What's New in Communicable Diseases

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SLUDGED BLOOD—After 16 years of study, Knisely and his associates [17] report that in the experimental animal as well as in the human subject, the following changes are absent in health but present in disease, either induced or naturally occurring: masses of agglutinated red cells (not rouleaux) of different basic sizes and degrees of agglutination appear in the blood vessels, in one or more of the following phenomena: reduction of the rate of blood flow in all open vessels; ingestion of the agglutinated sludge by the fixed tissue, endothelial, physiological phagocytes of the spleen and liver (hemophage cells); the sedimentation and settling out of the agglutinated sludge from the circulating plasma; either temporary or permanent plugging to a varying degree of arterioles and other vessels.

The effects produced in the blood by many different systemtic diseases of different origin, and by severe injuries, were studied in nearly 600 humans. The various lenses of a binocular dissecting microscope were focused on the capiliaries of the eyelid. Every severely sick person showed visible evidence of intravascular sludged blood and abnormal changes in the vessel wall.

This poses the following questions: In the presence of severe infections, in addition to attempting to free the body from invading organisms, must we attempt to prevent blood from sludging, entirely or in part, and if so, what is the best procedure? In addition, what steps may be taken to prevent destruction of normal blood cells, preserve adequate blood volume, prevent thrombi and emboli, and keep vessel walls intact?

Brucellosis.—It has long been known that controlled high blood levels of various members of the sulfonamide drugs will cure acute brucellosis in many cases. However, most patients are treated with inadequate levels without effecting a cure. In such cases, later treatment with sulfonamides inevitably results in failure, the surviving organisms having become entirely resistant.

While streptomycin prevents the growth of the bovine, porcine, and caprine strains of Brucella [14] in vitro, and at a concentration readily achievable in human blood, it has been disappointing in the prevention or treatment of the disease in the experimental animal and in treatment in the human. The combination of sulfonamides with streptomycin in treatment appears to be successful.[10,21] From present data it appears that streptomycin should be given in 0.5 gm. doses every six hours for seven days only. Concurrently, sulfadiazine is given in an initial dose of 4 gm., to be followed by 2 gm. every four hours thereafter for at least two weeks and preferably three. Our comment is that we prefer equal parts of sulfadiazine and sulfamerazine to either drug alone, and we believe that the blood level should be maintained at 15 mg. per cent or higher and the urinary pH kept at 7.4 or higher.

Vaccination against cholera produces antibodies that give a positive agglutination against Brucella organisms.[11] This should be taken into account in doing agglutination tests.

The inability to secure positive agglutination tests in many bacteriologically proven cases of brucellosis is well known. Absent or low agglutination may be due to the presence of blocking antibodies in the patient's blood, and upon their removal either the test becomes positive or the positive titer rises. Using rabbit or bovine sera entirely to replace all saline solution as a diluent for titrations, or as a suspending medium for the bacilli, causes the disappearance of these blocking antibodies.

Spencer's Disease.—This disease is variously known as intestinal "flu," epidemic nausea and vomiting, epidemic gastro-enteritis, "virus X," and what not. It is high time the profession settled on one name so that reports from different parts of the country will be clearly understood to denote the same clinical entity. Though long known in the East, it appears to have been relatively infrequent there until the war years. In California it has been endemic, with regularly recurring epidemics, for at least 35 years. It occurs principally in children, in annual fall and winter epidemics of variable proportions. Recently the disease was produced in humans experimentally by the oral feeding of throat washings and fecal filtrates,[13] proving it to be due to a filtrable virus, at least in this one particular Eastern epidemic.

Infectious Mononucleosis.—Before 1935, this was relatively a rare disease in California. Since that time it has become increasingly common, and at present enters into the differential diagnosis of most communicable diseases and all bizarre fevers. Severe anginose types may be distinguished from pharyngeal diphtheria only by the laboratory: the patients look, smell and act as though suffering severe diphtheria. However, cultures are persistently negative, and in time the typical Downey cell lymphocytes appear and the heterophile test becomes positive. A large spleen and enlarged lymph glands are suggestive. The systemic or typhoidal type may be impossible to diagnose for several weeks, the disease meanwhile resembling bacterial endocarditis, brucellosis, typhoid malaria, Q fever, intermittent fever, and other entities. Rose spots frequently appear on the skin, and the spleen enlarges. The morphological blood picture is variable, inconsistent and confusing, and often

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many days elapse before it becomes diagnostic. This is also true of the heterophile test. Incidentally, the heterophile test should be checked by absorption against false positives if the patient has received any serum. Results of blood cultures and Widal's test are negative.

In a recent autopsy study of a patient who was accidentally killed,[1] the disease was shown to have produced lesions of a focal infiltrative type in the brain, heart, lungs, spleen, adrenals, and testes. So far as the author is aware, this is the first report of lesions in the brain, although infrequently in clinical cases a meningeal picture or a Guillain-Barre syndrome does occur. Ruptured spleen is a common concomitant of the disease. In differential diagnosis the disease is often mistaken for infectious hepatitis. Pooled human convalescent scarlet fever serum, or immune serum globulin (gamma globulin) favorably alters its clinical course.

Infectious Lymphocytosis.—This entity unquestionably occurs more frequently than it is reported. The author observed three hospital cases last year. The lymphocyte count usually varies from 40 to 95 per cent of the total leukocyte count, which runs from 40,000 to 150,000. The lymphocytes are small to intermediate in size, being predominantly mature with nuclei of coarse, dark-staining, chromatic material, and with scant basophilic cytoplasm. These cells do not resemble the characteristic cells of infectious mononucleosis, and the heterophile test remains negative. In the three cases mentioned, the patients were children under seven. Mild coryza, slight cough, and sore throat were present. The cold agglutinin test was negative.

Reiter's Disease.—While atypical cases may not be possible to diagnose accurately, this disease is a real clinical entity and, in its typical form, would be hard to mistake for anything else, once one is familiar with it. The characteristic diagnostic triad is conjunctivitis, urethritis, and arthritis. Smears and cultures are free of organisms, but despite this, in some cases the disease has been erroneously diagnosed as gonorrhea on the sole basis of (sterile) urethral discharges. In a few instances, superficial ulcerative lesions develop on the buccal mucosa of the bladder or urethra, the glans penis, and the plantar surfaces of the feet. It has been mistaken for Stevens-Johnson disease, a type of erythema multiforme with conjunctivitis and buccal mucous membrane involvement. Dunham and associates [8] succeeded in isolating a filtrable virus from the conjunctival and urethral secretions of a patient with Reiter's disease. It grew in developing chick embryos, from which it was successfully inoculated into mice, 60 per cent of which developed conjunctivitis.

Mapharsen is the drug of choice in treatment.

Poliomyelitis.—Death during the acute stage of poliomyelitis is almost entirely the result of the patient's inability, because of the structures involved in the disease, to meet the basic, physiological, respiratory requirements of the human body. Generally speaking the only patients who die are those whose respiratory apparatus is involved. Therefore, early correction of respiratory deficiencies becomes imperative. Fundamentally, the problem resolves itself into two practical components: (1) maintain an open airway to allow adequate pulmonary ventilation; (2) where the patient's gaseous interchange is cut down through mucus secretions, pulmonary edema or atelectasis, to prevent or remove secretions and supply sufficient oxygen and air, or oxygen and helium. Respiration by means of the iron lung is indicated only when ventilation is deficient, which is to say, when the respiratory center is involved, or when there is a disabling paralysis of the intercostal muscles or diaphragm. In the former case, if aberrant and irregular impulses from the respiratory center cause the patient to breathe against the respirator, so that breathing is not synchronized with the machine, one or two doses of intocostrin (curare) almost invariably produces the desired effect. If obstruction to a free airway exists, tracheotomy should be performed at once. (We prefer the low type of tracheotomy.) Suction should be used during expiration. Kubicek and associates[18] point out that an arterial oxygen tension less than 100 mm. of mercury may injure vital centers. They state that the usual determination of the percentage of oxygen saturation of the hemoglobin, such as is advocated by Comroe,[4] is a useful adjunct in determining oxygen deficiency but may lead to erroneous conclusions regarding the actual amount of oxygen received by the ultimate cell. He points out, for example, that if the oxygen saturation of hemoglobin falls to 90 per cent from the norm of 96 per cent, the partial pressure of dissolved oxygen in the plasma falls from 100 to 60 mm. of mercury, a reduction of 40 per cent in the force which moves oxygen from the plasma to the cell. He further points out that the pathologic changes of the disease, itself, interposes physical barriers to the free interchange of gases, such as perivascular cuffing, edema of the tissues, and hemorrhage. By obtaining a free airway to increase the oxygen tension within the arteries, and by the use of medicinal oxygen, one may increase the transfer of plasma oxygen to the vital nerve cell and prevent destruction.

Waiting for cyanosis to indicate lack of available oxygen may be disastrous: By the time cyanosis is visible the flow of oxygen from capillary blood plasma to nerve cells has been decreased at least 65 per cent. This explains why cyanotic patients so frequently die—anoxemia of high degree was present long before any sign of cyanosis. The symptoms of anoxia are confusing. They are headache, tachycardia, hyperpyrexia (not usual in ordinary poliomyelitis), stupor, convulsions, or delirium. In therapy, equal parts of oxygen and helium are preferred to oxygen and air or to straight oxygen.

Comroe and Bothelo made arterial punctures and determined the oxygen saturation of the blood by the method of Van Slyke and Neill, and used their results to set the oximeter accurately at a known value.[4] Under such conditions the oximeter becomes

a reliable instrument and may be of greatest assistance in indicating the proper time to perform a tracheotomy.

Baker [2] believes that increase in pressure in the bronchial tree is of value in patients tending to develop pulmonary edema, and that fluids should be restricted to meet the requirements of the case.

If one is to judge from the clinical and laboratory findings as to the surviving patients, the strain of infecting virus in Southern California would appear to have changed with the epidemic of 1942, since the morbidity rate has been lower, the mortality rate higher, and the manifestations of the disease much more like those seen in the eastern United States than was previously the case. Over 400 cases occurred in Los Angeles County in 1947 and over 1,600 the year before. In previous years the situation in 1947 would have been deemed an epidemic.

Patients with no involvement of the respiratory apparatus are treated with hot blanket packs: The prone pack, the additional pack, or the full Kenny pack, according to the need of the individual. All these procedures have their usefulness in affording mental and physical relief to the patient. Physical therapeutic procedures aimed at the economic rehabilitation of the patient are started as soon as he is able to stand the necessary physical exertion.

Q Fever.—The disease is caused by Rickettsia Burneti, first described by Burnet and Freeman,[3] and it gets the name from the question marks they made in their note books while studying it, not from Queensland as is so often said. An organism isolated in this country by Cox [5] in 1940 has been found to be identical with R. Burneti. Unlike most rickettsia bodies, it is both intracellular and extracellular in tissues and exudates. It may be grown in developing chick embryos. Blood tests would indicate that many cattle workers in the western states have had the disease at some time in the past, and that it is an old disease, new only, in its recognition. Los Angeles County has had several hundred cases since 1946, although the diagnosis in some cases listed in 1946 was made only in retrospect and in the light of experience with new cases in 1947. The preliminary report emanating from the Public Health Service [16] shows that in Southern California most cases have been in dairy workers or those who lived around dairies. The organism was recovered from the milk of many widely separated dairies. No diminution in the quality or quantity of milk was noted; no demonstrable illness was found in any of the cattle of these dairies; local infection of the teats or udders without general symptoms is suspected in cows; no insect vector has been found.

Except in unusual circumstances, it is rarely possible to make a diagnosis of Q fever either early or during the clinical course of the disease. In retrospect, overlooked infections and incorrect diagnoses have been common. Patients acutely affected may have high fever, gross rales in the chest, blood-streaked sputum, mental confusion, meningism, pleural effusion, and moderate elevation of the blood sedimentation rate. Complement fixation or agglutination tests are rarely positive before the end of the second week or later. In a late stage of the disease, the blood may present the typical Downey-cell lymphocyte seen in infectious mononucleosis, but the heterophile remains negative.

After vague malaise, the onset is generally abrupt and the symptoms may be either of two types: (1) retro-orbital pain on ocular movements, accompanied by chills, chilly sensation, headache, and continuous or intermittent fever ranging from 100.5° to 106° F. Respirations usually remain under 30, the pulse is disproportionately slow for the fever, and cough sets in. (2) Onset is with conjunctivitis and rhinitis, after which it follows much the same course as the other. Differential diagnosis for the most part is entirely a laboratory procedure, and the diseases requiring consideration are, among others, primary atypical pneumonia, infectious mononucleosis, bronchopneumonia, rarely lobar pneumonia, influenza, psittacosis, dengue, brucellosis, typhoid, typhus, systemic tularemia, and pulmonary tuberculosis. In about 20 per cent of the cases a punctate rash appears near the end of the second week. It is ephemeral and not diagnostic, but has been known to last 72 hours, appearing first on the back, then the chest and abdomen. In over half the cases the chest involvement would be entirely missed unless chest roentgenograms were taken, and in this respect it closely resembles primary atypical pneumonia, the disease for which it is most often mistaken.

Rabies.—Recently for the first time a suspicion of rabies in a human was confirmed by injecting the patient's saliva into white mice.[7] Later, Negri bodies were seen in the brains of the animals when they were sacrificed after having contracted the disease.

Leprosy.—In view of many reported failures to induce leprosy by experimental inoculation through the years, the recently reported development of leprosy in designs tattooed on the arms of two Marines is noteworthy.[19] Both were tattooed at the same place in Melbourne, Australia, in June, 1943. Early in 1946 lesions developed in these tattooed areas, but not in those made at other times and places, and Hansen's bacilli were found present at the National Leprosarium in Carville, Louisiana. Cases of apparent accidental inoculation producing leprosy have appeared from time to time in the literature, however, and we have a case to add to this list. A Los Angeles high school boy worked in a stone quarry where many Mexicans were employed. These laborers habitually defecated on the floor of the quarry. (None of them, however, is known to have had leprosy.) One day a premature blast blew many fine particles of stone from the quarry into the skin of this lad's face. Two years later, granulomatous lesions developed around a number of these stony inclusions, and from them leprosy bacilli in typical arrangement were recovered.

Until recently, the treatment of leprosy consisted of providing good hygiene, plenty of rest, and a balanced diet; in short, the same treatment as that of uncomplicated pulmonary tuberculosis. Drugs were tried and found wanting. However, with the

advent of the newer sulfone drugs, new hope of arresting the disease is held out to lepers. Promin, diasone, and promizole have all produced sustained objective improvement in the lesions of leprosy.[12] Improvement has been slowly attained, but steady in progress. So far, promin seems to be the drug of choice.

Meningitis.—In the clinical teaching centers of the world, the approach to the diagnosis and treatment of the bacterial meningitides by different clinicians has been pretty much the same in principle, varying only in application according to individual thinking and experience, as to the number and site of spinal punctures done, the combination of chemotherapeutic agents with antibiotic drugs, etc. We believe that few experienced clinicians will be able to agree with the recommendations of Hoyne and Brown [15] in their analysis of 727 cases of meningococcal meningitis. They recommend that spinal puncture be done for diagnosis only, and in some instances even omitted for that; that penicillin be omitted in treatment as an unnecessary adjunct; that in treatment sulfathiazole is the drug of choice; and that the emphasis placed upon obtaining sulfonamide blood levels is unnecessary. Consensus in a voluminous world-wide medical literature is not in accord with these recommendations and findings: There is altogether too much evidence to the contrary. Sulfadiazine, or equal parts of sulfadiazine and sulfamerazine (which we have used in several hundred cases of meningitis of varying origin) have been shown to produce far fewer and less serious side-effects than sulfathiazole, and to be at least as effective therapeutically. In the various types of severe meningitis, the practice of doing lumbar punctures at four-day intervals, for eight to sixteen days after the cessation of active treatment, has often enabled us to forestall severe relapses. When the leukocytes in spinal fluid have been steadily diminishing, and follow-up punctures show a sudden or marked rise in the white cells in the spinal fluid, almost invariably a relapse is present; any other signs or symptoms may remain inevident for one or two days. We accept this, unless some reason is otherwise apparent to account for it, as an indication for the prompt resumption of therapy. It has been a particularly valuable procedure in the care of influenza-B meningitis cases. but at one time or another has been helpful in all types. Among the smooth strains of Haemophilus influenzae, six have been isolated. However, in over 90 per cent of the cases of influenzal meningitis, strain B is the cause. In this type Alexander's serum, sulfonamides, and streptomycin are all of value and we give them concurrently. In types other than B, the serum is omitted. If resistance develops to streptomycin, some strains are quite susceptible to penicillin as second choice.

Tuberculous Meningitis.—In cases of miliary tuberculosis with concurrent meningitis, or of uncomplicated tuberculous meningitis, streptomycin at times affords almost miraculous arrest, at other times total failure. The dosage has not been uniformly established, and reports almost too numerous to mention are appearing in current literature. We are finding that the proper dose has to be individualized to meet the needs in a given case, with procedures changed to meet requirements. When a relapse follows discontinuance of streptomycin treatment, resumption of therapy has often failed, due to the streptomycin-fastness acquired by the surviving organisms. That this is not always true is proven in the case of a patient at present undergoing treatment on our service. The patient, a three year old child, underwent 12 weeks of streptomycin treatment for miliary tuberculosis. The disease was arrested and the treatment stopped in May, 1947. Three months ago the patient was readmitted to the hospital with proven tuberculous meningitis, and streptomycin was given intrathecally and intramuscularly. Response was immediate, and at present, although treatment continues, the child appears perfectly well.

While dosage is varied to fit the requirements of age, sex, therapeutic response, tolerance to the drug, etc., the basic plan of streptomycin therapy for tuberculous meningitis, with or without miliary involvement, is to give 0.125 to 0.250 gm. intramuscularly every six hours, and 0.05 to 0.1 gm. intrathecally daily for six weeks. After that, treatment may be altered to fit requirements, but in any event usually continues at least three months.[6] If the same area of the spine is repeatedly and consistently used in doing daily lumbar punctures, the drug ultimately produces a yellow pigment in the spinal fluid. This pigment remains unidentified. Alternating cisternal with lumbar puncture will usually prevent its appearance. After six weeks, taps are only done two or three times weekly. From the treatment outlined, so far we have seen very few toxic reactions, none of them irreversible. Results have been as good as those obtained when larger doses were employed. With larger doses toxic manifestations appear in direct proportion to their increased size and the length of time they are used, and any deafness caused is permanent.

Chemotherapy and the Antibiotics.—In many infections formerly incurable, the outlook has been entirely changed through the proper selection and application of chemical and antibiotic agents to the treatment of them. The duration of many other diseases, and the rate of mortality from them, have been substantially reduced. Where the agents have been found effective, formerly useful procedures either have been altered or abandoned in nearly every instance. Although a detailed discussion of these remarkable drugs is not possible in this short presentation, a few things should be mentioned in passing. There is increasing evidence of many types that the pH of the urine should be maintained at 7.4 or higher when large doses of sulfonamides are used in the treatment of systemic infections, and that sodium bicarbonate should be replaced by the lactate or the citrates for this purpose. From the standpoint of preventing untoward side-effects, the use of two or more sulfonamide drugs in proper proportion appears to be an improvement over using any one of them alone, and therapeutically at least as efficacious. For the past 18 months we have been using equal

parts of sulfamerazine and sulfadiazine. This combination has been very effective therapeutically, and has produced far fewer disagreeable or dangerous side-effects than we have previously experienced in the use of sulfonamide preparations.

Eagle[9] has shown that a paradoxical reaction may appear in a certain number of strains of streptococci and staphylococci when the amount of penicillin in the blood is raised above the optimum level for their destruction. In other words, if penicillin is given in some of these infections beyond an optimum dosage, the procedure not only wastes the drug but causes failure to destroy the organisms. When possible, dosage should be calculated upon sensitivity ascertained in vitro.

Some chemicals have the property of preventing excretion of penicillin through the renal tubules,[20] thus producing very high blood levels though actually less penicillin has been given. This may be of value when inordinately high blood levels must be maintained, but none of the chemicals so far tested is fool-proof. Of those tested so far, caronamide seems to be the best, but it must be very carefully watched, for it invariably produces a reducing substance in the urine, believed to be a metabolite of the drug itself, as well as albumin, casts and red blood cells. These usually clear up in a few days after the drug is stopped.

Hyaluronidase, an enzyme, has become widely used as a rapid spreader through the tissues of fluids and sulfonamides. It overcomes the blocking effect of hyaluronic acid and allows rapid diffusion by increasing the permeability of the body's mucoid and connective tissues.

Para-aminobenzoic acid (usually abbreviated to P.A.B.A.) has become the drug of choice in treating the rickettsial diseases. The exception is Q fever, in which streptomycin is indicated. The dose of P.A.B.A. is 2 gm. every two hours. The patient should be alkalinized and the blood watched for any developing neutropenia.

The predicted threat of major introductions of tropical diseases into this country by returned service men has not materialized. However, in closing, it might be well to invite attention to the fact that in the past year cases of schistosomiasis, leishmaniasis, balantidium coli dysentery, fasceolopsis buski infestation, and Madura foot, among others, have been seen in this country.

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QUESTIONS AND ANSWERS

DR. CLINE: Dr. Bower, would you advise hospitalization of a patient with brucellosis previously inadequately treated with sulfa drugs, and the institution of treatment with sulfadiazine-sulfamerazine combined with penicillin or streptomycin?

DR. BOWER: Each case must be considered individually on its merits and a general answer cannot be given. We recently treated a 10 year old boy in whom the disease was proven, with sulfadiazine-sulfamerazine for three weeks, during the first eight days of which he received 0.5 gm. of streptomycin every six hours. For five years he had had fever daily, ranging from 99.6 to 102 F., and last year was inadequately treated with sulfadiazine alone. He has now remained completely well for over three months following the therapy. Penicillin is of no value in the treatment of brucellosis.

One should be sure of the diagnosis before resorting to such a strenuous regimen, and brucellosis may be one of the hardest of all diagnoses to make in the field of infectious medicine. The drug therapy outlined, powerful drugs are used, requiring that careful safeguards be set up for the patient. They are not fool-proof and are capable of causing great harm to the patient if not carefully watched. Also the treatment may have to be changed to meet changing requirements.