percent as psychoneurotic, and 13 percent as psychotic. Similarities and differences on demographic variables were presented and discussed. Fifty-two percent of the character and behavior disorder patients and 75 percent of the psychoneurotic patients were returned to full duty.

The role of the hospital ship as an intermediate echelon of psychiatric treatment in the war area was described.


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

# MEDICAL ABSTRACTS

## LOGISTICS OF VIRAL DIAGNOSIS

*M. Michael Sigel, PhD FAPHA, Amer J Public Health 57(8):1341–1356, Aug 1967.*

This paper contains a condensed version of a symposium presented at the Annual Meeting of the American Public Health Association in Chicago, 1965. The material has been organized and somewhat rephrased to insure a logical plan and continuity. In view of the large advances in virology during the past decade, this review of problems is of considerable significance.

## AMYLOIDOSIS

*Alan S. Cohen, MD, New Eng J Med 277(10): 522–530, Sept 7, 1967 (Part 1) and 277(12): 628–638, Sept 21, 1967 (Part 2).*

In the past 10 years, interest in this disorder has increased because it clearly has a wider clinical

significance than had been appreciated. It occurs not only in association with chronic infections and chronic inflammatory diseases but also in association with a surprising variety of tumors. In addition, in this era of pre-eminence of genetically determined diseases and their metabolic accompaniments, several varieties of heredofamilial amyloidosis are described.

These articles draw together and analyze what is currently known about the diagnosis, natural history and treatment of a wide variety of clinical "amyloid syndromes" after a brief review of what is known about the constitution and genesis of amyloid.

### ENVIRONMENTAL FACTORS OF CANCER OF THE COLON AND RECTUM

*Ernest L. Wynder, MD and Takao Shigematsu, MD, Cancer 20(9): 1520–1561, Sept 1967.*

Cancer of the large bowel is a common form of malignant disease in the United States. It accounted for more deaths than from any other type of cancer except pulmonary cancer.

This article has attempted to uncover factors that might affect the development of cancer of the large bowel. Since etiological factors may affect them differently, at least in degree, the data from the various parts of the large bowel are presented separately. Pertinent incidence data are presented first because they might offer important clues to the determination of etiological factors. Factors previously demonstrated or thought to affect the pathogenesis of this cancer are also reviewed.

### "IMMUNOLOGIC" ONCOGENESIS

*Philip J. Fialkow, MD, Blood 30 (3):388–394, Sept 1967.*

The observations that chromosomal changes occur in a variety of malignant tumors and that the known oncogenic agents—viruses, chemicals, and irradiation—produce chromosomal aberrations have stimulated interest in the role of these aberrations in the etiology of the tumors. In addition, it has been suggested that immunologic mechanisms may be related to the development of some neoplasms. In this article the possible relationship of this type of "immunologic" oncogenesis to that seen with viruses, chemicals, and irradiation are discussed in terms of chromosomal changes and cell death.

# DENTAL SECTION

### THE CROWDED ARCH

*T. K. Barber, J S Calif State Dental Assn 35(6): 232–240, June 1967.*

Orthodontic problems can be divided into two main categories: those cases having adequate space for the malaligned teeth, and those having inadequate space, the crowded arch. At least half of the crowded arch problems are due to hereditary, skeletal discrepancies. However, a great number are due to unfortunate environmental, or local occurrences during the developmental phases of the dentition change. The general practitioner possesses the ability to recognize and treat early the latter group, thus preventing the development of the crowded arch. Adequate arch length refers to sufficient space from the mesial of the first molar to the same surface of the first molar on the opposite side of the same arch to position properly all the appropriate permanent teeth. The principal environmental factors contributing to inadequate arch length are: (1) muscular forces, i.e., tongue, lips and cheek which mold the arch when the forces are properly balanced on the internal and external arch surfaces; (2) eruptive force exerts some degree of pressure on those teeth already in the arch; (3) occlusal forces, or those stresses placed on the teeth during mastication. Careful observation of these factors and maintenance of adequate records will make it possible for the general practitioner to prevent malocclusion resulting from this loss of arch length.

(Abstracted by: CAPT Nelson W. Rupp, DC USN.)

# PERSONNEL AND PROFESSIONAL NOTES

## DENTAL STANDARDS OF CANDIDATES FOR NAVAL ACADEMY AND NROTC PROGRAMS

"The needs of the service," policy changes, and progress have required a change in orthodontic dental standards for individuals applying for entrance to the Naval Academy and college NROTC programs. Revisions of dental standards and certain aspects of chapters 6–89 and 15–9 of the *Manual of the Medical Department* have been prepared and should be published in the near future.

Historically, it has been policy that individuals applying for entrance to the Naval Academy or college NROTC programs are disqualified if undergoing active orthodontic treatment with fixed orthodontic appliances. (Retainer appliances are permissible).

Justification for orthodontic dental standards has been predicated, in part, on the following factors:

a. That dental officers in the specialty of orthodontics were either nonexistent or not in sufficient numbers in the Navy to allow a complete orthodontic treatment program for individuals accepted in the Naval Academy.

b. That individuals who enter the Naval Academy or NROTC programs should not have serious dental defects which would significantly interfere with performance of duty or require extensive orthodontic treatment which would necessitate a frequent or prolonged absence from primary duty.

Accordingly, the following two recommendations have been approved by the Chief of Naval Personnel to change the orthodontic requirements for entrance into the Naval Academy and NROTC programs:

a. Candidates for the Naval Academy undergoing active orthodontic treatment will be temporarily disqualified. Each such applicant will be considered on an individual basis according to the professional judgment of the Permanent Board of Medical Examiners of the Naval Academy.

b. Candidates for entrance into the Naval Reserve Officer Training Corps, undergoing active orthodontic treatment, will be considered on the basis of a certifying statement concerning completion date of treatment by the individual's orthodontist.

### Training Applications Desired

Related to the above change in orthodontic standards and increasing pressures to provide limited orthodontic care for dependents at overseas bases, applications for training in long courses in orthodontics at civilian universities commencing during Fiscal Year 1969 may be considered. Applications should be submitted in accordance with ManMed 6–130 and forwarded so as to *be received in BUMED prior to 1 December 1967.* In conjunction with or immediately following submission of requests for assignment to the course of training, transcripts of academic records earned during predental and dental school training should be forwarded to BUMED Code 611. Any charges incurred in the procurement of the transcripts must be at the expense of the applicant. Additionally, a statement concerning the individual's motivation for requesting the training should be forwarded.

Applicants must be in the grade of lieutenant or lieutenant commander and have completed a tour of duty at sea or in areas considered foreign shore for rotational purposes. Each year of such training requires a service training agreement of three years after completion of the training.

## CIVILIAN EMPLOYEES

Although frequent references are made to the accomplishments of the Naval Dental Corps through the efforts of the dental officer/dental technician team, too often the support provided to the Dental Corps by many loyal civilian employees is not accorded its full share of recognition. The assistance and faithful service of the civilian employee is often remote to the dental team because of a lack of daily contact. This is inherent because of the nature of the tasks to which the civilian is usually assigned. With increased emphasis on the part of DOD to supplant military personnel with civilians, plus a changing philosophy of the dental profession to utilize auxiliary personnel to the greatest extent possible within existing standards of practice, the Naval Dental Corps now has more civilian employees directly associated with its efforts than at any time in its history. In this regard the following represents a partial list of categories of civilian employees reported on the Dental Service Report for the Quarter ending June 1967:

| | |
|---|---|
| Dental Hygienists | 67 |
| Dental Assistants | 110 |
| Laboratory Technicians | 19 |
| Clerical Personnel | 81 |
| Other | 44 |
| Total | 321 |

### NAVAL RESERVE OFFICERS

A Military Symposium for Naval Reserve Dental Officers will be held from 1630 to 1830 on 4 December 1967 in the Penn Top North, Statler Hilton Hotel, New York City, New York. The Symposium, which will be held in conjunction with the Greater New York Dental Meeting, is authorized one retirement point for accreditation to eligible Naval Reserve Dental Officers who attend.

# NURSE CORPS SECTION

### DIRECTOR, NAVY NURSE CORPS' CONFERENCE

The Chiefs of Nursing Service of all Naval Hospitals within the continental limits of the United States attended a 5 day Navy Nurse Corps Director's Conference from 18 to 22 September 1967 at the National Naval Medical Center, Bethesda, Maryland. CAPT Veronica M. Bulshefski, NC USN, convened the meeting for the Chiefs, Nursing Service. LCDR Katherine Wilson, NC USN, Chief, Nurse Corps Training Division, Naval Medical School, moderated the program.

RADM H. S. Etter, MC USN, Assistant Chief for Planning and Logistics, Bureau of Medicine and Surgery welcomed the chief nurses to the conference. CAPT John H. Stover, Jr., MC USN, Commanding Officer, Naval Medical School, addressed the group on new approaches to In-Service Education. RADM Frank T. Norris, MC USN, Assistant Chief for Personnel and Professional Operations, Bureau of Medicine and Surgery, discussed the professional medical staffing of a naval hospital.

CAPT Bulshefski spoke to the chief nurses throughout the program on topics relating to the theme of the meeting, "Nursing Administration in Action." She presented the status of the Nurse Corps, discussed personnel policies, education, and problem areas. She also informed the chief nurses of current projects and activities significant to nursing service in the Navy.

Other highlights of the meeting included various presentations by Nurse Corps officers who are assigned to nursing service administration, education, and research in naval medical activities, the Nursing Division, Bureau of Medicine and Surgery and the Bureau of Naval Personnel. Among the topics included were: Nursing Service Administration, Personnel Planning and Accounting, Nursing on the USS REPOSE, Nurse Corps Officer Procurement, Counseling and Developing the Young Nurse Corps Officer, Educational Opportunities for the Navy Nurse Corps, Personnel Actions for the Nurse Corps Officers and Programmed Instruction.

Distinguished civilian guest speakers and the titles of their presentations were: Miss Mary E. Brackett, Associate Director of Nurses, Hartford Hospital, Hartford, Connecticut, "Current Trends and Staffing Patterns;" Mary Liston, Doctor of Education, Dean, School of Nursing, Catholic University of America, Washington, D.C., "Newer Concepts of Basic Nursing Education;" Mr. Robert S. Connors, Representative, Government Educational Medical Region, IBM Corporation, "Automation in Patient Care;" Miss Julia Thompson, Director, Washington Office Government Relations American Nurses' Association, "Nursing Legislation;" Dagmar Brodt, Ph.D., Clinical Investigation Department, Naval Medical Research Institute, "Ward Manager Research Project," and Helen Creighton, R.N., J.D. Associate Professor of Nursing, University of Southwestern Louisiana, "Legal Aspects of Nursing."

Some of the papers presented at the conference will be published in forthcoming issues of the *U.S. Navy Medical News Letter.*

### DIRECTOR, NAVY NURSE CORPS, PARTICIPATES IN EDUCATIONAL TV PROGRAM FOR SCHOOLS OF NURSING

CAPT Veronica M. Bulshefski recently represented the Directors of the Army, Navy, and Air Force Nurse Corps in the production of a closed circuit TV class at Chicago Video Nursing, Chicago, Illinois. The film taped will be part of a course in nursing trends for student nurses in schools of nursing throughout the Illinois area. Other classes for this course will be taped by outstanding nursing leaders from all over the country. CAPT Bulshefski discussed the contributions and history of all military Nurse Corps.

LT Mary E. Sulkowski, NC USN, representing the three military Nurse Corps, discussed the nursing care of military casualties in Vietnam. Film from the USS REPOSE; Naval Hospital, Philadelphia; and Air Force Evacuation Activities was used. The TV production is supported by a United States Public Health Service grant.

# PREVENTIVE MEDICINE SECTION

## COMBATTING CHRONIC ACTIVE HEPATITIS — A LETHAL DISEASE OF YOUNG PEOPLE

*E. A. Gelzayd, Consultant 7(7):32–35, July–August 1967.*

In treating young adults, especially young women, one may someday encounter a bizarre form of liver disease that defies diagnosis—a form of hepatitis that progresses within 10 years to cirrhosis, liver failure, and, nearly always, death. This lethal variant, which is called chronic active hepatitis in young people, is usually called lupoid hepatitis, but you may have heard it called juvenile cirrhosis or, simply, chronic liver disease. Despite the uncertainity of its name, the disease does show a reliably distinct clinical pattern that should be recognized and treated vigorously.

*What Is It?* Chronic active hepatitis is a progressive form of parenchymal liver disease accompanied by symptoms suggesting a connective tissue disease and by L.E. cells in the peripheral blood. In many patients, it responds temporarily to treatment with corticosteroids. This disease was first separated in 1950–1951 from posthepatitis cirrhosis (or Laennec's cirrhosis) and the following changes were described in afflicted young women with chronic liver disease: postnecrotic cirrhosis with hepatic cell necrosis and destruction of the parenchymal plate ("patchy" necrosis); and pronounced infiltration of mononuclear cells into the portal tracts and necrotic areas.

Besides these liver changes were the following abnormalities: recurrent fever, malaise, jaundice, amenorrhea, arthralgia, arthritis, pericarditis, hepatosplenomegaly, elevated levels of serum gamma globulins, occasionally positive L.E. cell tests, and certain various serum and tissue antibodies—quite a few things that suggest autoimmune phenomena and a connective-tissue disease similar to lupus erythematosus.

Chronic active hepatitis is apt to occur during or after other diseases such as ulcerative colitis, autoimmune hemolytic anemia, rheumatic diseases, and Sjogren's disease. Like these diseases, chronic active hepatitis usually responds well (though temporarily) to treatment with adrenal corticosteroids.

*What Causes It?* No one knows for certain. Few victims have obvious causes of liver diseases such as alcoholism and nutritional abnormalities. Few have the usual signs of viral hepatitis such as acute onset of illness. The following have been suspected but never proven guilty: infection, hepatotoxins, hepatic circulatory disturbances, endocrine disturbances (particularly in women with striae, amenorrhea, acne, and hirsutism), heredity, and immune mechanisms.

This last possibility—immune mechanism—has inspired the interesting, though still unproven, theory that chronic active hepatitis results from a "smoldering virus infection." Since it does occasionally begin with an acute illness like that of viral hepatitis, it might begin when the hepatitis virus kills some liver cells. The body then might react to these killed cells by producing antibodies to them (presumably formed by the lymphocytic and plasma-cell infiltrates) which then kill more liver cells, and set in motion a circular pattern of continuing liver damage.

The presence of L.E. cells, which are a response to circulating antinuclear antibodies, supports the immunologic-reaction theory. So do other immunologic abnormalities that have been reported in patients with chronic active hepatitis: strongly positive autoimmune complement fixation tests (in 44% of one group of patients), hypergammaglobulinemia, circulating autoantibodies (including L.E. factors), lymphoid infiltration of the liver, and therapeutic response to cortisone. Thus, the immunologic reaction theory is a plausible one but must be considered strictly speculative. Many authors dispute this theory and suggest instead that chronic active hepatitis may be a form of viral hepatitis; that terminal viral hepatitis may occur coincidentally in a patient with lupus erythematosus; that it may be a liver-related variant of lupus erythematosus; or finally, that it may be a previously unrecognized primary disease of the liver.

*How Can You Identify It?* First, suspect it in every young adult with fever, malaise, persistent jaundice, moderate hepatosplenomegaly, or other signs of chronic liver disease. Then, look for the following disturbances that characteristically accompany it: mild arthritis, arthralgia, pleurisy, pericarditis, myocarditis, thrombocytopenic purpura, amenorrhea, gynecomastia, and ulcerative colitis (which occasionally precedes the liver disease). Skin rashes are common and may form the "butterfly rash" that is classic in lupus erythematosus.

TABLE I. *What Lab Findings Point to Chronic Active Hepatitis?*

| | Normal Liver Function Test Results | Chronic Active Hepatitis | Acute Hepatitis |
|---|---|---|---|
| BSP retention | 5% at 45 min. | Moderately high | Extremely high |
| Serum levels: | | | |
| Alkaline phosphatase | 0.5 to 4.0 B.u. (3 to 13 K.A.u.) | Mild to moderately high | High |
| Albumin | 4–5.5 Gm./100 ml. | Normal or low | Normal or low |
| Gamma globulin | 0.5–1.5 Gm./100 ml. | VERY HIGH | Slightly elevated |
| Glutamic oxalacetic transaminase | 4–40 u. | Moderately high | Extremely high |
| Glutamic pyruvic transaminase | 1–45 u. | Moderately high | Extremely high |
| Bilirubin | 1.0 mg./100 ml. | Slightly elevated | High |
| Cholesterol | 140–270 mg./100 ml. | Normal or low | Normal or low |
| Cholesterol esters | 75–210 mg./100 ml. | Normal or low | Very low |

Confirm the suspicion with liver-function tests. (See Table I.) In chronic active hepatitis, bromsulphalein retention rises to 20% or more. The serum bilirubin rises to 5 mg per 100 ml or more, especially during active phases of the disease. The serum alkaline phosphatase and serum glutamic pyruvic transaminase (SGPT) also rise to mildly or moderately elevated levels; severely elevated SGPT levels suggest acute liver injury. The serum cholesterol and cholesterol esters remain normal or slightly low.

Serum protein levels go strikingly high and constitute the most diagnostic biochemical change. The serum globulins rise to an average of 3 to 5 grams per 100 ml (almost twice normal) and have been reported as high as 11 grams per 100 ml. Elevated levels persist even after treatment and liver function tests return to near normal. This stepped-up production of gamma globulin accounts for other possible abnormalities: occasionally positive cephalin flocculation and thymol turbidity tests; positive L.E. cell tests (in ⅓ of patients with chronic active hepatitis); positive rheumatoid factors; false positive reactions for syphilis; and antibodies against liver, thyroid, smooth muscle, and red blood cells.

*Can You Cure It?* Unfortunately, no. Chronic active hepatitis usually progresses to irreversible liver failure in 5 to 10 years after diagnosis. Only a few patients survive longer than 10 years. At best, the prominent biochemical and clinical abnormalities can be only temporarily corrected by prescribing:

High-protein and high-carbohydrate diets to ensure adequate nutrition (if hepatic failure occurs, switch to low-protein and high-carbohydrate).

Adequate rest, particularly when the disease is "active" (when SGPT and bilirubin are high). Absolute bed rest is probably not necessary; eight hours of sleep plus an afternoon nap are usually enough.

Treatment for associated diseases: for diabetes, ulcerative colitis, thyroid disorders, and so forth.

Treatment of underlying anemia or vitamin deficiency (most likely deficiencies are of folic acid and $B_{12}$).

Do not prescribe any potentially hepatotoxic agents such as estrogens, oral contraceptives, testosterone, or the C17-alkyl steroids. Do not prescribe antibiotics except for specific infections (for example, of the urinary tract). Patients with chronic active hepatitis do not tend to develop any intrahepatic, portal or systemic bacterial infection and do not seem to be helped by antibiotics in any way.

*Do Corticosteroids Help?* Adrenal corticosteroids do help control both hepatic inflammation and immunologic disturbances but their long-range effect is unpredictable. Most patients promptly respond with renewed well-being, restored strength and appetite, and return of menstruation. Within 2 weeks, jaundice usually subsides and serum enzyme levels fall, in some cases to normal levels. Serum globulins fall more gradually, indicating suppression of hepatic inflammation.

Unfortunately, these apparently favorable responses may be misleading: the underlying liver disease may continue and progress to cirrhosis despite the near-normal test results; and, in some patients, hepatic fibrosis may even accelerate. The patient may also develop side effects: infection, diabetes, peptic ulcer, and osteoporosis. This strange unpredictability makes the decision to use steroids a thorny one indeed. However, using the following guidelines, steroids are prescribed when:

There are no specific contraindications to them.

The disease remains active (high bilirubin and SGPT) despite therapy, rest, and avoidance of hepatotoxins.

Serum gamma globulin levels are high, with a positive L.E. cell test.

Usually, 30 to 40 mg are given daily, gradually decreasing the dose to maintenance levels (10 to 15 mg) after the serum bilirubin and SGPT levels fall to normal. If the patient grows worse or develops another illness, the dosage is increased to as high as 30 mg daily, if not contraindicated.

Steroids should be continued indefinitely, depending on the results of repeated SGPT-bilirubin measurements and occasional liver biopsies. When biopsy shows restoration of the hepatic architecture, they can be discontinued. Recently, long-term usage of small doses of azathioprine (6–MP) has been effective. We can only hope that the future will bring other immunosuppressive agents that may truly cure or control chronic active hepatitis.

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

## MANTOUX TUBERCULIN TESTING

Following the widespread use of jet-injector guns for subcutaneous inoculations, which has proved to be a highly efficient means of administering immunizations on a large scale, increasing interest has been shown in the use of such devices for mass tuberculin testing. With the development of the intradermal head for jet-injector guns, some activities have begun to substitute this method for the Mantoux method prescribed for the Navy tuberculosis control program. The following is presented as clarification.

Although the advantages of the intradermal jet-injector techniques for tuberculosis testing are obvious, these advantages are of no consequence unless the technique can be shown to be accurate enough for the purposes of the Navy tuberculosis control program. Consequently, studies comparing this technique with the standard Mantoux test have been conducted at Preventive Medicine Unit No. 2, Norfolk, Virginia, and results of studies conducted elsewhere have been evaluated.

One such study was conducted at the Lincoln State School, Lincoln, Illinois, by Morse, et al. Using 5 mm or more of induration as a positive reaction, the agreement between the two methods was 97.45 percent in 1,254 patients. However, if a 10 mm reaction had been used as a lower level of positivity, seven gun positives would have been missed by the needle, and 140 needle positives would have been missed by the gun.

Bettag, et al, dual-tested 5,911 high school students in DuPage County, Illinois, and found 128 positive reactions. There were 2 false negatives and 6 false positives by the standard method, and 21 false negatives and no false positives by the jet-injector method. The latter was in contrast to previous testing results, when their had been a preponderance of false positives to the jet-injector gun.

Feigenbaum, et al, simultaneously tested 1,478 Vietnamese mental patients with jet-injector and Mantoux methods. Using an index of 10 mm as the dividing line between positive and negative reactors, 180 patients were tuberculin negative in the standard Mantoux group, and 771 were negative in the jet-injector group; a discrepancy of 591 cases or 47% of the 1,298 patients with 10 mm or greater induration using standard Mantoux techniques.

Preventive Medicine Unit No. 2, Norfolk, has reported a number of disadvantages to the jet-injector techniques, including: (1) the exact amount of PPD that actually was injected intradermally could not be determined accurately; (2) some persons were very difficult to inject intradermally, and (3) PPD was wasted in multiple attempts to raise wheals of adequate size. In this study, 21% of the 28 positive reactors (or approximately 2% of the population tested) would have been missed if the jet-injector gun alone had been used. This could be a potentially serious deficiency, if an active case of tuberculosis or a positive tuberculin skin test converter was missed because of a false negative reaction.

The Navy tuberculosis control program as described in BUMEDINST 6224.1C is based on serial tuberculin skin testing and is dependent upon a high degree of sensitivity, specificity, and *quantative comparability* attainable with the standard Mantoux test. Although the jet-injector technique may be satisfactory for and adaptable to large-scale screening, it cannot be considered to be adequate for the Navy control program at the present time. Activities, therefore, are advised that Mantoux testing as required by BUMEDINST 6224.1C is defined as administration of appropriate doses of PPD skin test material intradermally by needle exclusively.—Tuberculosis Control Section, BuMed.

## NOSOCOMIAL STREPTOCOCCAL INFECTIONS

*USDHEW PHS NCDC Morb & Mort Wkly Rpt 16(27):221–222, July 8, 1967.*

Between 22 April and 8 May 1967, five cases of nosocomial beta-hemolytic streptococcal infections occurred on the obstetrical-gynecological services of a moderate-sized hospital in New Jersey. Three cases of endometritis occurred in women who had delivered 36 to 60 hours previously. The 4th case was in a patient who developed peritonitis and septicemia 36 hours after a subtotal abdominal hysterectomy. A postoperative wound infection developed in a 5th woman 2 days after tubal ligation. The 4th patient died with overwhelming sepsis; the other recovered following antibiotic therapy.

An investigation was initiated after the first 4 infections. Throat cultures were taken from all personnel who had worked on the obstetrical or surgical services during the time these patients were hospitalized. No Group A beta-hemolytic streptococci were found. When the 5th case became apparent on 8 May, further studies were undertaken. The patient, operating room, and nursing schedule records were examined. Fifteen individuals who had had common contact with more than 1 of the infected patients were questioned about recent infections, had blood drawn for ASO titers, and had cultures taken of their nose, throat, and rectum. Vaginal cultures were also obtained from the nursing staff.

All physicians and nurses denied symptoms of recent infection, and none had been in contact with all of the cases. However, 1 physician had delivered the first case and was present at the operations on the 4th and 5th cases. The second culture survey demonstrated that this physician was carrying a beta-hemolytic Group A streptococcus in his nose, but not in his throat. All cultures obtained from other personnel were negative for Group A streptococci. A streptococcal organism of the same type (M: nontypable, T:28) was isolated from both the index case and the fatal case. The streptococcus isolated from the 5th case was a different type (M:12, T:12). Isolates from the remaining 2 cases were not saved and could not be typed.

One nurse had performed the perineal preparations of the first 3 cases. She had no contact with the other cases. Serologic studies revealed that she had an elevated ASO titer.

The exact means by which all cases acquired infection could not be established. It was hypothesized that the physician, a nasal carrier of beta-hemolytic Group A streptococci, transmitted this pathogen to the index and 4th cases. The nurse may have acquired the organism by contact with the physician, become asymptomatically infected, and transmitted streptococci to the second and third cases at the time of perineal preparations. The origin of the organism responsible for the 5th case, which was unrelated to the other cases on the basis of typing, could not be established.

The physician was treated with oxacillin and his nasal cultures became free of streptococcus. No further cases have occurred since May 8.

## STROKE IN YOUNG ADULTS

*S. Louis, et al, Ann Intern Med 66:932–938, May 1967.*

The characteristics of the "stroke-prone" individual were studied in a group of young adults who had suffered a nonembolic cerebral infarction. The assumption made was that the causatively contributory factors and associated diseases would be more easily apparent in patients developing stroke at unusually young age. Fifty-six patients were reviewed who developed stroke at 50 years of age or less and who were followed for an average of 27 months. The incidence in males and females was the same up to 45 years of age, following which males predominated. Hypotension at the time of stroke occurred in only 4 patients. Hypertension was present in more than half. Diabetes was present in 22% of patients; a further 20% became diabetic within 3 years of the stroke. Abnormalities of serum lipids were found in half of those tested. In 4 patients specific arterial lesions such as systemic lupus erythematosus or syphilis were found. In only 4 patients of the 56, none of the above associated disorders was detected. Thus, diabetes, prediabetes, hypertension, or an abnormal blood lipid pattern were found in 86% of the patients studied and constitute some of the features predisposing to cerebral infarction.

## MULTIPLE SCLEROSIS IN THE U.S.

*Statist Bull Metrop Life Insur Co 48: 6–8, June 1967.*

Multiple Sclerosis is a chronic neurological disorder which attacks and often cripples young adults. Its cause is unknown and its recognition is

elusive, especially in its early stages. As yet no prevention, cure, or specific treatment has been found.

The precise number of persons in the United States currently afflicted with multiple sclerosis is not known. Estimates, based on morbidity surveys conducted within the past 20 years, range widely from about 70,000 to ¼ of a million persons. The latter figure probably includes many cases, especially in the early stages, which may not have been diagnosed or reported because of the remittent nature of the disease. Since the surveys were conducted by different investigators in widely separated communities, they may not be representative of the prevalence of the disease nationally. The prevalence in the cities of the North was found to be substantially higher than in those of the South, ranging from 64 per 100,000 white persons in Rochester, Minnesota to 13 in New Orleans, Louisiana.

Mortality from the disease is higher in the colder regions of the country. All the northern states had death rates above the national average of 0.8 per 100,000 population whereas those in the southern latitudes had rates below the rate of 1.4 in Montana, in North Dakota, and in Nebraska was almost 5 times greater than in Georgia and Louisiana.

Mortality from multiple sclerosis is largely concentrated in midlife. More than half of all the deaths during 1960–64 occurred between the ages of 45 and 65. The mortality rates rose steadily with advancing age, from 0.4 per 100,000 population at ages 25–34 to a maximum of 2.1 at ages 55–64, then declined. Among females, who generally have an earlier age at onset of this disease than males, peak mortality rates began about 10 years earlier. Females registered higher rates than males at all ages under 65, with the greatest disparity before midlife. Considered by color, the rates for whites were almost twice those for nonwhites, the disparity increasing with age. A possible explanation for this difference is the fact that a far greater proportion of nonwhites than whites live in or have recently migrated from the South, where multiple sclerosis is reported lower in mortality and believed to be lower in prevalence than in the North. The tendency of females and the white population to seek medical care more promptly permits early and continued observation, so important in diagnosing this disease, and this may be a significant factor in the wide differences in mortality from this disease reported by sex and color.

Mortality statistics are believed to understate the number of persons dying from multiple sclerosis. In addition to the mortality ascribed directly to the disease, a considerable number of deaths are reported as due to other causes but with multiple sclerosis mentioned as a contributory or complicating factor. This is shown in a 1955 study of multiple causes of death. Almost 30% of the nearly 2,000 death certificates mentioning multiple sclerosis were ascribed to other causes. Some further understatement may result when the disease does not appear on the death certificate either because it is in a period of remission or because it has been misdiagnosed.

The course of multiple sclerosis is often characterized by relapses of increasing severity and duration, with about 50% of those affected becoming incapacitated within 10 years after onset. It is an important cause of disability among young adults: 71% of the 2,000 workers who were granted disability benefits by the Social Security Administration on account of this impairment during 1964 were under age 50. In contrast, only 14% of the 11,368 workers disabled by strokes, the leading crippler of the central nervous system, were under age 50.

The increasing disability and premature death which have long been regarded as characteristic of the course of multiple sclerosis are not necessarily inevitable. There are cases in which relapses are infrequent and mild, allowing full enjoyment of active life for many years. In a follow-up of 241 patients first seen within 3 years of onset, 32% were not disabled in the tenth or later years of the disease. Included in this series were a small number of patients who had not suffered a relapse after the first year. Other studies have also pointed to the advisability of a more positive attitude in the prognosis for this disease. Better care of multiple sclerosis victims and the application of chemotherapeutic and antibiotic agent to the treatment of complications are believed to have prolonged the life of many. Authorities now think that the average duration of life from onset of this disease may be about 27 years, whereas a quarter of a century ago it was thought to be from 5 to 10 years.

With the recognition that no specific cure is yet available for multiple sclerosis, stress is being placed on research. The National Institute of Neurological Diseases and Blindness, U.S. Public Health Service, and the National Multiple Scle-

rosis Society provide financial grants in support of research relating to the cause, prevention, diagnosis and treatment of multiple sclerosis. New approaches are being sought and new groups of neurological research scientists are being recruited. Until there is a breakthrough in our knowledge, reliance will have to be placed on medical management directed toward protection from serious complications, amelioration of symptoms, and rehabilitation vigorously pursued through the development and expansion of community and patient service programs.

## KNOW YOUR WORLD

Did You Know?

That *Trichinae* were found in 140 (4.5%) of tissues examined from 3,095 human autopsies, done in 23 states?

The Veterinary Medical Research Institute, Iowa State University is participating in the National Trichinosis Study.[1]

---

That 50 human cases of psittacosis were reported from 19 states in 1966, 11 cases less than 1965?

This is the lowest total since 1951 when 25 cases were recorded. Parakeets and pigeons, the 2 most common reservoir hosts since 1962, accounted for 73% of the 45 cases; parakeets were the most probable source of infection for 24 cases—53%; pigeons 9—20%. Pet-bird owners, with 21 of 45 cases (47%) comprised the largest exposure category. Only 1 case reported in a poultry processor was in a 9-year-old child who helped his mother gather chicken eggs for subsequent sale. Two common-source outbreaks, implicating pet parakeets, involved a man and his wife, and the other, a mother and 3 children. Of the 45 human cases studied, 29 (64%) occurred in males, ages 3–82 years, with a mean of 43 and a median of 46 years. No turkey, chicken, or other avian ornithosis outbreaks were reported in 1966.[2]

---

That $20,720 was awarded to the Veterinary Department of Ohio Department of Health, Columbus, to trace how eggs and egg products contribute to Salmonella disease in humans?

This study includes identification and tracing of salmonella bacteria found in both raw and finished egg products; determine frequency of and extent of contamination; precise types of bacteria on or in eggs entering processing plants; flocks from which eggs originated and how the flocks became infected, to sample eggs before and after the various processing steps to identify at which step contamination occurred, and environmental factors involved on the farm and in plants. This project has been recommended by the National Advisory Council on Environmental Health.[3]

---

That 167 cases of yellow fever were diagnosed as clinically suspected with 63 deaths during the 1966 outbreak in Brazil?

Twenty-two of the fatal cases were confirmed by histopathological examination of liver specimens. Clinical cases occurred in 3 States: Parana—100 cases with 32 deaths; Rio Grande do Sul—22 cases with 13 deaths; and Santa Catarina—45 cases with 18 deaths.[4]

---

That the 2-cigarette pack-a-day man at age 25 has an 8-year shorter life expectancy, if he continues the habit, than a nonsmoker? [5]

---

That of the more than 2,000,000 diabetics in the United States in 1964 and 1965 had family histories of diabetes?

About 17% of diabetics had diabetic mothers; 8% had diabetic fathers. About 1/4 interviewed reported diabetic brothers or sisters, and 7% of the diabetic parents had diabetic children.[6]

---

That at present there are between 15 and 20 million infectious cases of tuberculosis throughout the world and that the disease causes from 2 to 3 million deaths per year?

More than 80% of the burden of disease falls on the developing countries.[7]

---

That a trial of oral amantadine prophylaxis in naturally occurring acute respiratory illness was conducted among mentally retarded but educable children during the winter and spring of 1965?

For the 4 months of the trial, amantadine was well tolerated at a dose of 1.0–2.5 mg per Kg/day, and over 97% of the prescribed doses were ingested. Amantadine and placebo groups did not substantially differ either in streptococcal illness during the trial or in any illness category during a 3-week pretrial observation. However, the amantadine group experienced significantly less respiratory illness during the trial than did the placebo group. Part of this difference is attributed to the successful chemoprophylaxis of influenza $A_2$. The remainder

was accounted for by the decrease in upper respiratory illnesses that have not yet been etiologically identified. These upper respiratory illnesses occurred in a single wave involving 5 of the 16 housing units.[8]


References

1. USDHEW PHS BuPrevDis&EnvContr CDC Vet PH Notes, p 5, Aug 1967.
2. USDHEW PHS NCDC Morb & Mort Wkly Rpt 16(24): 287, Aug 26, 1967.
3. Indiana State Bd of Hlth "Bulletin" 69(4): 2, Apr 1967.
4. PASBU WHO Wkly Epid Rpt XXXIX (33): 182, Aug 16, 1967.
5. "Hospital Tribune," Med News, Inc 1(34): 1, Sept 25, 1967.
6. "Hospital Tribune," Med News, Inc 1(34): 4, Sept 25, 1967.
7. WHO Chronicle 21(8): 353, Aug 1967.
8. Finkles, J. F., et al, Amer J Epid 85(3): 403–411, May 1967.


# EDITOR'S SECTION

## GARY P. WRATTEN SURGICAL SYMPOSIUM

A symposium on current surgical practices will be held at Walter Reed Army Medical Center, on Monday, Tuesday and Wednesday, 1, 2 and 3 April 1968. The Surgeon General of the Army has given his strong support to this seminar. An outstanding program has been arranged which will include recent advances in the fields of general surgery and the surgical specialties, new advances in clinical research and new procedures and techniques. Civilian surgeons of national prominence are included on the program.

Medical officers are urged to make application for presentation of papers, to be limited to 15 minutes with few exceptions. Case reports will also be accepted, limited to 5 minutes. Submit the title of your paper together with an abstract of not more than 50 words and time desired for presentation, to LCOL Joseph H. Baugh, MC USA, Chief, General Surgery Service, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D.C. 20012, (with a copy to BUMED) not later than 1 January 1968.

The symposium is open to surgeons of the Army, Air Force, Navy, Veterans Administration, Public Health Service, and also civilians, particularly from Reserve Corps and National Guard. All are invited and encouraged to attend. Social events will include the wives. There will be a "get acquainted" cocktail-buffet on Sunday evening, 31 March, and a cocktail-banquet on Tuesday evening, 2 April, at the Walter Reed Army Medical Center Officers' Club.—Training Branch, BuMed.

## NEW EXPERIMENTAL HEART VALVE REPLACEMENT

Surgeons of the Public Health Service's National Heart Institute have devised a heart valve replacement which combines the post-operative advantages of valve transplants (homografts) with the ease of insertion of artificial heart valves.

The new experimental device, designed by Drs. Nina S. Braunwald, James C. A. Fuchs, and Lawrence I. Bonchek of the NHI Surgery Branch, has proven highly successful in calves and promises to avoid the clotting problems occasionally encountered with artificial valve replacements.

Heart valves severely damaged by rheumatic fever are usually replaced by artificial valves, most commonly ball valves and caged disc (lens) valves constructed from plastic or metal. However, a common problem following the insertion of artificial valves has been clotting complications, which occur in 10–40% of patients who survive cardiac valve replacement with such prostheses.

Artificial aortic valve replacements pose relatively few clotting complications because they are continually swept clean by strong surges of blood from the contracting left ventricle. However, replacements for the mitral valve (between the left atrium and left ventricle) and the tricuspid valve (between the right atrium and right ventricle) must be installed at sites where lower blood pressures and flow velocities prevail. Under these conditions, clots can form more readily on valve surfaces. Improved valve designs and materials have helped to reduce this problem; but, even so, patients fitted with artificial mitral or tricuspid valves must usually remain on continuous anticoagulant therapy.

Clinical experience of the last four years indicates that homograft valves—specifically healthy aortic valves taken from donors dying of other causes—are almost entirely devoid of clotting complications.

However, each homograft valve must be trimmed and fitted at the time of operation and its installation is technically more difficult than is the insertion of an artificial valve. Both factors substantially increase the time required for valve installation,

and hence the critical period during which the patient's heart must be stopped and his circulation maintained by the heart-lung machine.

The NHI surgeons attacked all of these problems in the design of their new prosthesis. It consists of a homograft valve—in this case, the aortic valve of a calf—supported by a specially designed thrombus-resistant prosthetic frame. The frame is composed of a base ring and three vertical struts.

Aortic homograft valves were obtained from freshly slaughtered calves, trimmed, and measured with a dilator. Prior to attaching the homograft to the frame, the investigators covered the frame and sewing ring with a highly porous fabric (60 denier polypropylene). Their earlier studies had shown that artificial valves could be made highly resistant to clot formation by covering all fixed parts with a porous fabric to promote tissue ingrowth. The valves are then attached to the metal frames and cloth-covered sewing ring is attached to the outer surface of the base ring.

Then, each mounted valve was subjected to performance tests in the transparent chamber of a pulse duplicating machine. Those valves which demonstrated satisfactory function (documented by still photographs, motion pictures, and by dye injections above the valves) were chemically sterilized and stored in a cold antibiotic solution before implantation in calves.

The availability of completely fabricated valves of fixed diameter for immediate use eliminated the need to trim and fit a valve during surgery. Furthermore, the valves were simpler to install than nonfabricated homografts and could be placed at any valve site within the heart.

Following the fabrication and sterilization process, the mounted homografts were implanted in 23 calves: the tricuspid valve was replaced in 14 animals and the mitral valve in nine others. This constituted a stern test of the clot-resisting properties of the new prosthesis while also demonstrating that mounted aortic homografts could be used to replace diseased valves elsewhere in the heart.

The fabric sewing ring of the mounted homograft permitted the use of conventional interrupted suture techniques and shortened the time required for installation to roughly the same as that required for insertion of a rigid caged-ball prostheses. Each calf received penicillin and streptomycin for five days postoperatively, but no anticoagulants were employed.

Catheterization studies one to three months following operation in 12 calves indicated complete effectiveness of the valves in 11. After the animals were killed, the autopsy examinations showed that 18 of the 21 calves had efficient valves with clean, well-supported leaflets. Failures in three were attributed to improper placement of the prosthetic struts in the earliest valves. This was later corrected and slight adjustments were also made in placement and suturing of the frame.

These experiments have shown the value of inserting aortic homograft valves supported on nonthrombogenic prosthetic frames. The design combines the clot resistance of homograft valves with the ease of insertion of caged-ball valves. It also permits the use of an aortic valve homograft in the replacement of mitral or tricuspid valves.—National Heart Institute, Bethesda, Md.

## MARIJUANA

*The Medical Letter 9(19), Issue 227, Sept 22, 1967.*

In the United States, marijuana is the term for any preparation or extract of the Cannabis plant or hemp. Most of the active ingredients are in the resinous exudate of the unfertilized flowering tops of the plant, which can grow wild in any temperate climate. The drug is not available legally in the United States except by a special license from the Internal Revenue Service for research purposes. It has no recognized medical uses.

Marijuana is generally smoked as a cigarette but the resin is sometimes ingested in candy form, a practice most common in eastern countries. The chief active ingredient is believed to be tetrahydrocannabinol; different preparations contain varying combinations of isomers of tetrahydrocannabinol, their strength depending on where the plant was grown, the method of cultivation, how long it was stored, and the methods used in extracting the resin. At present the potency of marijuana cannot be determined by chemical analysis.

*Subjective Effects*—When marijuana is smoked, the subjective effects usually start promptly and last for about four hours. A dreamy state in which ideas and images flow freely and pleasantly is common. Time sense, spatial relationships and body image can be markedly distorted. There is an intensified sensory perception, especially of auditory and visual stimuli. The usual mood is one of euphoria or exaltation but there may be mood swings or even a predominant depression. Uncontrollable laughter and hilarity are common, especially when marijuana smoking is a group affair, as it usually is.

*Serious Reactions*—Advocates of lifting the legal restrictions on the use of marijuana state that it is no more harmful than alcohol or cigarettes. However, reports of serious adverse emotional reactions and personality changes are increasing. Panic, gross confusion, impulsive and aggressive behavior, depersonalization, depression, and paranoid behavior have been reported, especially when marijuana is combined with other drugs, such as alcohol and amphetamines. With large doses (or in susceptible persons) delusions or hallucinations can occur (P. Dally, Brit. Med. J., 3:367, Aug. 5, 1967; E. Tylden, Brit. Med. J., 3:556, Aug. 26, 1967). Indolence and neglect of personal hygiene may follow prolonged heavy use, and intellectual functioning and memory may be impaired. Descriptions of the behavioral and subjective effects of large doses of marijuana and of LSD (Medical Letter, Vol. 9, p. 1, 1967), mescaline and psilocybin are remarkably similar.

*Physical Effects*—The drug sometimes causes postural hypotension. Mydriasis, conjunctival congestion and photophobia are frequent. Muscular incoordination, spasms, urinary frequency, dryness of the mouth, nausea, vomiting, and diarrhea sometimes occur. Increase in appetite is common, and has been attributed to hypoglycemia. Hypothermia has been observed and some workers have noted a Raynaud-like syndrome (cold extremities and white finger tips). Large amounts of marijuana depress respiration, which is the first sign of impending death in animals given lethal doses.

In present patterns of drug abuse, marijuana is often the first drug to be used. There is no evidence that it has any physiological or emotional effects which directly lead to more serious drug abuse, but its use may facilitate contact with persons or groups using more dangerous drugs. There has been no evidence of physical dependence on commonly available preparations of marijuana; withdrawal symptoms have been reported, however, following the use of the more potent material available in North Africa and now coming into the United States.

The hazards of occasional marijuana smoking should not be exaggerated, but attempts to limit the use of the drug remain appropriate, especially since relaxation of controls would probably lead to greater importation and use of more potent preparations with a resultant increase in serious side effects.

DEPARTMENT OF THE NAVY

BUREAU OF MEDICINE AND SURGERY
WASHINGTON, D.C. 20390

OFFICIAL BUSINESS

PERMIT NO. 1048

POSTAGE AND FEES PAID
DEPARTMENT OF THE NAVY