

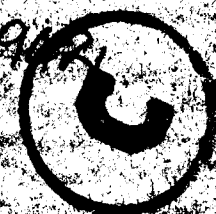
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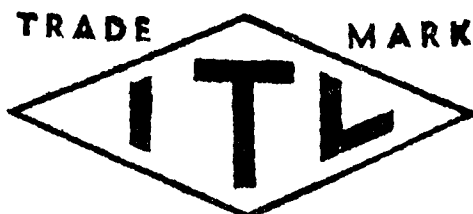


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MEDICO-SURGICAL SUGGESTIONS

(Established 1932.)

VOL. XI

SEPTEMBER 1942

NO. 9

THE ROLE OF THE PATHOLOGIST IN THE MANAGEMENT OF CANCER.

JAMES P. SIMONDS, M. D.

Chicago.

It was a great pleasure to hear the internist and the surgeon on this symposium state that the pathologist should be recognized as a consultant. My concept of the role of the pathologist in the management of cancer can be very briefly stated. He occupies the key position in any clinic or other organization devoted to this purpose. His contribution is of equal importance with that of the surgeon, the roentgenologist and other specialists.

A convenient and, for our purpose today, useful approach to the role of the pathologist in the management of cancer is a historical one. Prior to the middle of the last century, when Virchow began his work on the diagnosis, differentiation and classification of tumors, it was almost impossible to differentiate between cancer and many other diseases, particularly chronic inflammations such as the infectious granulomas. His diagnosis and classification were based upon the answers to two questions applied to each tumor. Is the tumor organoid—composed of parenchyma and stroma and thus resembling an organ; or is it histoid—composed of only one type of tissue? Is the tumor typical, and reproduces the normal histologic architecture of the organ or tissue of origin; or is it atypical and varies markedly from the normal structure of the organ or tissue from which it is derived? Organoid tumors were epithelial in character and were papillomas, adenomas or carcinomas; histoid tumors were of mesenchymal origin and were any of the connective tissue tumors, including sarcomas. "Typical" tumors were benign; the "atypical" ones, malignant. Virchow's classification of tumors were purely morphological.

This contribution of Virchow stimulated great interest in the subject of tumors in general and in cancer in particular. The literature teemed with papers based upon studies of tumors of all types and locations with their morphologic classification. It soon became

evident, however, that this method of study did not reach the problem of the fundamental nature of cancer. Such study was purely descriptive and not explanatory and seemed to end in futility. Perhaps the acme of this futility was reached in Bland Sutton's book, "Tumors: Innocent and Malignant," first published in 1894 and now out of print. It contains descriptions of the most unusual and bizarre tumors encountered in his own practice and found in the literature—mandibular tubercles on children, dogs and on the bust of a money-lender found in the ruins of Pompeii; cervical auricles on children, goats and the faun of Roman mythology; epithelial horns; and hairlip frogs. He even quotes Shakespeare in connection with hairy moles—Cressida's remark concerning Troilus' beardless chin: "Alas, poor chin, many a mole is richer" (in hair.)

Virchow's method and its results in the mere accumulation of facts concerning the morphology of tumors also led to a state of the utmost pessimism concerning cancer. The cases of this disease that were examined were almost invariably in a late stage, or even had resulted fatally from the tumor itself. It became the fixed idea of surgeons and pathologists that all cases of cancer were totally hopeless. It was only much later that they came to realize that cancer is at first a local disease and can be eradicated if it is in an accessible location. Although Virchow's work revealed nothing as to nature and causation of cancer and induced a feeling of deep pessimism concerning treatment of the disease, his method of asking two definite questions concerning each tumor is still used in diagnosis today by every pathologist, either consciously or unconsciously.

Interest was again aroused in cancer by the work of Jensen in 1903. Others had transplanted tumors from one animal to another but Jensen was the first to do this through a long series of animals and to formulate the principles that govern successful transplantation. In fact, the Jensen tumor is still being carried on in rats to this day. Again there was great activity in cancer research. Many investigators searched for transplantable tumors, and the Flexner-Jobling and many other strains were discovered and are still being transmitted from animal to animal of the same species. But it was soon found that transplanted tumors differed in several significant respects from spontaneous tumors. For example, they do not readily metastasize. Again, the search for the secret of the true nature of cancer seemed to have gone up a blind alley. But, as with the work of Virchow, at least one important contribution came from the study of transplanted tumors. It was found that they could be made to metastasize if they were massaged or roughly handled. This was a very definite warning to surgeons that spontaneous cancer, which naturally tends to metastasize, must be handled with great gentleness. It is quite possible that the lives of many patients have been saved by the observance of this principle.

After the exhaustion of the apparent possibilities of Jensen's method, again there came a period of pessimism in connection with the discovery of the true nature of cancer. But a few years later interest was again aroused by another discovery. Fibiger found in a cancer of the anterior stomach of a rat an animal parasite which was identified as one stage in the life history of a small cestode worm. The larval stage of this worm was passed in a certain species of cockroach. Fibiger searched the City of Copenhagen for a supply of these insect pests that were infested with this parasite. Finally he located what appeared to be an inexhaustible supply in a sugar factory in the outskirts of the city. But just as he was ready to begin work on his problem, the factory burned down and this investigation came to an end. It is doubtful whether it would have yielded much of value because of its evident limitations and uncertainties. But even so, several tumors of lower animals were discovered that were associated with the presence of an animal parasite, for example, the sarcoma of the liver of rats associated with the presence of *Tenia crassicolis* as described by Bullock and Curtis.

The present stage in the study of cancer began in 1914 when Yamagiwa and Ishikawa announced that cancer of the skin could be induced by painting the skin of rabbits and mice with crude tar. Soon thereafter Strangeways and his co-workers and others began to search among the numerous constituents of crude tar for the particular substance or substances that possessed carcinogenic action. When it was discovered that some of the carcinogenic agents were closely related chemically to sex hormones and to bile salts, work on the effort to discover the true nature, and the mode of origin, of cancer became widespread. Workers everywhere plunged into this problem. The synthesis by chemists of new substances which are not found in nature has revealed a great number of chemical compounds capable of inducing cancer.

The successful production of cancer by artificial means has yielded two very significant byproducts. It has pointed out the importance of chronic irritation in the mechanism of cancer production, and it has made possible the microscopic examination of the very early stages in the development of malignant tumors—a study that, if left to chance findings in human material, would progress very slowly. The recognition of early cancer has acquired special importance in connection with the role of the pathologist in the management of cancer, as will be pointed out later. Furthermore, only one trained in tissue pathology is capable of judging the results of attempts to produce cancer by any means because no agent can be said to have induced cancer until its presence has been proved by microscopic examination. As every pathologist knows, it is by no means always easy to differentiate between a very early stage in the development of malignant tumors and the hyperplasia of tissues that

frequently follows irritation and inflammation. Sometimes the pathologist is not sure and expresses a guarded opinion.

The attitude of both the pathologist and the surgeon toward cancer has changed very much during the last twenty-five years. The old pessimism has given place to hopefulness and even to optimism, sometimes, perhaps not fully justified. It is because of the discovery that early accessible cancer can frequently be completely eradicated that the modern methods of management of cancer have been developed and are being applied successfully in an increasing number of patients. Such management depends, for its success, upon the co-operation and collaboration of many different people.

In the first place it was necessary that the lay public be informed concerning certain signs that might mean cancer in order that those who show these signs will seek medical attention without delay and at a time when successful eradication is possible. The American Society for the Control of Cancer has been especially active in spreading information—because of the wholesome enthusiasm with which it has been spread one might almost call it propaganda—concerning hope of cure of accessible cancer if adequate treatment is instituted early. The education of the public concerning cancer has been so rapid that there is danger that it will outstrip the education of the medical profession in the best methods of management and treatment of this otherwise uniformly fatal disease. In any case, patients with cancer are seeking medical attention much earlier than heretofore and both pathologists and surgeons have been forced to become acquainted with the characteristics of its early stages—the one with its symptoms and physical signs, the other with its microscopic appearance. These may be quite different from those of the late stages of the disease when any sophomore medical student could make the clinical and microscopic diagnosis with only a small percentage of error.

The underlying principle of the modern management of cancer is co-ordinated team work. It is improbable that any one man can perform all of the duties and carry out all the techniques required. Any organization that sets itself up to manage cancer must contain at least a surgeon, a roentgenologist and a pathologist. This is the minimum requirement in personnel. There should also be added to the group an internist to advise concerning the diagnosis of cancer in deeper organs, and specialists who devote attention to each particular part of the body for advice in connection with tumors in special locations. It would be well also to have on such a team a chemist and a physiologist. These could offer many helpful suggestions concerning special features of this disease. Each member of the team should have equal standing. Each should be considered as a consultant who is able to add something to the full understanding of cancer and to its management in any particular patient.

It is my duty to discuss the contributions which a pathologist may add to the study of cancer by such a team. As stated earlier in this paper the pathologist occupies the key position in such a scheme. The final diagnosis depends upon him. For cancer can not be accurately diagnosed except by microscopic examination. No method of treatment of this disease can be said to be effective unless all of the cases treated have been proved to be cancer by this means. Furthermore, the pathologist on such a team should see every patient before treatment is instituted. In certain cancers, particularly of the breast, the pathologist should be present at the operation in order that he may direct the removal of a portion, or preferably all of the tumor, for quick frozen section diagnosis. His report will determine the extent of the operative procedure.

In any doubtful case in which a biopsy is to be taken the pathologist should be present to direct the taking of the tissue for microscopic examination. He will not attempt to dictate to the surgeon how he shall operate on the patient, but he should be permitted to dictate how and from what part of the growth the tissue shall be taken for biopsy diagnosis. For the success of a biopsy and of the treatment which follows depend entirely upon the securing of a piece of tissue that truly represents the lesion in question. A biopsy improperly made may be worse than useless because it may give rise to an entirely erroneous diagnosis and thus jeopardize the life of the patient and, incidentally, the success and reputation of the team.

It is not necessary in this symposium to discuss the value of biopsy in the diagnosis of cancer. This operation may be accompanied by some dangers but they are potential rather than actual. If properly made a biopsy is practically harmless. This is particularly true in cases of lesions of the breast in which the suspicious "lump" is entirely removed with an electric knife which kills all cancer tissue through which it passes. By means of a biopsy such unfortunate mistakes as the radical removal of a breast for a benign lesion or the amputation of a leg for a giant cell tumor of bone may be avoided.

The pathologist can also direct the performance of an Ascheim-Zondek test, or one of its modifications, for the purpose of determining the nature of certain tumors of the sex organs and for purposes of prognosis after operation. A consistently negative test after operation in a case that was previously positive will enable the physician to render a favorable prognosis.

The grading of tumors can be done satisfactorily only by a pathologist especially trained in tissue work. The principles established by Broders for grading cancers of the lip have been applied, with the necessary modifications required by the differences in the tissue involved, to cancers in various parts of the body. The grading to tumors is considered by many pathologists to be of less value than

the amount of work entailed. Grading of cancers of the tongue have been shown by Broders to have some prognostic value. Shields Warren has demonstrated a similar value in cancers of the cervix of the uterus. Two noteworthy difficulties present themselves in the grading of tumors. In the first place, personal judgment and experience of the pathologist are important factors. For this reason there may be a lack of uniformity in the grade in which a particular tumor is placed by different pathologists. In the second place, as Bell has aptly remarked, a cancer is as malignant as its most malignant portion. Hence the determination of the grade of any tumor may be misleading if it is based upon a general average of the microscopic appearance rather than upon those portions which show the greatest amount of anaplasia. However, in spite of labor involved and the pitfalls into which it may lead, the grading of tumors might well be made a part of the routine study of every case of cancer in any well organized tumor clinic. The results must always be interpreted in the light of the limitations of the procedure.

Finally, the pathologist may add much to the selection of the type of treatment to be instituted. This applies particularly to the problem of whether surgery or radiotherapy shall be the treatment of choice. Every pathologist is, or should be, familiar with the principles upon which the decision as to degree of radiosensitivity or radioresistance of a tumor is based. The diagnosis of each tumor should be accompanied by an opinion by the pathologist as to its probable degree of radiosensitivity. This will not only be of aid in determining whether surgery or radiation will be used as the chief method of treatment but also the likelihood of destruction of remaining tumor cells by radiation after surgical removal of the primary growth.

As a member of a team devoted to the management of cancer the pathologist can add much that will be of value to the patient and to the team as a whole. He will make the final diagnosis by microscopic examination. He should direct the surgeon in taking tissue for biopsy in order to obtain the most reliable results. By making an Ascheim-Zondek test in tumors of certain organs he can determine before operation the nature of the tumor, if the test is positive, and after operation can furnish a basis for an accurate prognosis. For whatever it is worth, he can determine the probable grade of malignancy of a tumor. He can give an opinion as to the degree of radiosensitivity of a tumor and thus aid in determining the type of treatment and the probable success of destruction by postoperative radiation of tumor cells that may have been left behind.

For his contribution to have the highest value the pathologist must be recognized as an equal of other members of any team of clinic devoted to the management of cancer. As stated in the beginning it is a pleasure to hear an internist and a surgeon state that the

pathologist should be recognized as a consultant in medicine. When this is done not only the management of cancer but the practice of medicine and surgery in general will be greatly improved. For the pathologist, whose speciality is disease, can add much to a study of its varying manifestations.

(*Illinois Medical Journal*, May 1942.)

THE ROLE OF PATHOLOGY IN MEDICINE

M. HERBERT BARKER, M.D.

Chicago.

On first thought, it is presumptuous for an internist to discuss pathology in medicine. Hearing the word pathology strikes me with awe. However, on second thought, we all employ, directly or indirectly, the old the rapidly increasing knowledge of pathologic phenomena in everyday medicine. From prenatal days until one dies, there is a constant parade of his problems passing before men of medicine who require specific knowledge about his anatomy, physiology and pathological processes. These problems require detailed information and experience with nutrition and resistance as well as the variations and fundamental functions caused by infection, endocrine, malignant and degenerative disorders. The man of medicine, assuming such responsibility for his fellow men, will call more and more for new and better assistance in the prevention, diagnosis, and treatment of disease. It is not surprising, therefore that the clinical pathologist is rapidly rising in our minds as the man to whom we must turn for much of this information.

We internists have been slow in appreciation of the broad aspects of clinical pathology. We are quick with our demands, but we have given little attention to facilitating the development and functions of the laboratories which so greatly amplify our diagnostic and therapeutic efficiency.

The rapid development of the use of vitamins has pressed the development for specific laboratory tests of vitamin deficiency which are being pursued on a wide front. Likewise the clinical demand for improved specific serum for the treatment of pneumonia vividly focused our attention to the great variations of the pneumococcus long observed in the test tubes of the bacteriologist. In this same vein, the newer knowledge of serology shows the reactions to be general biologic phenomena. The man who has had much experience with syphilis uses the positive serologic reaction as a reason to investigate the cause of the reaction. The so-called "typing" of the reaction, then, may tell whether it is one of general biological phenomena which may have been altered by temperature or by electrolyte, cholesterol, pigment or protein content brought about by

disease other than syphilis. Accordingly, Kahn has recently stated that such consideration should remove the objection to routine testing of blood, and it would enhance the control of syphilis. Certainly, there is much more involved in the care of syphilis than has been determined thus far by a test tube reaction. To pounce upon a human being with a barrage of powerful metals without careful consideration and continued observation is to be discouraged. It is under just such circumstances that the well trained clinical pathologist should be invaluable.

When Dr. Philip Hillkowitz gave his presidential address before the second annual meeting of the American Society of Clinical Pathologists in 1923 he stated that the "functions of the clinical pathologist are not ended when he or his technicians carry out laboratory tests ordered by the clinician—it should be emphasized that the pathologist is a consultant in medicine—his office a meeting place for attending physicians."

Amplifying the same thought, Dr. Wm. MacCarthy, the next president of the society, said in his address the next year, "The pathologist of 1940 will be the greatest correlator of all the facts about disease from the standpoint of the whole patient and not a narrow specialist upon an organ or technical laboratory procedure." Such well trained pathologists, he hoped, "might be the leading medical consultants in the year 1940."

Then, no doubt, 1940 seemed far away, but it has passed and I fear that we have far from reached the goal to which Doctor MacCarthy aspired. Not only have we failed to develop such an interlocking service which would embody biochemistry, physiology, serology, immunology and tissue growth, destruction and repair, but we have failed to develop the properly trained men to make such consultation possible. I understand that there are only about 500 certified clinical pathologists in the whole country. What, then, are the possibilities for improvement?

Obviously, much of the fault must lie with us internists who have not clearly visualized the scope and growth of modern medicine. Next, the clinical pathologists have not presented their work and their departments so that the proper understanding and development could be achieved. Certain fundamentals warrant our attention. The first is to broaden the base of our teaching of clinical pathology in our medical schools. The curriculum is more than crowded now, but room or adjustments must be made for necessities as they arise, just as we have always met the orderly growth of medicine. The old course in biochemistry was not popular when I was a student because it did not seem related to the care of the patient. In contrast now, biochemistry has a real place in the medical student's mind. Here at Northwestern University Medical School, one of the most popular courses is applied physiology which

employs a lecture on fundamentals by the chairman of the department, combined with one by a member of the clinical division, who correlates and applies the principles discussed.

I was surprised last week when polling the students of one of our medical clinics for their choice of topics to be discussed during the remainder of the term, to find that a high per cent of the class requested that a whole section be devoted to the use and interpretation of clinical pathology.

The second point suggested for improvement should be the encouragement of the selection by the students of clinical pathology as a specialty. His training would be long and carefully planned. My assignment to a luetic clinic for a year and a half was disturbing when at first it seemed to delay my course to a proficiency in cardio-vascular disease. However, it was a most valuable training and it caused me to appreciate fully a real syphilographer. One who is best qualified to treat syphilis must not only know serology, chemistry, dermatology, and diseases of the bones, liver, heart and blood vessels; he also must be skilled in neurology and psychiatry as well as one of the most delicate fields of psychology. No less training is needed for a skilled clinical pathologist who is to serve the modern hospital staff. Not only should the man who has thus prepared himself for the specialty of modern pathology be trained in clinical medicine, surgery and obstetrics, but he should have ample facilities for close contact with the patient in his hospital. Unless one keeps in close contact with clinical medicine he rapidly drifts away from its alert study collectively and certainly individually. The application and diversified study by laboratory means would be less apt to stop short if he were in direct contact with the patient and his problem. Our X-ray colleagues demonstrate this point well by requesting a statement about the case, and even then, they alter and repeat their studies very frequently after seeing the patient in the X-ray department. I am sure that diagnostic contributions would be obtained likewise by the ward walks of the clinical pathologist. The pathologist who has had a proper clinical training would add greatly to the teaching of the attending and resident staff if he were assigned a regular ward round. Again, a fine type of clinical research should be made possible for the hospital pathologist. His attending staff rarely has the time or the facilities to carry a problem to a successful conclusion. But the aggregate material thus assembled by the various members always supplies more excellent problems than can be solved in a decade. Research by the full time man often needs stimulation by contact with the pressure of disease in action. The observation that in pneumonia the chlorides often largely disappeared from the urine was discussed many years before explanation was made of where they had gone. The reason is not yet known. The clinical observation of the role

of salt in edema of heart or kidney disease has only recently received physiologic attention. However, some of these observations have opened the way for the study of the hormones which affect specific minerals of sodium and potassium as well as water balance.

The very common clinical problem of cardiac efficiency as influenced by specific minerals, diuretics and diets is only in its elementary chapters of investigation. The use of hormones and minerals in pregnancy, surgical shock, and burns, as well as in the new field of amino acids and chemotherapy all invite investigation. We do not know how an infecting agent causes disease, what changes occur in the host or what secondary changes of cellular processes or vital forces take place so that proper nutritional, mineral or chemotherapy can be supplied. It is important, therefore that adequately trained assistants and properly equipped laboratories should be put at the disposal of the clinical pathologist in order to carry out proper research in each community, which in turn stimulates every member of its staff.

When one of our profession may be induced to become a truly well trained clinical pathologist, we should make certain that his salary is commensurate with his training and his ability. Only then, may we expect to find our better students entering this fascinating field of diagnostic medicine.

Lastly, the lack of the average institution's facilities for making many specific procedures and the burden of their cost, brings great pressure upon the state and county for tax supported laboratories and civil service employees. The rate and size of growth of these institutions and their ultimate effect on the private practice of medicine as we know it today, deserves our urgent consideration.

The newer field of microbiologic methods should be pressed in the attempt to reduce cost as well as advance our knowledge. Dr. Ide in 1901 was aware that certain essential foods were necessary to grow yeast plants. Five of these essentials (called bios) are identified as thiamin, pantothenic acid, pyridoxin, arotin and inositol. Thus five of the seven vitamin B fractions may be studied with the yeast plant. This shock should stimulate research farther for simple and inexpensive means to supplant long, tedious and burdensome studies in animals. It is far from being humorous that the cockroach can be reared to maturity without vitamin A.

Thus it would seem that the role of pathology is greater than we are at present equipped to utilize. Growth will be along the line of broad fundamental training in medical schools, encouraging able men to enter the field of clinical pathology as a specialty, and supplying them with adequate facilities for clinical contact, clinical research and clinical teaching.

(*Illinois Medical Journal*, May 1942.)

THE MANAGEMENT OF MENTALLY HANDICAPPED CHILDREN

BERT L. BEVERLY, M.D.

Chicago.

A mentally handicapped child is one whose growth and development deviates from the average to such a degree that he requires special consideration in his home, in his school and sometimes from society. It is obvious that this is a broad definition and includes many groups of children. There are (a) those youngsters in whom there is a marked limitation of intelligence development with amentia on the one hand or extreme intellectual development with genius on the other hand. As pointed out by Wile "the inferior child, struggling against incapacity and over-demand, is no more a victim than the superior child whose mental gears are not enmeshed in educational dynamics. Among these groups are found an abundance of non-conformists in behavior, retardates, truants, delinquents and neurotics." (b) Children with special intellectual defects such as lack of cerebral dominance with poor visual word memory, making it very difficult to impossible for the child to learn to spell and read; (c) individuals with a disturbed emotional balance as from the epidemic type of encephalitis and (d) those with any of these defects and an additional physical handicap. In this paper I wish to discuss particularly that large and important group of boys and girls who are of less than average intelligence and especially the educable mentally subnormal group.

Cerebral agenesis is the most common cause of mental deficiency. Tredgold states that 80 per cent. of amentia is due to inheritance. Birth and other injuries, anoxemia, encephalitis, vascular accidents, poisoning (especially lead) and deteriorating diseases are other causes. The etiology is important in giving prognosis of the mentality of possible offspring.

The attitude of authoritative individuals towards methods of management of (educable) mentally retarded children has changed during the past twenty-five years. In 1912 Fernald called feeble-mindedness "the synonym of human inefficiency and one of the greatest sources of human wretchedness and degradation. The social and economic burdens of uncomplicated feeble-mindedness are only too well known. The feeble-minded are a parasitic, predatory class, never capable of self-support or of managing their own affairs. The great majority ultimately become public charges in some form. They cause unutterable sorrow at home and are a menace and a danger to the community." The psychological, criminological, sociological and educational literature of that period was filled with writings echoing this attitude. The State institution was offered as the logical

guardian of these children and the relief of parents and society from them. Gradually these ideas have given way to the obvious necessity of the constructive utilization of the mentally handicapped in the community and to the realization that there are many kinds of work which they can do efficiently.

Fernald was one of the first to introduce this new era for the mentally handicapped. In a follow-up study of all of the individuals discharged into the community from the Massachusetts School for the Feeble-minded from 1890 to 1914 he found that 35 per cent. were out-and-out failures. He also pointed out that only the most promising were allowed to go home. Similar studies since that time by Matthews, Storms, Folley, Brown and others has revealed that between 21 per cent and 40 per cent. were failures; i.e., they became serious behaviour problems or completely dependent for support. In other words, the results of training these children in State institutions have not been encouraging.

Another important reason for changing from institutional to home rearing of educable mentally subnormal children is the practical one—that it is not possible to provide State institutions for even a small percentage of the mentally handicapped. No state in the country has been able to provide an institution for as many as 10 per cent. of the group. For example, a survey of the Chicago public schools in 1932 by Strayer revealed that there were 21,000 mentally handicapped children of school age in Chicago at that time. It is estimated that there are over 200,000 children in Illinois with varying degrees of deviation from the normal. Furthermore, it is the higher grade of subnormal (those that have seldom been considered institutional cases) who need the most specialized attention and this help has always been recognized as a community problem.

The relationship of mental retardation to delinquency has been demonstrated many times. In a study of 1000 male juvenile delinquents in the Boston Juvenile Court, Glueck found that 28.2 per cent. were dull, 17.1 per cent. borderline and 13.1 per cent. feeble-minded, making a total of 58.4 per cent. who were mentally retarded. This is a far higher proportion of children of lower intelligence among delinquency groups than in the general population. In their studies of male and female delinquents, Glueck and Glueck found that 78.9 per cent. had been retarded two or more years below the school grades normal for their ages. Wile quotes the following figures, giving the percentage of delinquents who were mentally retarded; 37.8 per cent. in Boston in 1937; 29 per cent. in New York in 1932; and in Chicago in 1923, 58.6 per cent. of the 14 to 15 year-old boys going through the Boys' Court were retarded two or more years.

No one knows the percentage of those individuals who are dependent upon society for support the relief load in normal times—who are mentally retarded.

The figures quoted above do not prove that mental retardation, *per se*, is the cause of delinquency or dependency. These problems may result from mental retardation but more often they are symptoms of social maladjustment; they are signs of serious personality disorders which are on emotional basis more than due to intellectual defect. When we consider the fact that mentally deficient children are usually rejected and suffer the consequences of disturbed parental attitudes, and if it is recalled that they are generally denied the opportunities in school which gives them the satisfaction of accomplishment enjoyed by normal children and that after repeated school failures they are untrained for any kind of employment, we see reason for personality difficulties which might result in delinquency or dependency. From a study of individuals by Shimberg and Richenberg, it was found that those mentally retarded individuals with poor personality traits made a adjustment while those with good personality characteristics were far more successful. In this conclusion we recognize a similarity between the mentally handicapped and normal individuals; that is, adjustment in society depends upon normal emotional growth and development more than upon any other factors. Since the mentally handicapped have more serious environmental obstacles to meet than normal children, their emotional problems are greater and there is more maladjustment among them.

If we define a healthy individual as one who is able to use his resources and capacities in the most efficient manner in order that he may enjoy the greatest possible accomplishments for himself and society, it becomes obvious that an individual who is retarded mentally can enjoy physical and mental health. The problem, then, is to provide these children with the environmental needs for reaching a maximum growth and development. Here again the general determinants for mental health are similar to those for normal children.

The first essential for mental health is security in parents. This is the feeling that is given a child by parents who accept him as he is even though they may not be satisfied with him. Lack of affection of this quality is the biggest obstacle to mental health for mentally defective children. It is very difficult for parents to accept a mentally retarded child. Whether it is a rejection on a biological basis, their fears that the child will not be a credit to them, or the fear for the future welfare of their defective offspring, parents reject such a child and react to him by over-protection at some times, and over-correction in the effort to force him to act like a normal child at other times. It follows, then that the earlier a diagnosis of mental deficiency can be made and the more parents can be helped to accept the situation, the greater will be the security that they can

give the child. Parents, especially mothers, know when their babies are not normal; they are seldom fooled.

The infant who is hopelessly retarded should be placed in a public institution or under private care before the mother leaves the hospital. This should be done to protect the mental health of the parents and of the other members of the family.

Further security can be given the mentally handicapped child if he is accepted in the community and the school. Most teachers resent this type of a pupil and make every effort to rid their classes of them. The reasons for this attitude on the part of school teachers are probably the same as those of parents. There are the additional reasons that teachers are commonly held responsible for the child's failure to progress and the public lands the teacher who can demonstrate the best learners.

The second essential for mental health is but a corollary to the first and is expressed in allowing the child to grow up according to his own patterns. This is accomplished by understanding the youngster's growth pattern and gauging expectations to meet his ability so that he can get satisfaction from learning to do things up to the limit of his capacity. As pointed out by Healy, when expectations from a child exceed his ability he reacts by over-compliance on the one hand or over-defiance on the other. These reactions, based on insecurities and accompanied by fear and resentment, lead to dependency or delinquency.

It is generally recognized that mentally handicapped children require especially trained teachers and special educational facilities. It is also accepted that State programs with special aid to co-operating local school systems can best supply that training. Some states, notably Massachusetts and New York, have had programs for many years. More recently Pennsylvania, Wisconsin, Ohio, Michigan, California and finally Illinois are developing programs to meet this great need. Their programs vary in detail but follow the general principles that the children who are kept in their homes or in suitable boarding homes and have individual programs in school based on their individual capacities and scholastic needs develop the greatest mental health.

There are several types of education for handicapped children. There are those who can continue in regular classes in schools with a special consideration from teachers. Those children with more marked handicaps do best in special classes in regular school systems.—Especially when they reach the age at which vocational training is indicated, special schools in regular school systems may offer the greatest advantages. Finally there are those children with such marked handicaps—intellectual, emotional or both that they must be cared for or trained in state institutions.

A further aid to the mentally handicapped may be offered by a state protectorship set up with state funds for such special supervision, training and treatment as the child may need. This protectorship should be in co-operation with the parents, community and local school systems. It would relieve a great deal of anxiety which exists in the minds of parents relative to the welfare of their handicapped child after they can no longer supervise his welfare.

When children have security and are allowed to grow up according to their own patterns, they develop habits of self reliance which give them the self confidence and courage to face problems and solve them. Good habits are developed by doing the things they want to do. Health habits, play habits and habits of industry unconsciously develop while children are following a program adapted to their individual abilities and emotional needs. They accept responsibility for their behavior up to the limit of their mental capacity. Even though they do not have the intellectual capacity to do the abstract reasoning and complicated thinking of normal individuals their emotional growth is usually normal and they accept adults' norms of right and wrong to a remarkable degree.

Finally, mentally handicapped children need a place in society. In order to have security in their parents and community, as well as sufficient confidence in themselves, and to get satisfaction out of doing things they are able to do, it is necessary for them to feel that there is something they can do well and to feel that they are needed in society. After they have been trained in school to do a certain type of work, the co-operation of labour organizations and industry are necessary in order that they can find employment.

In any large program of this kind there will always be some children who can not grow up successfully because of home conditions. Supervision by public agencies is necessary in some cases and others require boarding homes; others will develop unmistakable signs of delinquency and may have to be placed in a State institution. The large majority, however, can have mental health and lead useful and happy lives.

SUMMARY.

1. A mentally handicapped child is one whose growth and development deviates from the average to such a degree that he fails to make an adjustment in his home, in the public or private educational institution and (or) in society.

2. It is now generally recognized that educable mentally handicapped children should be reared in their homes rather than in State institutions. State institutions are for uneducable children and for those who become uncontrollable behavior problems.

3. While there is a relationship between mental deficiency and delinquency and dependency, the etiology is primarily emotional rather than intellectual. Mentally handicapped children do not

have the environmental advantages of normal children and therefore develop emotional difficulties more frequently.

4. Mentally retarded individuals can have mental health as well as can those with normal intelligence.

5. Mental health is based on security in parents and on allowing children to grow up according to their own individual patterns. Children then form habits of self reliance which give them confidence in their ability to face difficult situations.

6. It is generally accepted that mentally handicapped children require especially trained teachers and special facilities in the school systems.

Extracts.

PULMONARY CONCUSSION.

BY G. R. OSBORN

Osborn compares cases of pulmonary concussion seen in civil life with those resulting from the detonation of high explosives. Among 94 necropsies of persons dying through road accidents there were 8 pulmonary lesions and 3 of these were comparable to bomb cases; these were in girls aged 4 and 8, and a boy of 7. The girl of 4 had been run over by a motor lorry, causing a fracture dislocation of the sixth and seventh cervical vertebrae. Her lungs showed multiple hemorrhages on the pleural surface; they were most numerous in the parts of the lungs which enter the phrenicocostal sinus of the pleura and posteriorly, but there was some rib marking on the lateral surfaces. The liver showed a small laceration of the posterior surface, and there were some peritoneal tears in the transverse colon. The girl of 8 had been knocked down by a motor car; she showed a little bruising over the left lower ribs but was otherwise unmarked. There was a profuse hemothorax from the left and a smaller one from the right side. There was a large tear near the hilus of the left lung, and contusions were scattered throughout both lungs. The liver was torn but the spleen was not marked. The boy of 7 was run over by a motor lorry. The trunk was not marked. The left lung showed a widespread hemorrhagic lesion about the hilus posteriorly, "rib markings" and a wedge-shaped contusion of the part in the phrenicocostal sinus. In the right lung there were innumerable pinpoint hemorrhages due to inhalation of blood from the left; the spleen showed superficial bruising. The other 5 pulmonary injuries were lacerations from fractured rib ends. The 3 cases in children with flexible chest walls were accompanied by

abdominal injuries. Review of road and bomb cases emphasizes the point that there is an abdominal component, to the force which produces the basal contusion of the lung. If this wedge-shaped lesion is present the question is not "Is the underlying spleen or liver damaged?" but "How severely is it damaged?" It may be a trivial bruise or a hopeless tear. Most war cases have come from bombed houses and shelters; pulmonary concussion appears to be infrequent in persons injured in an open bombed area. Pulmonary concussion should be looked for in every case from a bombed house. Factors which tend to minimize pulmonary concussion are advancing years, obesity, rigidity of the chest wall, the position of expiration and heavy clothing. So far too little attention has been paid to the fact that the abdomen is suddenly compressed as well as the thorax. Should operation be indicated the organ responsible for the abdominal hemorrhage is likely to be demonstrated by the side of the phrenicocostal sinus lesion. If this is placed posteriorly as well as laterally on the right side the tear in the liver will probably be inaccessible. Tears of the spleen are likely to be at the upper pole. Pulmonary concussion is not necessarily fatal. Death results from acute pulmonary edema and embolism. The latter may be prevented by regular active movements of the lower limbs aided by raising the foot of the bed to prevent hemostasis and by avoiding dehydration and other causes of hemoconcentration. Probably the most important act in treating hemorrhagic concussion of the lungs is to anticipate and prevent pulmonary edema and embolism. Knowledge that the case has occurred in a confined space will help the clinician if he keeps in mind that these patients are prone to suffer from carbon monoxide poisoning (Hadfield and his colleagues). The state of the patients is of greater importance than the size of the bomb or the distance from it. Knowledge of the details of the bomb may be misleading. In houses the positive pressure cannot fall until the walls and the roof are blown open.

(British Medical Journal.)

MORE OBSERVATIONS ON BRONCHIECTASIS

BY S. S. MISRA, M.D., (Hons.) M.R.C.P. (London.)

Physician, Medical College Hospitals, Lucknow, U.P.

The condition appears much commoner than was previously supposed due to faulty textbook pictures which described only the advanced variety. Young children often suffer from it. In the pathogenesis, the old view of fibrosis of the lung leading to dilatation of the bronchi is being modified by the newer theory of atelectasis

as an invariable precedent of bronchiectasis. The base of the left lung is the one most commonly affected. Radiologically, the varieties are cylindrical, fusiform, beaded, saccular or mixed. Clinically, in young children, bronchopneumonia after measles or whooping cough or small pox or influenza is the commonest history, while in the older people, unresolved pneumonia or chronic bronchitis are the important factors. The symptoms and signs in children are quite different from those in the older people. The signs are variable depending on the emptiness or fullness of the dilatations. In the adults, three varieties are described: firstly, chronic persistent cough with impaired percussion note on one or both sides with deficient or harsh breath sounds associated with medium or coarse crackling persistent crepitations—the bronchiectasis sicca; other varieties are recurrent haemoptysis variety and the classical variety of textbooks. Repeated sputum examinations are essential to exclude pulmonary tuberculosis. With the above symptoms and signs, diagnosis is not difficult, but wherever possible and practicable, a lipiodol skiagraphy may be done. The prognosis appears to be less grave than was previously supposed. In the treatment, prophylaxis is very important. To be successful, diagnosis must be made early and treatment with prolonged and persistent postural drainage should be adopted in young children with the above history and in adults after delayed resolution of pneumonias. Medical treatment consists of fresh air, good food, avoidance of cold and improvement of general health with cod liver oil and vitamins, and the use of creosote if the sputum is foul. On the surgical side, A. P., phrenic evulsion and thoracoplasty have been found to be useless. The operation of choice is lobectomy, which however, is a somewhat dangerous procedure.

During the present year, in about 8½ months (January to April and August to December) 26 cases were clinically diagnosed as suffering from bronchiectasis in the Medical Out-Patient Department.

An analysis of these 26 cases revealed the following important facts: 16 cases were lipiodolised and found positive, 10 did not agree to the employment of lipiodol. 12 cases were above 20 years and 14 cases below 20. All the cases except three were males. All cases came during the cold months and the majority were from the poorer class. In children all had recurrent bronchopneumonia after measles or whooping cough or small pox; in adults 5 followed lobar pneumonia, 5 chronic bronchitis, 1 influenzal bronchopneumonia and 1 acute abscess. There were six cases of bronchiectasis sicca, 3 of classical variety. Sputum examined was found to be negative. Lipiodol skiagraphy showed that the left lower lobe was involved in 16 cases, right lower lobe in 5 and both lower lobes in 5 cases and most of them were of the mixed or cylindrical variety; worm eaten appearance of fibrosis was seen in a few cases.

Clinical picture. Children give the history of recurrent attacks of bronchopneumonia. During the attack the child is usually seriously ill and responds well to adequate dosage of M. B. 693 or sulphanilamide. At times the physical signs present which is a suggestive point in diagnosis.

Adults—Bronchiectasis sicca. Six cases belonged to this series. In these the symptoms were usually chronic cough which did not respond to the sedative cough mixture. Previous history was variable—usually of chronic bronchitis or poorly resolved pneumonia. In the latter type, the signs were impaired note at once, deficient air entry or harsh breath sounds with persistent crackling crepitations, while the trachea and apex beat were pulled to the affected side. In the former variety the only signs were harsh breath sounds with medium or coarse crackling crepitations, while the apex beat and the trachea were in their normal position.

Recurrent haemoptysis variety—3 cases. In these cases the predominant symptoms was haemoptysis. Usually these are mistaken for and treated as cases of pulmonary tuberculosis.

Classical variety—3. In them, very little can be done. They present the classical picture. Only two cases are mentioned.

Diagnosis. Repeated and careful sputum examination is essential to exclude pulmonary tuberculosis, even in young children by examination of the vomitted matter. The author's experience has been that the signs mentioned above are so typical that lipiodol examination need not be insisted upon by the practitioner unless the patient can afford it. Apart from pulmonary tuberculosis, other conditions to be differentiated from are putrid bronchitis, chronic lung abscess, empyema, syphilis of the lung, and moniliasis.

Prognosis. This is distinctly hopeful. Several cases have been under treatment for from 6 months to 18 months and have shown improvement in general health and have been allowed to go back to their work. Real improvement or even cure is only possible if prophylaxis is insisted upon in every case and early postural treatment is instituted which is only possible if early diagnosis is made. This again can take place only when the newer ideas in early diagnosis are popularised.

Treatment. Prophylactic. This is of great importance and consists mainly in the early institution of postural drainage done for 10 to 15 months thrice daily. This has to be maintained for life in the not-so-recent cases. Deep breathing exercise are distinctly helpful. Nasal sinuses should also be attended to in all cases.

Curative.—(1) Surgical. A. P., phrenic evulsion and thoracoplasty have been found to be useless. Lobectomy and even pneumonectomy would be the ideal treatment provided the patient is agreeable to run the risks and suitable surgeons found to perform the dangerous operation.

(2) Medical. (a) General treatment should not be overlooked, and must be assiduously carried out; cod liver oil and the vitamins with good food, fresh air, sunlight, deep breathing and regulated exercises are important. Both stock and autovaccines have been found to be of little value. (b) If cough is unproductive, a stimulant expectorant with iodides will help a lot. If sputum is foul creosote in rectified spirit may be added to the mixture. Creosote baths are also helpful.

(3) Postural treatment is of great help, particularly for the prevention, in all children getting recurrent attacks of broncho-pneumonia and adults with delayed resolution. It should be started as soon as possible with the help of a modified Nelson's bed, raising the foot of the bed by 9 inches. The exact posture of the patient will depend on the site of lesion. Later, the patient can lie across the bed with his head touching the ground and the trunk upside down, while an attendant prevents him from falling off. This has to be done for 15 minutes, three or four times a day, 2 hours after the last meal, for many weeks and months. This posture is eminently suited for the majority of cases where the lesion is situated in the posterior parts of the lower lobes, specially the left lower lobe.

(J.L.M.A.)

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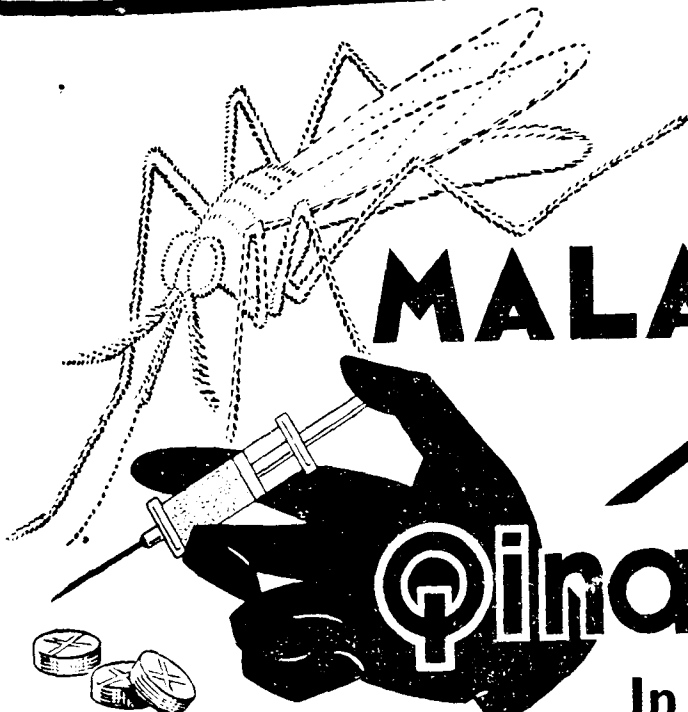
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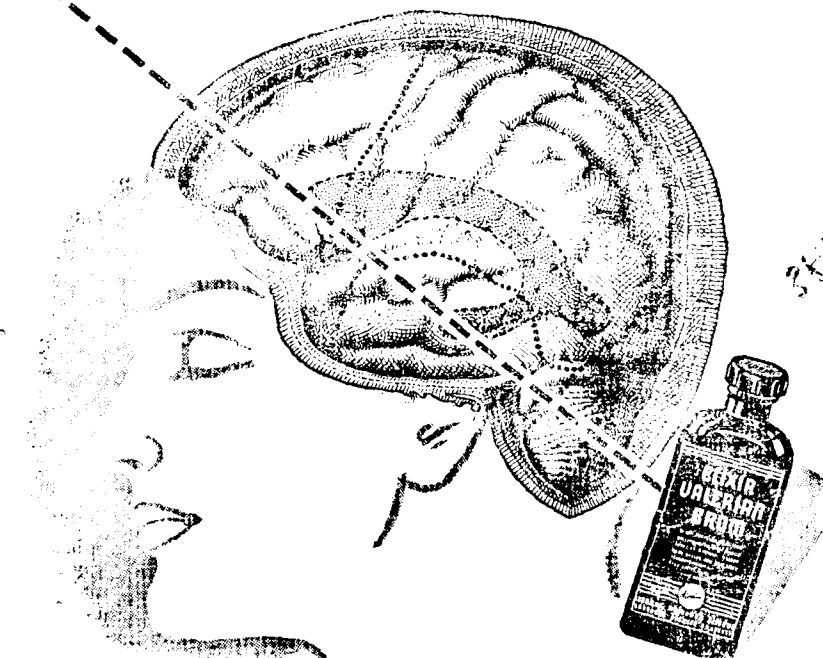
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GONOCOCCAL INFECTION IN THE FEMALE.

ROBERT M. LEWIS, M. D.

New Haven, Conn.

Some have suggested that, since both diagnosis and determination of cure are difficult in the female, our main efforts toward eliminating gonococcal infections should be exerted in treating the male, reasoning that if all men are cured there would be no question of further female infections. To me the matter appears closely analogous to the problems raised in malaria control. In both instances the patient must be cured and also prevented from infecting an intermediate host. The intermediate host, be she a woman or a mosquito, must also be prevented from spreading infection. In the case of women, they must be cured and kept from continuing the chain of gonococcal infections. The destruction of the mosquito is simpler than its cure.

Although the treatment of gonococcal infections of men, women, and girls differs but little, diagnosis and determination of cure are often vastly more difficult in the case of the female than in the male. Studies of the last 10 years have made it wise for us to forget much that was accepted as known fact before that time. We agree with Josh Billings—the immortal—who said, "It is better not to know so many things, than to know so many things that aren't so."

Ten years ago there was almost a superstitious fear of the dangerous possibilities of institutional epidemic contagion from single cases of gonococcal vaginitis of prepuberal girls. At the turn of the century Holt had reported a large number of cases of gonococcal vaginitis found in the wards of certain New York hospitals which had necessitated their temporary closure. Other epidemics of vaginitis in institutions were soon reported. Isolation technic in hospital care of patients was then but little understood or practiced. The diagnosis of gonococcal infections depended entirely on the microscopic examination of spreads, which we now recognize as a thoroughly unreliable procedure in all but acute infections in the female.

If possible, the treatment of gonococcal vaginitis of girls was even less satisfactory than its diagnosis. A host of antiseptic washes applications to the vaginal surfaces, suppositories or pastes had

for a day their supporters, but each was found inadequate. But little was known of the methods of spread from case to case, and it was almost believed that the gonococcus could flit about of its own volition infecting innocent girls with diabolic intent. Hospitals, schools, and child caring institutions often would not admit children with even a past history of gonococcal infection. The question was frequently raised whether children with such infections were ever cured. With no adequate studies of groups of untreated control cases, these questions were difficult to answer positively.

Nine years ago the author of this paper became convinced that local treatments, such as applications and douches, could only be expected to destroy organisms on the surface of the vaginal mucosa. As the infection always lies deep in the mucosa and submucosa, it could not be affected by such forms of treatment. In their place estrin therapy was proposed. Girls treated with estrogens develop in a few days a tremendous thickening of the epithelial layer which covers the vaginal mucosa. At the same time the vaginal secretions change from near neutral to strongly acid (pH 4.5 to 5). In artificial media an acidity greater than pH 6 is fatal to the gonococcus. Children treated as just described usually showed marked clinical improvement in 2 weeks time. In 3 or 4 weeks, or even sooner, it became difficult or impossible to demonstrate gonococci in their vaginal spreads. Control by means of cultures, though attempted, was not successful at the time this form of treatment was introduced. Culture technic was then in its infancy.

This method of treatment, I still believe, had advantages over the previous methods of local treatment. The introduction of a vaginal suppository did not upset the children under treatment as the forcibly given vaginal douches which preceded it had done. Clinical improvement following the use of estrogenic suppositories was sufficient to cause their widespread adoption in the treatment of gonococcal vaginitis.

Unfortunately, treatment with estrogens is still employed. As I shall mention later, it has no curative value and is not to be compared with the brilliant results now obtainable with sulfathiazole. Some 3 years ago the New York City Department of Health with aid from Government funds organized a group of physicians to study the whole problem of gonorrheal vaginitis in girls. I have enjoyed my association with these investigators and will presently review some of the conclusions which seem justified by the study. Many of them have been published by Alfred Cohn and his associates. At the outset we aimed to find out as much as possible about the etiology, pathology, and prognosis of gonococcal infections in children and hoped to determine the best way of treating such infections, as well as studying the community problems that they involved. We believed that a study of the behavior of the gonococcus in the simple structure of

the child's vagina might well throw light on its behavior in adult female infections.

It is generally believed that gonococcal infection of the vagina of the adult female is transient and uncommon during the years of estrogenic and menstrual activity. Before and after this era the epithelial layers of the vaginal mucosa are thin and few in number, and are highly susceptible to invasion by the gonococcus. In children the accessory glands of Skene and Bartholin are rarely involved. Gonococcal urethritis is common, but usually subsides in untreated cases before the vaginitis is cured.

Whether the glandular structures of the endocervix of the child are commonly involved, along with the infection of the vaginal mucosa, is still not positively known. In my own opinion, such infection is rare or does not occur. This is a difficult question to settle without an adequate number of autopsy investigations, and fortunately few children with gonococcal infections die, even from intercurrent disease. Infection of the portio vaginalis, the extension of the vaginal mucosa into the cervical canal, is the rule and has been observed in several post mortem specimens. One does not see any evidence of mucopurulent discharge from the cervical glands even in acute cases. This one would expect if such glands were the seat of an active infection. Tubal infection secondary to a gonococcal vaginitis is rare in the child (2 to 4 per cent) and even before the days of sulfathiazole usually subsided rapidly with rest in bed. I have, however, seen three such little patients who died with pelvic abscesses.

The New York study was based on 381 culture-positive cases taken from a total of 1,715 children with vaginitis who were examined. Of this culture positive group 32.9 per cent yielded negative spreads. The far greater reliability of cultures, as compared with spreads, was evident. The author has come to believe that spreads are rarely of any value in establishing a diagnosis of gonococcal infection of women or girls in any except acute cases where perfectly characteristic organisms are found in abundance. The possibility of error is too great when only a few shadow forms or poorly stained organisms are discovered. In the latter event opinions of experts are often at variance and a faulty conclusion is easily reached. About 45 per cent of the children yielded positive rectal cultures while only a few yielded positive rectal spreads. None of them showed evidence of proctitis, and the rectal infection proved only transient. Occasional rare cases of severe gonorrheal proctitis have been observed elsewhere. In no instance in the children studied did a gonorrheal ophthalmitis result from the vaginal infection.

All of us were impressed by the fact that gonococcal infection of girls was not as prevalent as we had supposed. In 1939 only 316

such cases were reported to the Health Department in New York City. In Baltimore during the same year only 136 cases were recorded.

A study of an untreated group of 42 girls used as controls was illuminating. In 78.8 per cent of them spontaneous cure occurred in 25 weeks or less, as shown by cultures, spreads, and clinical appearance. The remainder, while clinically normal, continued to yield positive cultures on occasions, interspersed with negative laboratory results. These we considered to be latent infections with an indefinitely long duration. A similar situation is often encountered in cases of adult gonococcal endocervicitis. While some patients yielded positive cultures until cured, not a few were found with only occasional positive laboratory reports interspersed among frequent negative results during the course of their infection. In one instance only 2 positive laboratory reports were secured from a girl during an observation period of 13 months, although cultures and spreads were frequently made during that time. Had these children been examined in the ordinary clinic, or during a visit to a consultant for diagnosis, the likelihood is great that they would have been classed as cases of nonspecific vaginitis.

The specialist often finds himself in a dilemma when children with a long standing vaginitis are brought to him for diagnosis. It is necessary in every case of this kind to explore the vagina carefully to eliminate the possibility of a concealed foreign body as a possible cause of the vaginitis. The usual spreads and cultures for gonococci and other organisms should be taken. Vaginal secretion diluted with normal salt solution must be examined to eliminate a possible trichomonas infection. Cultures for Monilia, yeasts, and Aspergillus infection are also indicated and are best made on Sabouraud's medium. If all these investigations prove negative, the possibility of a latent gonococcal infection, not shown by culture, still remains. If cultures cannot be repeated often, it is best under the circumstances to try the effect of a course of treatment with sulfathiazole. If all evidence of infection rapidly disappears as a result of such therapy, one is entitled to believe that the case was probably one of latent gonococcal infection. Before trying such a therapeutic test, of course, every effort should be made to secure positive laboratory evidence of a gonococcal infection.

We turn now to the study of a group of children treated with estrogens. Many of us had long been concerned over the number of recurrences observed among children after an apparent cure by estrogenic therapy. At first we believed that reinfection explained this disappointing situation. The New York study group soon discovered that, although the children treated with estrogens improved clinically and spreads (which were the previous criteria of cure) frequently became negative, cultures often remained

persistently positive. When cultures were used, it soon became apparent that real elimination of the gonococcal infection did not occur any more quickly or in any greater percentage of cases than in the group of children who received no treatment whatever. It is evident that the estrogenic treatment was but a palliative procedure which carried patients along until spontaneous recovery occurred.

With the use of sulfanilamide, about 50 per cent of the girls treated were cured within 2 weeks.

Sulfapyridine in doses of $\frac{1}{2}$ grain per pound of body weight, not exceeding 2 gm. per day for from 7 days to 10 days, given in 4 divided doses at 4 hour intervals rapidly cured 85 per cent of a series of 89 cases.

Sulfathiazole given in the same manner is preferable to sulfapyridine because it is less toxic. The percentage of cures resulting is equal to the figure just given for sulfapyridine. None of the children so treated showed evidence of toxic effects from the drug. Treatment with sulfathiazole is now the method of choice and is usually dramatically successful. Patients so treated should be followed for 6 months before being pronounced cured.

Often it is difficult, or even impossible, to determine the source of infection in individual cases. In the past it was generally believed that if newborn female children were infected at birth, clinical evidence of such infection would appear within 2 or 3 weeks. It now seems possible, or even likely, that birth infection may be latent for months before the onset of symptoms. Different studies of infected children have shown that in a large percentage of such cases (probably 50 per cent) a parent or other member of the family, also has gonorrhea.

Intensive study of a limited number of cases of gonococcal vaginitis by a psychiatrist convinced us that sex experimentation was frequent among infected children who were 6 years of age or older. Among them one child 8 years old gave a history of promiscuous intercourse with boys. Another 6 years of age was also found to have had intercourse. Rice, in a similar study made in Boston, found that 8 of a group of 15 girls with gonococcal vaginitis gave histories of sexual contact with boys or men. To us it seems likely that school infections are more apt to be acquired in this way than any other.

Nelson has long held the opinion that children with gonococcal infections should be excluded from school only as long as a free vaginal discharge persists. We are in full accord with this point of view. In one institution that came under the notice of Benson, a group of infected girls used the same toilets as an uninfected group for many months. None of the uninfected children developed vaginal or other infections. During the period of our study no epidemics of

gonococcal vaginitis occurred in the schools of Greater New York, although children often attended their classes for many weeks before their infection was found or reported. None of us has ever seen a case of gonococcal vaginitis that could be proven to have its origin at the toilet. We believe that the practice of setting aside special hospital wards for children with gonococcal infections is unnecessary. Ordinary present-day isolation technic is sufficient to prevent cross infections from occurring.

The course of adult gonococcal infection parallels that of childhood infection remarkably. Instead of a vaginitis, urethral and endocervical infections are of primary importance. The more serious upward extension to the tubes producing an acute, and later chronic, salpingitis is probably far less common than generally supposed. As a guess, I venture to say that not more than one infection out of six is followed by tubal infection. If the patient is under adequate treatment with sulfathiazole, such a sequel almost never occurs. The same rule holds true in the case of the male; *i.e.*, a patient with an anterior urethritis under adequate treatment with sulfathiazole very rarely develops posterior urethritis, prostatitis, or epididymitis.

Our present conception of the course of gonococcal endocervicitis seems to stand in need of sweeping revision. In the past it has been taken for granted that the initial gonococcal infection ordinarily produced a red, swollen, acutely inflamed cervix accompanied by a free discharge of mucopurulent secretion—the textbook picture of acute endocervicitis. As the acute inflammatory phase subsided, the cervix, it was thought, necessarily remained enlarged and swollen, with granular or eroded areas about the os and a continuous mucopurulent discharge. The practitioner was led to believe that there was a typical picture characteristic of gonococcal cervical infection could he but recognize it.

We now realize that the acute inflammatory reaction, which we have been led to expect, is not a necessary prelude to chronic endocervical infection with the gonococcus. In fact, there is reason to believe that such a response at the onset of the infection is the exception, rather than the rule. Occasionally, as a result of public education, women now present themselves for examination at the office with the man responsible for their infection. Under such circumstances accurate data regarding the time of infection may be available. In such an instance, 10 days after exposure the author has seen a patient with an apparently normal cervix except for an excess of mucus secretion. Both spreads and cultures showed gonococci in large numbers. In cases of chronic infection the experienced examiner can detect more often than the tyro some of the stigmata, such as evidences of infection of the cervix, thickening of the bases of the broad ligaments, indurated or tender tubes,

eversion of the urethral mucosa or the telltale reddening of the orifices of the accessory glands. Sometimes even these evidences may not be apparent, and positive laboratory evidence may then come as a surprise. A carefully taken history may yield a valuable clue but is usually not helpful unless trouble and time have been taken to win the patient's confidence beforehand. Pelouze (Bull. New York Acad. Med., 17: 39—44 Jan. 1941) in discussing the treatment of gonorrhea in the male with sulfanilamide remarks that asymptomatic carriers, dismissed as cured, may transmit the infection to women, who in turn become asymptomatic carriers of the gonococcus. He adds that such a woman may again transmit the infection to still another man, who will develop a typical acute urethritis. The implication contained in this discussion is evidently that sulfanilamide treatment of the original patient has transformed the gonococcus so that the woman secondarily infected may become a latent carrier of the disease. My own interpretation of this situation is different. We are becoming aware that in many women gonococcal infection produces no striking inflammatory reaction. Such cases may well be regarded as latent carriers and are by no means rare. Pelouze's patients were probably not unusual, and the course of their infection was presumably uninfluenced by the sulfanilamide given to the men who were responsible for their infection.

Until now good standards of medical practice have required the demonstration of gonococci by laboratory methods before a positive diagnosis of gonorrhea was established. In recent acute infections of women and girls, positive spreads and cultures can ordinarily be secured and are rightly considered proof of the diagnosis. In some instances we have followed untreated patients for as long as 6 months, while spreads and cultures remained positive. Sometimes only positive cultures are found while spreads early become negative. Usually after a few weeks or months it becomes impossible to find any positive laboratory evidence of infection, although gross inspection of the cervix assures us that infection is still present. Provocatives under such circumstances usually give us little or no assistance. It is probable that the invading gonococci lie in the submucosa, or in the depths of mucous glands which have been sealed. Earlier in this paper the case of a child was mentioned from whom in 13 months of investigation only two positive cultures were secured. We have seen not a few women with gonococcal endocervicitis with similar laboratory records. A much larger and more thorough study of prostitutes by Mahoney and his associates convinced them that it is impossible to secure laboratory evidence of gonococcal infection in a great majority of cases of long standing gonococcal endocervicitis. In his series of clinically infected

prostitutes, only 21 per cent. gave positive cultures, while far fewer of them yielded positive spreads.

In my own considered opinion, spreads are only of real and great value in the diagnosis of gonococcal infection of girls or women during the acute phase of their infections. At this time if large numbers of characteristically formed, gram-negative intracellular diplococci are found, a positive diagnosis is justified. In the later stages of the disease I believe that a study of spreads is apt to be more misleading than helpful unless thoughtfully interpreted by a well-informed clinician. Too often a positive diagnosis has depended on the finding of a few poorly stained diplococci or organisms thought to resemble diplococci. I am satisfied to confess my own inability to identify gonococci with any certainty under such circumstances. I have seen many cases where errors in doing so have been responsible for most unfortunate situations.

Van Slyke in a recent meeting of the New York City Health Department reported the results of an extraordinarily interesting study of the value of laboratory methods in the diagnosis of chronic gonococcal endocervicitis in women. Spreads were taken from the endocervix of 664 prostitutes who gave clinical evidence of infection. Three competent observers examined the same Gram-stained slides. One of them reported 160 as positive for gonococci, another found only 64 positives, and a third found only 44 of them positive. The microscopists by whom this study was made were all trained experts in this field of work. If these are the results under ideal conditions, can we have any confidence whatever in spread diagnosis of this type of case? Van Slyke further reported that in the same group of patients cultures were made by three excellent laboratories using the same preparations of media. Identical swabs from the cervix in peptone broth were used by all three laboratories. The results of the cultures in the three laboratories were in total agreement in only 75 per cent. of the culture positive cases. In a further investigation Mahoney and his associates report that 1,598 prostitutes with presumable gonococcal infections were studied with repeated cultures. In only 21 per cent. was it possible to confirm the clinical diagnosis with cultures. In most sections of this country the use of cultures to detect gonococcal infections is, of course, not possible. The futility of taking spreads in rural areas with untrained personnel is well illustrated by the following observation. In a certain maternal health clinic in a southern rural community, serologic examinations for syphilis of pregnant patients was undertaken. Of 6,888 patients, 16.1 per cent. yielded positive reactions. It is fair to assume that in this group of patients gonococcal infections must have been frequent. Spreads were taken from the same group of patients and examined in laboratories of the State for gonococci.

Of the entire lot, only 4 spreads were reported as positive. No comment is necessary!

In the New Haven City Clinic before the advent of sulfathiazole, the average duration of observation and so-called treatment for women with gonococcal infections was 14.3 months before they were considered cured. Twenty per cent. of such patients became delinquent. During the past year this figure has been reduced to 10 per cent. A group of 12 such patients on admission to the clinic gave positive laboratory evidence of gonococcal infection. They were kept under observation for periods ranging from 7 to 67 months. After the first few months, positive spreads could rarely be found. Cultures and spreads finally become entirely negative, but the clinical appearance of the cervix suggested latent infection. When treated with sulfathiazole the appearance of the cervix in every case rapidly improved or became normal. This again seems to show that infection often persists in spite of laboratory evidence to the contrary.

In the New Haven Hospital Gynecological Dispensary, an excellently run clinic with ample laboratory facilities near at hand, only four cases of gonococcal infection were found among a consecutive series of 2,500 gynecologic patients examined. Evidently many must have been overlooked.

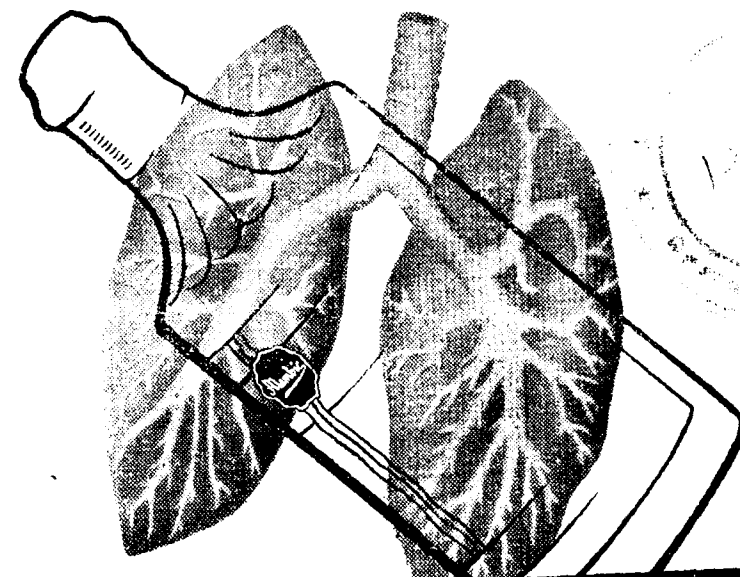
Until now good practice, as previously mentioned, has demanded laboratory identification of the presence of the gonococcus before a diagnosis is made or reported. The facts just outlined convince me, and I hope the reader also, that we have been misled by our dependence on laboratory reports. Obviously the greater number of chronic gonococcal infections in woman will remain unrecognized unless we radically alter our attitude. Obviously, also, the diagnosis must often be made with only the support of a carefully taken history and a painstaking pelvic examination backed by sound clinical judgment. Ordinarily the physician has refused to make a diagnosis of gonococcal infection without positive laboratory support lest he embroil himself in legal or other difficulties. He must, in the future, be encouraged to use careful clinical judgment and stand by it. Certainly in the past, before blood cultures and Vidal reactions were available, no physician hesitated to recognize and label cases of typhoid fever, even though many errors resulted. It is important that our city courts recognize a clinical diagnosis as sufficient to commit women for treatment. Demanding laboratory evidence or reliance on spreads alone is a mistaken policy. I by no means suggest that we should omit every possible effort to secure positive laboratory evidence in cases of gonococcal infections in women, nor do I suggest that we return to the day when leukorrhea was considered sufficient evidence of such an infection. I do suggest that we recognize the fact that laboratory help is not available in the

majority of cases of chronic gonococcal endocervicitis. I also suggest that spreads, as now generally used, are often misleading after the acute phase of an infection is passed, and that in chronic cases of infection of girls or women this laboratory procedure is rarely helpful and often misguides us. At best, and only when spreads have been examined by experts, positive or negative reports may be considered as supportive evidence in arriving at a diagnosis. Positive spreads, even under such circumstances should not be regarded as conclusive evidence of infection. Negative reports are, of course, valueless. The usual procedure in qualified laboratories is to render a report stating that gram-negative intracellular or extracellular diplococci morphologically resembling gonococci were, or were not, found. Only too often this sort of report is interpreted as meaning "has (or has not) a gonococcal infection."

Rarely in cases of specific chronic endocervicitis after a series of negative findings one may suddenly encounter a spread which shows large numbers of gonococci which stain and are grouped as in an acute case. Here we are either dealing with a reinfection or possibly a sealed off focus may have discharged its contents. This is, however, an uncommon occurrence. Suspicious spreads may point the way to repeating cultures which have been negative. Ordinarily neither negative nor positive spreads are reliable evidence of infection or its absence in chronic cases. When clinical judgment suggests the likelihood of a gonococcal infection, women and girls should be treated with sulfathiazole. If the responsible physician is unable to make even a clinical diagnosis with reasonable certainty, sulfathiazole treatment can be given without mentioning the possibility of gonorrhea to either the patient or the health authorities. The many pitfalls, difficulties and errors in making clinical diagnoses without positive laboratory findings to support us, will be admitted by everyone familiar with this field of work. I do not mean here to minimize them.

In New Haven we have had adequate supplies of sulfathiazole since June 1940. Rapid cures have reduced the daily attendance of our city clinic by 50 per cent. In both men and women we have used the dosage established by others—2 gm. per day in four doses, given for 12 days. In about 100 cases so treated no case of severe drug intoxication has developed. We believe that our rate of cure is about 85 per cent. or better. Reinfections, sometimes frankly admitted by patients, make an accurate calculation difficult. Patients with gonococcal infections who are pregnant respond well to treatment with sulfathiazole. Under such conditions sulfathiazole therapy is not harmful to either mother or child and is urgently indicated. In his clinic at Fall River, E. L. Meritt, probably influenced by Hawthorne's "Scarlet Letter", makes it a practice to paint the genitalia and their surroundings of uncured patients with

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mercurochrome. His object in so doing is, of course, to present a warning sign to future candidates for infection. We plan to follow his example.

Physicians are rapidly becoming aware of the brilliant results obtainable with sulfathiazole therapy in the treatment of gonococcal infections. We believe that success in eliminating such infections depends at present on our willingness to recognize the limitations of laboratory procedures and particularly the fallacy of relying on spread reports in chronic female infections. The need of a reliable diagnostic procedure, such as a compliment reaction or skin test, is obvious. At present none such exists. Diagnosis in the majority of our cases among the female population must depend largely on the willingness of the physician to use sound clinical judgment and to act upon it.

MANAGEMENT OF CHRONIC ARTHRITIS.

EUGENE F. TRAUT, M.D.

Chicago.

The so-called specific arthritides are the best understood group of chronic arthritis. They are due to generally accepted causes such as injury, gout and the definite infections: tuberculosis, gonorrhea and undulant fever.

Although the causes are well recognized, effective, particularized therapy exists only for gonorrhea and, to a less degree, for gout. Sulfonamide preparations and fever cure most cases of gonorrheal rheumatism. Suitable diet and hygiene can in large measure prevent exacerbations of gout. Colchicum and the dangerous cinchophen are almost specific in relieving the acute manifestations of gout.

The great group of nonspecific arthritides are helpfully classified by the American Rheumatism Association according to the constitution of the patients, the changes in the joints and the clinical propensities of the joint disease into atrophic and hypertrophic types.

Atrophic arthritis predominately afflicts youth and young adults of the asthenic, Stiller or ptotic constitution. The predisposed type of individual tends to be tall, thin, narrow faced, long-necked, low in energy. His extremities are long and the musculature is sparse. The blood pressure is usually low. Such patients are chronically fatigued. They are prone to upper respiratory infections. They were formerly classed as "phthisical" because of their long, narrow chests.

Chronic arthritis peculiar to this asthenic type of person tends to ankylose in grotesque positions leading to the synonym of arthritis deformans. It is commonest in women. The small joints are predisposed. It often starts in the fingers progressively implicating the wrists, ankles, feet and elbows. Feeding is often difficult due to ankylosis of the jaw joint. The symmetry of its progress is striking. The soft tissues attached to the skeleton participate in the deforming process. The muscles atrophy. Contractures appear. The periarticular tendons and ligaments and joint capsules thicken. Even in a bed-fast victim x-ray may show no intra-articular pathology. The cartilaginous surfaces may be quite smooth. Excess of synovial fluid leads to swelling of the joint, often fusiform. This may fluctuate. Then the x-ray shows an increase of joint space. Later destruction of cartilage and of the specific serous secreting membrane, the synovium, causes the joint space to appear narrow. The destruction of cartilage may be circumscribed leading to a punched out appearance of the joint surface. Coincidentally the bones lose calcium becoming increasing transparent. The type of arthritis may selectively attack the spine producing the poker back or Strempell Marie deformity. An additional, painful complication of the spinal type is the radiculitis expressing itself as sciatic, intercostal or brachial neuralgia.

The patient with atrophic arthritis wastes, looks and feels sick. Anemia of the secondary, small-celled type develops. Appetite fails. A low-grade, septic fever is often present. The sedimentation time becomes rapid. We have found streptococci in the blood of many of such patients. This clinical picture has led to the name of chronic infectious arthritis. In children it is called Still's disease.

Treatment of these victims is analogous to that of another chronic infection, tuberculosis. To secure the best co-operation and prevent the migration of patients from doctor to doctor the patient with chronic, progressive, ankylosing, deforming arthritis must be given the proper perspective. He must be told that he will be sick for months, that recurrences and set-backs are part of the usual clinical course, that he should not expect too much from any one therapeutic procedure be it surgical as extraction of pulpless teeth; medical as the use of any new drug or type of injection; or physiotherapeutic as fever, massage or a light. He must be assured that he has a good chance to get well. With very few exceptions these patients can be truthfully promised marked benefit from meticulous care carried out for months. Those with rheumatoid characteristics preponderating (less tendency to ankylosis) are as likely to be cured or freed of symptoms as patients with peptic ulcer or such infections as tuberculosis or chronic gonorrhea.

The management involves foremost such hygienic measures at rest, definitely prescribed and sufficiently prolonged. In many instances complete temporary invalidism will have to be accepted as the shorter and ultimately cheapest road to recovery. Partial casts or splints may be needed to maintain rest of involved parts. Salicylates remain the most commonly used drugs for joint pains. Salicylates given as acetylsalicylic acid with calcium salts are demonstrably superior. For two years I have used a mixture of acetylsalicylic acid and calcium glutamate. In the stomach this mixture forms the very soluble, active calcium acetylsalicylate. As contrasted with aspirin this does not irritate the gastrointestinal tract and gives prompter effect. It is usually well tolerated by patients intolerant to aspirin.

Dietary adjuncts as vitamins and calcium and phosphorus are important. The best diet seems to be one carrying an abundance of minerals, vitamins B, C and D and enough roughage to secure good bowel activity. Calories are sought in the fats rather than in carbohydrates. A high intake of meat and eggs is necessary to combat anemia and compensate for the rapid protein break-down. It is generally recognized that a generous protein intake increases resistance to any infection.

Physical therapy, supervised by a physician, is indispensable. Heat can be best applied locally with heat lamps or bakers. Hot compresses are especially valuable in the exacerbations. Contrast baths lend themselves to treatment of subacute or chronic arthritis of the extremities. Wax baths can be best utilized in treating the hands or feet. Fever therapy has proved of temporary benefit in a few stubborn cases.

Patients with atrophic arthritis tend to assume the posture of fatigue: kyphotic upper spine with winged scapulae, lordotic lower spine with protuberant abdomen, flat feet. They are coached in the postural exercises of Goldthwaite and Osgood and corrective foot exercises are stressed. Proper shoes are definitely prescribed and are adjusted to the patient's needs by soft felt padding.

Diathermy is the most convenient and effective means of heating deep tissues. Short wave high frequency has practically replaced conventional diathermy. In my practice it is applied by means of the induction field utilizing a cable electrode.

Floundering in skepticism of procedures long regarded as helpful in some patients with arthritis but not curative in all, trying in vain to reduce treatment of disease to a physiological experiment, some writers discredit the most helpful doctrine ever advanced in the treatment of chronic arthritis, the theory of focal infection. While infected tissues have undoubtedly been removed without obtaining hoped-for improvement, Billings' and Rosenow's teachings have done more to reverse the progressive invalidism of chronic

arthritis than any other suggestion thus far advanced. In spite of a tendency to belittle focal infections, removal of pulpless teeth in adults or tonsillectomy in more youthful patients results in recovery or improvement too frequently to be called coincidental. Perhaps the best evidence for the causal connection of infected foci has been the exacerbation or intensification of symptoms ensuing after inopportune surgical disturbance of suspected foci. To avoid this I permit tonsillectomy, tooth extraction, or prostatic massage only after the patient's general health has improved or joint manifestations have subsided.

Rejecting infection as one of the causes of chronic arthritis has led to abandoning vaccines in its treatment. I regard streptococcus vaccines properly made and intelligently used as an important aid in the recovery of that class of patients presenting findings characteristic of infection. Vaccines also may be used to prevent the exacerbations occurring after removal of infectious foci. Vaccine is injected subcutaneously in subreaction doses. I have found vaccine superior to other forms of foreign protein therapy.

I have never seen any improvement from sulphur or iodides. Change of climate has in my experience never resulted in any more benefit than would be expected from removing the patient from his responsibilities.

Gold therapy is undergoing a revival. It is my practice to reserve it for patients with an arthritis, predominantly atrophic, not responding to other measures. Its dangers are universally recognized. I have yet to see a patient respond favorably to gold injections, regardless of preparation used, after failing to benefit from the more commonly used, safer methods. It has seemed to me that the patients most improved by gold have been those with some evidence of liver damage resulting from the medication.

Careful x-ray treatment often relieves the symptoms of Marie-Struempell's disease, poker back or atrophic arthritis of the spine.

The patient with hypertrophic arthritis is usually past middle life. His constitution tends to be sthenic, robust. He boasts of never having been ill. He has often led a vigorous life. He tends to be overweight. His face is round, his complexion plethoric. His neck is thick and short. His chest is deep and round. His blood pressure is often elevated. Such patients have good appetites. They are commonly constipated.

Trauma plays an important role in the location of the joints involved by hypertrophic arthritis. Typists and pianists develop arthritic finger joints. Miners have arthritis of the spine. The obese patient has arthritis of the knees and lumbar spine. The Charcot joints of tabes are currently regarded as hypertrophic arthritis due to trauma.

In hypertrophic arthritis the arthropathy has a predilection for the large joints, often on one side. In the small joints hypertrophic arthritis is represented classically by Heberden's nodes in the terminal joints of women at the time of the climacteric.

The x-ray shows spurs and frequently narrowing of the joint space due to erosion of the cartilaginous plates.

Although the shafts of the patients' senile bones are often thin and brittle from osteoporosis the trabeculae about the joints are extra heavy. The cartilage is progressively eroded until finally the eburnated bone ends are approximated.

Bony overgrowth and muscular spasm due to pain may limit the motion of the osteoarthritic joints but true ankylosis, as seen in the atrophic type, never develops. Osteophytic growth and changes in alignment may result in very marked deformity. In this country arthritis deformans in atrophic arthritis. In England arthritis deformans is taken to mean hypertrophic or osteoarthritis. Because of the patients, their well-nourished state, their plethora and high blood pressure such patients have incorrectly been said to have gout. This incorrect diagnosis has erroneously raised the incidence of gout.

Joints afflicted with hypertrophic arthritis have to be protected against trauma. With this in mind the obese patients must reduce weight. Many of them are women in the menopause leading to the designation menopausal arthritis. These women irrespective of their basal metabolic readings frequently improve on thyroid. Estrogenic substances have failed consistently to benefit patients with arthritis. These substances, theelin and estradiol, have very effectively removed the arthralgic complaints of the climacteric. Only second to the metabolic approach is the effectiveness of physical therapy in hypertrophic arthritis. Reassurance that invalidism with deformity is unlikely is of great help in treating this type of arthritis.

A great group of patients present features common to the hypertrophic and to that sub-group of atrophic arthritis termed infectious. This motley, unclassified group actually embraces most patients with chronic arthritis. For sake of a better term their arthritis is said to be mixed although either the atrophic or hypertrophic characteristics predominate, marking the trend of the arthropathy. This mixed group requires a careful appraisal of the possibilities of the therapy used in the two great groups. Such patients have a better outlook for recovery than patients with classical atrophic or hypertrophic joint charges

(*Illinois Medical Journal*, March 1942.)

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Extracts.

NERVOUS COMPLICATIONS OF SULPHONAMIDE THERAPY.

LANCET, LONDON, 2: 162-163, Aug. 9, 1941.

Since the introduction of the sulfonamides, the nerve lesions which may complicate the acute infections seem to have been less rare, presumably because more patients with severe infections now survive long enough to develop them. However, these nerve lesions must be distinguished from the peripheral neuritis which may be produced by the drugs themselves. Clinically, the course of the two is different. In the septic type the lesion is either a radiculitis, especially in streptococcal infections, or is confined to the ulnar or peroneal nerve, which becomes acutely painful and tender, with paresis of the muscles it supplies. The sulfonamides, however, produce progressive paresthesias of the extremities with muscular weakness beginning in the small muscles of the hands and feet and gradually spreading centrally accompanied by sensory and reflex loss; in one or two extreme instances there has been complete loss of power in all four limbs. The sulfonamide neuritis was originally described as a rare toxic effect of sulfanilamide, usually after considerable overdosage, the amount given ranging from 50 to as much as 130 gm. With the development of the sulfanilamide derivatives, particularly paminobenzene-sulfondimethylamide, case-reports have been fairly frequent, and total amounts of only 12 to 30 gm. of this compound have caused the trouble. The etiology of the neuritis is not fully understood, but experiments in pigeons have shown that vitamin B1 will prevent paralysis, and in patients undergoing sulfonamide therapy there is a decreased excretion of vitamin B. Unfortunately treatment of the neuritis with vitamin B1 is only partly successful. All that can be done is to stop the drug and rest the affected parts with splints until the acute phase has subsided. When there are signs of returning activity, steadily increasing faradism with remedial exercise should be given. In severe cases, recovery is slow but the ultimate prognosis is still good.

The pathology of the lesions has not been studied adequately in the human subject, but much has been learned from animal experiment. Chickens are peculiarly liable to have lesions of the central nervous system during sulfonamide therapy, and in them Beiter and his colleagues of Minneapolis have demonstrated myelin condensation with axon swelling from this cause. Sulfanilamide was found to be the least harmful to the central nervous system of the drugs studied, the order of increasing toxicity being then sulfapyridine, sulfathiazole, and sulfamethylthiazole. In chickens it was possible to demonstrate selective variations in the concentration of different compounds in different parts of the central nervous system after

treatment, and similar variations are probable in man. The human sciatic nerve may certainly show concentrations appreciably higher than those found in the blood, and if this is representative of other peripheral nerves, the mechanism of neuritis becomes understandable. The importance of the selective distribution of sulfonamides between blood cells and plasma has been pointed out, and a study of the distribution in other organs seems worth attempting.

Some patients have had acute meningitis following intrathecal use of sodium sulfapyridine; and paralysis of the cauda equina, with slow recovery, has been reported after other soluble compounds administered in this way. Other patients have had injuries to the gluteal or sciatic nerve, particularly its external popliteal division, after careless intramuscular injections of sodium sulfapyridine, which may produce local necrosis. It is unwise to give more than 1 gm. in 33⅓ per cent. solution into the buttock at any one time. If more must be given, it should be given intravenously.

Mental disturbance more pronounced than that commonly arising during acute fevers may be observed during sulfonamide therapy and may be a sign of overdosage (Johnstone, D. F.; Forgacs, P.: Brit. M. J., 1: 772, 1941). An extreme example of this was in the patient who had acute mania after he had been given 4 gm. of sulfapyridine every 4 hours for 3 days by mistake. He recovered both mentally and physically on withdrawal of the drug. Less severe examples of confusion are sometimes seen after moderate dosages, indicating that the patients are sensitive to the drug. A peculiar form of delayed cerebral depression may follow any of the sulfonamides now in common use. This depression is characterized by inability to concentrate or add up simple sums, with loss of the fine coordinating power necessary for performing acts such as typing. The final prognosis of this is good. The amide of nicotinic acid may be effective in such cases.

FACTORS IN STABILITY & VARIABILITY OF SEMEN SPECIMENS.

BY ROBERT S. HOTHKISS.

From the Department of Surgery (Urology), the New York Hospital,
New York City.

The author reported on 642 specimens of semen collected from the 22 men and discusses the volume, cell count, motility and morphology of the specimens.

(1) *Volume of the Ejaculate.* The largest specimen received measured 7.8 cc. and the smallest 0.4 cc. The smallest sample

measuring 0.4 cc. was taken 3 hours after one measuring 4.2 cc. had been collected. The "average volume" of all the 642 specimens was 2.6 cc.

(2) *Cell Counts.* The largest number of spermatozoa per c.c. found in any specimen was 922,500,000 while the smallest number was 5,600,000. The man who produced the greatest number of sperm per cc. also had the greatest range in cell count. The average cell count per c.c. for the 46 specimens submitted by this man was 408,100,000. The man who had the lowest average cell count, which was 24,210,000 per c.c. also exhibited the minimum range of all the men. The total cell counts followed the trend of the counts per c.c.

(3) *Motility.* Three factors were considered evaluating the grade of motility: (1) The percentage of active cells (2) The type of motility and (3) the duration of activity. Each was given a grade ranging of 0—1—2—3—4 representing values from the poorest to the best. All of the 642 specimens had active spermatozoa and in no instance were the spermatozoa entirely inactive. The greatest range of motility was 3.5 grades. The average of motility for all the specimens was 2.5. The mean deviation for all specimens was only grade 0.48. The maximum mean deviation was 0.9 and the smallest mean deviation grade 0.25.

(4) *Morphology.* The "ovalcells" represent the "normal forms". The largest percentage of this type found in any specimen was 97.8 per cent. while the smallest number was 72.3 per cent. The least any man varied in the percentage of normal forms was 8 per cent. The maximum mean deviation was 3.2 per cent a minimum mean deviation was 1.4 per cent. The amorphous and duplicate cells were next in order in variability. Least common of all the abnormal cells were the "Gaint cells".

(5) *Viscosity.* The most viscid specimen received had a grade 4 motility and a cellcount of 74,000,000 per c.c. Mean deviation was 13.3 seconds for all the specimens.

II. The effect of frequency of coitus upon the semen was observed by comparative analysis of 276 specimens. The "repeat specimens" were all collected within 24 hours or less after a previous ejaculation. There was marked drop in volume and cell count in repeat specimen. The total count is reduced 50 per cent. The grade of motility and the morphologic types are relatively unaffected.

III. The effect of sexual rest on the semen. Specimens were collected after 72 hours' abstinence. The mean deviations were essentially unchanged.

IV. The effect of an illness upon the seminal specimens. The effect of an illness upon the seminal specimens was evidenced in the case of 1 man who had a streptococcic tonsillitis and was confined to bed for 6 days with a temperature that reached 102 F. Prior to

his illness his specimen had a volume of 7.0 c.c. fair motility, and a cell count of 119,500,000 per c.c. The number of normal spermatozoa was 77 per cent. Following his illness there was a progressive decline of his specimens and a low point was reached 30 days later. At that time all motility had disappeared, the cell count fell to 13,500,000 per c.c. and the number of normal forms was 69.5 per cent. Two months passed before the specimens had gradually regained their former values.

Effects of environmental factors. Artificial fever lowers the sperm count in man. A higher relative sterility is seen in the summer months. The frequency of coitus did not materially alter the morphology of the spermatozoa.

Conclusions.

There were wide variations in the volume, cell count and motility of the spermatozoa in 642 specimens of semen submitted by 22 men.

The morphological types exhibited relatively less fluctuation than did the other three characteristics of the semen.

The viscosity of seminal specimens varies considerably.

Frequent coitus tends to reduce the volume and cell count without materially altering the motility and morphology of the spermatozoa.

Illness may have an adverse effect on semen which is most marked about 30 days after the disturbance in health. Recovery takes place at a slightly slower rate. It is possible that season and temperature may have some effect on the semen.

(*Journal of Urology*).

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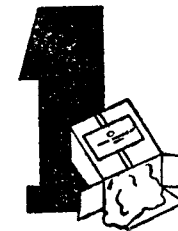
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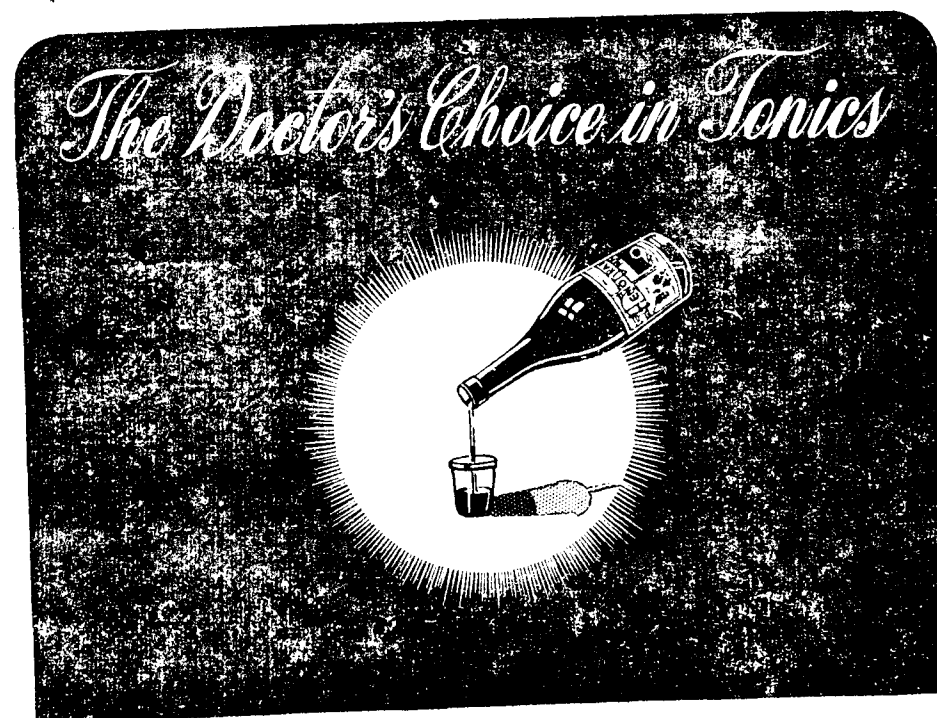
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THE SULPHONAMIDES IN SURGICAL PRACTICE.

DR. U. MOHAN RAO, M.S., F.R.C.S. (ENG.)

No detailed or exhaustive review of the various uses of Sulphonamides in Surgery is attempted in this short article. I shall use the word 'Sulphonamide' in the generic sense, embracing Sulphanilamide, Sulphapyridine (M. & B. 693), Sulphathiazole (M. & B. 760) and Albucid (Schering).

TREATMENT OF ACUTE INFECTIONS:

(i) *Streptococcus haemolyticus*.—Infections with this organism are perhaps the commonest acute infections that we have to deal with on the surgical side. Every surgical ward is bound to contain cases of cellulitis, lymphangitis, filarial funiculitis, filarial epididymo-orchitis, lymphadenitis, streptococcal tenosynovitis etc. In all these conditions the administration of Sulphonamides is indicated. The introduction of this group of drugs has reduced the mortality and morbidity from such infections in a very striking manner. The fall in the high temperature back to normal and the improvement in the general condition of the patient is very dramatic in the large majority of cases. It has been our practice, in Dr. N. Mangesh Rao's wards in the General Hospital in treating these conditions, to give three tablets of Sulphanilamide (1½ gms.) by mouth as soon as a diagnosis is made. 2 hours later two tablets (1 gm.) are given and then one tablet (½ gm.) is prescribed every 4 hours, *both by day and by night*. This medication is continued until the infection has been overcome or until signs of Sulphonamide poisoning appear or until a total of 40 tablets (20 gms.) has been given. The tendency to nausea that some patients have with the oral administration of Sulphonamides is largely mitigated by administering the tablets in milk or orange juice.

If the patient is vomiting (apart from the effects of an over dosage) then one of the soluble forms of Sulphonamide can be given by intra-muscular or intravenous injection. A 10 per cent solution of Soluseptasine, 5 cc. given every 6 hours as an intramuscular injection, has controlled the infection in the large majority of cases seen here.

(ii) *B. Coli* infections, like pyelitis or cystitis, respond dramatically to a similar dosage of Sulphonamides as mentioned above.

(iii) *Pneumococcus* and *Meningococcus*. In much the same category as the streptococcal infections mentioned above, are the infections caused by the above organisms. But these infections, apart from the rare pneumococcal peritonitis or arthritis, are so scarcely encountered in general surgery, that I do not propose considering them further here.

(iv) *Gonococcus*.—The introduction of the Sulphonamide group of drugs has completely revolutionised the treatment of gonorrhoea and certain other venereal diseases, especially *climatic bubo*. The correct period when the Sulphonamide therapy has to be started, the controlled dosage and the judicious local treatment require such specialist knowledge that I do not propose to dabble with them here.

(v) *B. welchii* and other gas-forming anaerobes. Experiments on animals persuade us that the Sulphonamides have a place in the prophylaxis and treatment of gas gangrene. It has been stated in foreign journals and our limited clinical experience in this hospital has amply justified the belief, that the value of antigas-gangrene serum is enhanced by the simultaneous use of Sulphonamides. In practice, those cases likely to develop gangrene seriously, that is those injuries resulting in extensive damage to muscle, would in any case be given Sulphonamides as a prophylactic against streptococcal infection, so that this problem is of practical importance only in an established case of infection with "gas-forming anaerobes".

I would like to bring in here the condition of peritonitis, either localised or generalised, secondary to some intra-abdominal catastrophe—e.g. acute gangrenous appendicitis, or a perforated duodenal ulcer. In these conditions the organisms responsible for the high toxicity may be one or all of streptococcus haemolyticus, *B. coli* & *B. welchii*—the very organisms on which the Sulphonamides may be said to have an almost specific action. Hence it is but rational to adopt Sulphonamide therapy in these cases, *after surgical treatment has dealt with the cause of the peritoneal infection*.

(vi) *Staphylococcus*.—Sulphonamide and Sulphapyridine are practically without effect in staphylococcal lesions, but there is some slender and as yet unsubstantiated evidence, that Sulphathiazole may be effective. Should this claim prove true, Sulphathiazole would be indicated in conditions like carbuncles, pyæmic abscesses and hæmatogenous osteomyelitis.

Should Sulphonamides be used in the presence of pus?—I cannot do better than quote Mr. Atkins, F.R.C.S. "In vitro experiments show that the activity of the Sulphonamides is seriously impaired if not abolished by the presence of pus and there is no reliable clinical evidence to the contrary. This is not to say, however, that the

Sulphonamides necessarily have no place in the treatment of an infective condition in which pus has formed. The localised abscess may represent a pocket of resistance where a "suicide squad" of leucocytes is fighting a heroic and successful engagement surrounded by an enemy which is elsewhere infiltrating and advancing. The Sulphonamides may do little to influence the fate of the Tobruk or Odessa, but might be the decisive factor in the major campaign. Such a conception, if substantiated would bring us back to our original stand-point that the Sulphonamides are of value in an acute infection. The presence or absence of an abscess in conjunction with this infection would not influence our treatment, nor would our treatment influence the abscess. *Thus a localised abscess is no indication for the use of Sulphonamides, but is an indication for surgery*".

Treatment of Chronic Infections.—Though the Sulphonamide group of drugs have been tried in cases of chronic *B. coli* pyelitis and cystitis with some success, the claims put forth are so conflicting and the results so varied, that it is still too early to make any dogmatic statement as to whether these drugs have any effect on chronic infective conditions met with in surgery. Infections with *Actinomyces* and *Maduromyces* have sometimes responded well to very heavy doses of Sulphapyridine.

Local applications.—It will be appreciated at once how important this is from the point of view of the surgical use of these drugs. If these drugs are active locally (as they have been proved to be clinically and experimentally) it means that a high concentration can be obtained just where it is wanted, with a minimal flooding of the circulation: so that cyanosis, vomiting, agranulocytosis and other evidences of toxic effects of the drug will be less common. Local concentrations from 75 to 100 times higher than systemic concentrations may be obtained directly at the source of the infection. Such high concentrations naturally have a higher bacteriostatic effect than the usual blood levels. In many ways these Sulphonamide compounds used locally are ideal tissue antiseptics in that they seem to have a low toxicity for the tissues, unlike other antiseptics.

A. MODES OF APPLICATION.

(i) *As a powder*, the Sulphonamide is sprinkled or dusted on the wound. It is distributed over the wound by means of a spoon, Volkmann's spoon, a pepper-pot or an insufflator. This is most used for shallow open wounds. Enough powder should be applied to cover the whole surface liberally.

(ii) *Tablets*.—It is difficult to transfer powder to the bottom of a deep sinus. Small bits of the tablet are easily inserted into the desired position by sinus forceps or Spencer Walls artery forceps and they are then crushed to hasten disintegration. On withdrawal of the forceps, the crushed tablet is left in position.

(iii) *As an ointment with vaseline*.

(iv) *As a paste with glycerine.*—This was used by Robson and Wallace in the treatment of burns of the face and hands, with very good results. The water-soluble Sulphonamide compound "Albucid" was used. The paste which they prepared contained in addition to Albucid and glycerine, kaolin and cod liver oil.

(v) *As a solution with saline,* either a saturated solution of Sulphonamide in saline (Mayo & Miller) or a mixture of 3 gms. of Sulphonamide in 100 cc. of saline.

B. INDICATIONS.

(i) *Compound fractures.*—The local use of Sulphonamide pack in the treatment of compound fractures has yielded very good results. It must be emphasised here that removal of foreign bodies, dead or grossly lacerated skin margins, muscles and other tissues must be done with as great and meticulous care as was done before the advent of Sulphonamide therapy. *Local Sulphonamides will never replace surgery and careful debridement and toilet of the wound cannot be dispensed with, but must precede the local use of Sulphonamides.* These drugs will assist the tissues to control such small foci of infection as have been left behind and will render the granulations clean and free from virulent pathogenic organisms.

5 to 15 gms. of powdered Sulphonamide should be applied locally. The wound is then either allowed to heal by granulation or dealt with either by primary or secondary suture, following the usual surgical principals. The fracture has to be reduced and immobilized as usual.

(ii) *Lacerated wounds* of the limbs either due to automobile accidents or due to gun-shot injuries not involving the skeletal system, have been treated successfully, as in (i).

(iii) *Burns.*—Those who have seen the unfortunate results of the indiscriminate application of tannic acid to burns of the face and hands will be convinced that the general use of coagulating agents should not be advocated; oedema and sepsis under a rigid tan, together with prolonged immobilization, have played their part in producing limitation of movement (in the fingers), contracture and in extreme cases, ischaemic necrosis. The burns caused by destructive agents of the present "total war" were so numerous, that the evolution of new methods of treatment became an urgent necessity. The local use of Sulphonamides either as a paste or with "tulle gras" forms only a fraction of the recent advances made in the treatment of burns. "Tulle gras" is a wide-mesh gauze impregnated with soft paraffin, balsam of Peru and other ingredients. Through the wide mesh secretions are easily absorbed by the outer dressings. The dressings can be easily removed without pain or bleeding.

(a) The glycerine-sulphonamide paste (made with Albucid) has given very good results in burns of the hand (2nd and 3rd degree),

of the face, scalp, penis and scrotum. The early movements that is permitted in the burnt parts, especially the fingers, is of very great value in the subsequent restoration to normal functional activity.

(b) Others have used Sulphonamide and "tulle gras", again for burns of the face and limbs. They claim that this treatment is almost painless and advocate its routine use in all superficial burns of moderate extent. The burnt area has got to be cleansed first and then it is thickly covered with Sulphonamide powder and "tulle gras" is stretched firmly over it in overlapping strips. A fresh application of powder is then made and several layers of gauze are super-imposed and lightly held in position with a bandage or face-mask. Only the dressings upto the "tulle gras" is to be changed at intervals of 24 or 48 hours or even longer, depending upon the discharge on the superficial layers of gauze.

Both the above methods have been used with success in infected burns also.

(iv) *Peritoneal infections.*—Sulphonamides have been used locally (in addition to their general administration) as a therapeutic agent in peritoneal infections. In cases where there has been extensive soiling of the peritoneal cavity from a perforated peptic ulcer or from a gangrenous appendicitis, their use has definitely reduced the mortality and the morbidity. The powdered, sterilized drug is sprinkled into the peritoneal cavity as near the focus of infection as possible, after this focus has been dealt with. This is considered by Mueller and Thompson to be a better method than that of depositing the drug in a mass in one place only. While some authorities strongly discourage the use of more than 5 gms. of Sulphonamide intra-peritonally, at one time, others have used this drug upto a maximum of 12 gms., with no harmful effects. In cases where the infection is less severe, doses less than 5 gms. have been recommended. In these patients with gross peritonitis, the application of Sulphonamide to the wound in the abdominal wall, greatly helps to diminish and control the infection in the abdominal incision also.

Local Sulphonamide is being used with good results in elective surgery of the gastro-intestinal tract, as a prophylactic agent. This routine use in abdominal surgery when actual or suspected peritoneal soiling has occurred, e.g. in partial gastrectomy, colectomy, ileocolostomy etc., has reduced the incidence of peritonitis and death. For such prophylactic use, doses of the drug between 2 and 8 gms. have been used. We have used Soluseptasine 10 per cent, 10 cc. locally with good results.

(v) *Gas-gangrene:* I have mentioned already that the Sulphonamide pack definitely diminishes the incidence of sepsis in a wound where the tissues have been grossly lacerated. In addition to this this pack acts as a prophylaxis against infection with anaerobic organisms.

In an established infection with anaerobic organisms also, these drugs have a local use, in addition to their general administration. But this therapy is useful only when it is used in addition to surgery.

(vi) Other conditions like empyema cavities, soft chancres and phagedenic ulcers have responded well to local Sulphonamide therapy.

Lastly, it is common knowledge that aspirin, sulphur containing compounds of the "salts" type and eggs (according to some) should be withheld during a course of Sulphonamide therapy, but it is not so widely realized that a patient undergoing such a course should not be exposed to direct sunlight and artificial sunlight should never be prescribed; otherwise severe skin reactions may follow.

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VITAMIN K IN HYPOPROTHROMBINEMIA

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Vitamin K deficiency is characterized by a depletion of plasma prothrombin which is intimately involved in the mechanism of blood clotting. A prothrombin deficiency may be the cause of excessive and dangerous bleeding in patients with hepatic or gastrointestinal disease.

The mechanism of action of vitamin K is not known except that it is utilized by the liver in the formation of prothrombin. Experimental observations indicate that liver damage is responsible for the reduction of plasma prothrombin and that the liver is the chief, if not the only, source of prothrombin. There is very little storage of vitamin K and very little reserve of prothrombin in the body.

Clinically, liver damage may occur in various diseases. Any obstruction to the extra biliary passages may secondarily cause liver damage. Primary liver disease (through infections, neoplasms, metabolic or other factors may affect the liver to such an extent as to result in a lowered plasma prothrombin level.

Natural vitamin K is a fat soluble substance whose absorption depends upon the presence of bile salts in the intestinal tract. A good general diet apparently contains enough vitamin K to maintain a normal plasma prothrombin level.

Two synthetic compounds with vitamin K activity have been used successfully in patients with a hypoprothrombinemia. An oil soluble compound 2-methyl-1, 4-naphthoquinone was used orally and a water soluble compound 4-amino-2 methyl-1-naphthol hydrochloride was used parenterally. The prothrombin determinations were done by the Smith method, and recorded in percentage of normal clotting activity. Bleeding levels could occur at any level below 70 per cent.

No correlation between the degree of jaundice or the duration of the jaundice and the plasma prothrombin level could be made of eighteen jaundiced patients with reduced plasma prothrombin. Five patients who had dangerously low levels did not have jaundice. Conversely, several severely jaundiced patients had normal prothrombin times. Thus one may assume that jaundice in itself is not a criterion for the determination of prothrombin levels. From these observation it appears that any patient suspected of liver damage with or without jaundice, and with or without bleeding, should have a prothrombin determination if one wishes to eliminate entirely the danger of haemorrhage.

Oral Administration. The initial oral dose used was 6 mg. of vitamin K and 2 Gm. of bile salts. As a routine, 5 grains of bile salts were given with every milligram of vitamin K. A daily dose of 1 to 3 mg. maintains a normal prothrombin level in most patients.

The following case illustrates the effect of the therapy:

Case M.R. presented a moderate degree of jaundice of four weeks duration due to a common duct stone. Preoperatively, the prothrombin activity was 61 per cent. This patient was given 1 mg. of vitamin K three times daily, with 0.33 Gm. of bile salts to each milligram. After one dose the level in twentyfour hours was 74 per cent. In forty-eight hours after three doses the level reached 91 per cent. Such a gradual rise in the plasma prothrombin was obtained with small doses. The level dropped to 75 per cent. in twenty-four hours. Then, an initial 6 mg. dose of vitamin K and 2 Gm. of bile salts were given. A rise to 95 per cent was then attained in one-half hour. Other patients responded similarly to these doses. There is no objection to the use of small doses of the compound when preparing a patient for surgery; however, if an immediate effect is sought, an initial large dose is recommended.

Vitamin K must be given with bile salts when the latter are not secreted into the intestinal tract. I found that in the absence of bile salts the response was negligible. This was illustrated by the case of A.K.; the patient gave a good response to an initial dose of 6 mg. of vitamin K with bile salts, rising from a 57 per cent. level

to 100 per cent. However, after the prothrombin time had dropped in twenty-four hours to 47 per cent. 6 mg. of the vitamin were given without bile salts, but there was no response. Then 1 mg. of the compound was given with 0.33 Gm. of bile salts, three times daily. After one dose, the level was 63 per cent. After four doses, the level was 100 per cent. Another patient, O.P., was treated for acute intestinal obstruction by Wangensteen gastric siphonage for seven days. A vitamin K deficiency was present due to starvation. It was assumed that bile salts were secreted into the intestinal tract. Three milligrams of vitamin K were given without bile salts. The duodenal tube was clamped off for two hours. The level rose from 79 to 91 per cent. in twenty-four hours. The next day it dropped to 70 per cent. Six milligrams of vitamin K, without bile salts, were given and in one-half hour the level was 90 per cent. In twenty-four hours it was again 90 per cent; and in forty-eight hours it was 100 per cent. Thus, one is guided by the etiologic factor of the vitamin K deficiency as to whether or not bile salts should be given in conjunction with vitamin K.

The response to the synthetic vitamin K is apparently immediate. When an adequate dose was given, a rise in the prothrombin level was obtained within one hour. Five patients were observed to determine the rapidity of action of the compound. In the case of A.K., one hour after an initial large dose, the level rose from 57 to 100 per cent. In the case of M.R., a 75 per cent. prothrombin activity increased to normal one-half hour after an initial 6 mg. dose. In three other cases there was a similar rise in one-half hour from 70 to 90 per cent, 66 to 80 per cent, and 74 to 90 per cent, respectively. Absorption of vitamin K is presumed to take place in the small intestine. The effect was so rapid that one might even suspect absorption from the stomach. Since the absorption was so fast, I gave the vitamin to several patients who were being treated by constant gastric siphonage. The vitamin K was given by mouth, the duodenal tube clamped off for an hour, and the prothrombin level determined. The response was good in all cases.

An attempt was made to determine the duration of the effect of a single dose of the vitamin. The rise in prothrombin activity following a single dose was maintained in some cases for only twelve hours; in others, for three days; and in some it remained normal. Clinically the patients with severe liver damage did not maintain the prothrombin level as well as did those with relatively good liver function. Undoubtedly there were exceptions to this. With such a variation in the maintenance of the plasma prothrombin one can be assured that the level is kept within normal limits only by repeated prothrombin determinations. Maintenance doses of 1 mg. of the vitamin, three times daily, are recommended if one is unable to perform the test very often. In the case of M.R. it was seen that

the preoperative level of 75 per cent. was increased to 100 per cent. by a single 6 mg. dose. There was no abnormal bleeding. Not until the eighth post-operative day did the level begin to drop. On the tenth day it had reached 55 per cent. At this point therapy, to which the patient responded immediately, was started. This patient maintained a normal level for eight days postoperatively after only a single initial large dose of the compound.

A normal prothrombin activity before surgery does not indicate that the prothrombin percentage may not drop to dangerously low levels post-operatively. The fourth to the seventh day after operation was found to be the time when levels were lowest. The postoperative prothrombin level in seven cases following biliary surgery is illustrated. Five of these patients were jaundiced; two were not jaundiced. In all of them there was a significant drop in prothrombin activity between the fourth and seventh day, an interval that might be called the "critical period." Patient P.V. had a moderate degree of jaundice of four weeks' duration, due to a common duct stone. This patient was given daily doses of 3 mg. of vitamin K with bile salts preoperatively. On the fourth postoperative day the level dropped to 63 per cent. Six milligrams of the vitamin, with bile salts, were given, and in one-half hour the level was 100 per cent. This level was then maintained on a 3 mg. daily dose until the patient was on an adequate diet.

No toxic effects and no nausea or vomiting were observed after the administration of the synthetic vitamin K. One patient was given as much as 50 mg. of the compound in a twenty-four-hour period without any ill effects. However, the prothrombin level could not be elevated above normal even with this relatively massive dose. From this observation I supposed that there was a stabilizing effect, most likely through the liver, which prevents an increase in the plasma prothrombin level above normal.

Parenteral Administration. Parenteral administration of vitamin K has several advantages over oral administration. By the oral route, there may be a lack of absorption due to intestinal obstruction, paralytic ileus or some other intestinal complication. By the parenteral route the vitamin may be given to patients who are unable to take it orally because of nausea or vomiting. In the treatment of hemorrhagic disease of the new born, for which vitamin K is very effective, the parenteral method is especially indicated. When the substance is administered parenterally it is not necessary to give bile salts.

The average initial dose was 1 to 3 mg. given intravenously and followed by 3 mg. daily as a maintenance dose. Some patients received 1 mg. daily and responded favorably, while others needed larger doses. The intramuscular dosage used was the same. However, this method of administration was not as satisfactory as the

intravenous route. The case of O.M., a patient with cholelithiasis and jaundice due to secondary hepatitis, illustrates the intravenous therapy followed. The level of plasma prothrombin before operation was 84 per cent. On the first day following cholecystectomy the level dropped to 62 per cent. Two milligrams of vitamin K was given intravenously, and in one-half hour the level reached 86 per cent. In two hours it was 100 per cent. Twenty-four hours later it had dropped to 71 per cent. At this time 1 mg. was given intravenously three times a day. The prothrombin level returned to normal and remained there with these regimens. H.M., a patient with jaundice due to a common stone, illustrated the more gradual response by the intramuscular route. The prothrombin level on the first day following a choledochostomy was 83 per cent; on the second day 70 per cent, and on the third, 64 per cent. Three milligrams of vitamin K was given intramuscularly. In one hour the level was 75 per cent; in two hours, 76 per cent, and in three hours, 80 per cent. The percent then dropped to 74 in four hours. Three milligrams of vitamin K was given intramuscularly with 1 Gm. of bile salts, by mouth. In one hour the level increased to 78 per cent; in two hours, to 84 per cent, and in three hours, to 86 per cent. The level again dropped in twenty-four hours to 67 per cent. Vitamin K (2-methyl-1, 4-naphthoquinone), 2.4 mg. with 1 Gm. of bile salts, was given by mouth. In twenty-four hours the level 68 per cent; in forty-eight hours, 90 per cent, and in seventy-two hours, 96 per cent. This level was maintained. However, the patient was improving generally, and the two factors were probably responsible for the continued oral improvement.

The response to the intravenously given vitamin K-active compound occurred within three-quarters of an hour to one and one-half hours. Patients with relatively good hepatic function showed normal levels of plasma prothrombin within two hours after the administration of the active compound. However, patients with damaged livers often did not reach a normal level of plasma prothrombin until twelve hours had elapsed; others never reached normal levels, in spite of the prolonged therapy.

SUMMARY

The dosage of the oral and parenteral synthetic compounds recommended varies from 1-3 mg. daily.

The dose of the vitamin K-active compound needed to maintain the plasma prothrombin varies with the individual patient. Maintenance doses can be estimated only by repeated prothrombin determinations. One milligram three times a day or 3 mg. in one dose are satisfactory amounts for maintenance.

Bile salts are necessary with the vitamin K compound for the patients who do not have bile salts secreted into the intestinal tract.

Bile salts need not be given when parenteral vitamin K therapy is used. However, their value in patients who are not secreting the bile salts into the intestinal tract must be recognized. Their deficiency may produce adverse effects in the body economy not related to prothrombin.

The effect of the parenteral water soluble compound vitamin K is no greater than that noted after the use of the oil-insoluble vitamin K compound, but vitamin K has the distinct advantage that it can be given at any time in the pre operative or postoperative course, regardless of whether the patient is able to take or absorb drugs by mouth.

A pronounced response to the synthetic vitamin K compound almost invariably occurs within one-half to one hour.

Patients with relatively good hepatic function showed normal plasma prothrombin levels within two hours, while patients with damaged livers reached normal levels in twelve hours or did not reach normal levels in spite of prolonged vitamin K therapy.

Clinically, patients with severe damage of the liver did not give as good a response to the vitamin K-active compound or maintain the level of plasma prothrombin as well as patients with relatively good function of the liver.

No nausea or vomiting and no toxic effects were observed following large doses of the synthetic compounds.

In biliary tract surgery the fourth to the seventh postoperative day constituted the period when the prothrombin activity most frequently reached dangerously low levels.

Hypoprothrombinemia may occur with or without jaundice and with or without bleeding; no definite relationship between the degree or the duration of the icterus and the decreased prothrombin levels can be made. However, it is true that prothrombin levels approaching the bleeding levels will be encountered more frequently in jaundiced patients, particularly if considerable liver damage is present.

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CONVULSIONS IN INFANCY AND CHILDHOOD.

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The proper treatment of convulsions is to find out the cause and remove it, but sometimes immediate treatment may be required. The child is to be sponged or immersed in a tub, of tepid or cool water. Hot baths are to be avoided since further elevation of an

already high fever may be dangerous. A lukewarm saline enema may be given. Question of sedatives must be considered. Intramuscular phenobarbital (gr. $\frac{1}{4}$ — 1), chloral hydrate (grs. 3 — 10) by rectum, or small doses of morphine (gr. $\frac{1}{100}$ — $\frac{1}{12}$), often suffice. If these have no effect within 10 — 15 minutes ether or chloroform should be administered. Seizure should not be allowed to last too long as far as possible. Cerebral damage may be done by the convulsion itself. Phenobarbital (gr. $\frac{1}{4}$ — $\frac{1}{2}$) by mouth should be given when the child is able to swallow, and may be repeated at four or six hours intervals for twenty-four hours to forestall recurrence.

Status epilepticus requires more heroic measures. Intravenous sodium luminal (1 — 3 grs.) or sodium amytal (1 — 5 grs.) may be slowly injected with the needle in place until relaxation occurs. Tremendous doses may be necessary and are comparatively safe. Lumbar puncture is at times effective. One per cent methylene blue intravenously (10 — 20 cc.) has been used with success; it may be repeated every four to six hours.

CONVULSIONS OF THE NEWBORN PERIOD.

1. *Intracranial hemorrhage of the newborn.* Intracranial hemorrhage is the most frequent cause of still births and death within the first two weeks of life.

Etiology. It is very common in prematurity. Other causes are difficulty and prolongation of labor, disproportion between the size of the fetal head and the birth canal, the use of instruments, manual extraction and excessive rapidity of the second stage and hemorrhagic disease of the newborn.

Symptoms and signs. One of the cardinal signs of this disease is convulsions and these within the first two weeks of life are strongly indicative of intracranial hemorrhage. Supratentorial hemorrhage may not cause convulsions. It causes cyanosis, nervousness, hypertonicity, shrill cry, bulging fontanelle, vomiting, pupillary and cranial nerve palsies and twitching movements as well as convulsions. Infratentorial hemorrhage usually gives rise to cyanosis, intermittent periods of apnea with deepening of the cyanosis, and slowing of the heart rate and flaccidity. Convulsions rarely occur in this form.

Prophylaxis. All the various causative factors must be guarded against. Vitamin K should be given to all premature infants and even to all newborn infants.

Treatment. The prime therapeutic agent is rest. The infant must not be allowed to attempt to nurse at the breast, feedings should be given from the bottle, syringe or medicine dropper.

Sedatives. Phenobarbital gr. $\frac{1}{8}$ by mouth every three or four hours may be sufficient. Chloral hydrate grs. 2 — 3 every four hours may be given by rectum. Morphia in doses of $\frac{1}{60}$ — $\frac{1}{100}$ grs. may also be given.

Lumbar puncture may be done in suitable cases.

2. *Tetany of the newborn.* Tetany as a cause of convulsions in the first few weeks of life ranks very low.

Etiology. Rickets in infants or defective intake of vitamin D or calcium by the mother are the common causes. There may be depressed function of the parathyroids.

Symptoms and signs. Symptoms usually manifest from the age of one week onward. Nervousness, twitchings and frequent short convulsions are usual. The only reliable sign is lowering of the serum calcium to 8 mg. per 100 cc. or less.

Prophylaxis. The gestating mother should take adequate vitamin D and calcium.

Treatment. Sedatives should be given as usual to control the convulsions and afterwards calcium and Vitamin D should be given. Calcium gluconate may be required to be given in doses of 300,000 units in two successive doses.

3. *Hypoglycemia of the newborn.* This is seen in offspring of diabetic mothers.

Etiology. This is due to hypertrophy of the pancreas of the fetus attempting to overproduce insulin in an effort to burn the excess of sugar transmitted to it from the maternal blood.

Symptoms. Many of the babies are oversized and overweight. Cyanosis and vomiting, and difficult or rapid shallow respirations are most common, muscular twitchings and convulsions may be present.

The blood sugar in the term infant is below 70 mg. per 100 cc.

Treatment. Oxygen is given for cyanosis and the child kept in a warmed crib. Glucose may be given by mouth, per rectum or intramuscularly in 5 per cent — 10 per cent. solutions.

Other causes of convulsions in the newborn are tetanus neonatorum, icterus gravis neonatorum, infections as sepsis, pneumonia and pyelitis, congenital heart disease and congenital brain defect.

CONVULSIONS AFTER THE NEWBORN PERIOD

1. *Febrile convulsions.*

Particularly between the ages of six months to three years convulsions may occur at the onset of any kind of acute infection such as meningitis, pneumonia or pyelonephritis, tonsillitis, or any acute exanthemata.

These children usually have but one, sometimes several attacks of convulsions at the onset of successive infections. Some continue to have convulsions later on for no good reason.

Treatment. The seizures are usually short and not severe. Cool sponge or cool bath is useful as is a cool enema. A mild sedative may be required.

2. *Intracranial infections,* such as meningococcal meningitis, pneumococcal meningitis, B. Influenzae meningitis, streptococcal

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meningitis or tuberculous meningitis may give rise to convulsions. Primary virus infections of the meninges such as poliomyelitis, encephalitides and lymphocytic, choriomeningitis may also give rise to convulsions.

The treatment is no general lines, sedatives, lumbar puncture and specific measures.

3. *Encephalitis* may occur during the course or shortly following an attack of measles, influenza, mumps, vaccinia or varicella. No treatment is effective for such cases.

Convulsions may occur during pertussis and the treatment is entirely symptomatic.

4. *Intracranial vascular disease.* (a) Subdural hematoma. This is consequent upon trauma to the head, but symptoms may not appear until weeks or months after the injury.

(b) Pachymeningitis hemorrhagica. This is mainly due to scurvy or congenital syphilis.

Signs and symptoms. The second form is commonest between the ages of six months and two years. Chief symptoms are failure to eat properly and gain weight, vomiting, periodic convulsions, enlargement of the head and failure to develop mentally. The fontanelle becomes full and tight, the veins of the scalp distended and hemorrhages appear in the fundus.

In older children the disease is usually traumatic in origin and the signs are those of a space consuming irritating mass over the cerebrum.

Treatment. This is directed to removal of the cause. Traumatic cases may require operation.

(c) Subarachnoid haemorrhage. In children it is due to rupture of an intracranial aneurysm, situated in or near the circle of Willis.

Signs and symptoms. There is sudden apoplectic attack, preceded usually by severe headache or convulsions, and followed by pain in the back of the head and neck and stiffness of the back and neck. There may have been a period of supra-orbital pain, with various cranial nerve palsies, chiefly affecting the eye. The spinal fluid is bloody.

Treatment. Absolute rest is essential for at least a period of six months.

Operation may be done if the aneurysm can be located.

(b) Cerebral sinus thrombosis. This may be primary and invariably involves the superior longitudinal sinus. It is caused by increased viscosity of the blood and slowing of the rate of blood flow. The secondary form includes the thrombosis resulting from the spread of infections from neighbouring structures (mastoids, meninges, sinuses, scalp) usually into the lateral or cavernous sinuses, and thrombosis occurring in the course of septicemia. This type seldom gives rise to convulsions.

Signs and symptoms. It occurs shortly after the onset - several days to several weeks—of a disease in which severe dehydration is prominent. Usually this is an acute diarrheal disease. There may be no symptoms whatsoever. If the thrombosis is extensive convulsions mark the onset and generalised rigidity or haemiplegia may develop. The retinal vessels become engorged and haemorrhage may be seen; the scalp veins may become distended and thrombosed. Death is the usual outcome; recovery may be followed by mental retardation epilepsy and spastic paralysis.

Treatment. Preventive. Severe grades of dehydration in illnesses in which vomiting and diarrhea are prominent and in severe burns must not be allowed to develop.

Curative treatment is directed towards the specific disease as well as surgical treatment.

4. *Strumpell Marie encephalitis*. There is thrombosis of the middle cerebral artery or venous sinuses.

Signs and symptoms. There is sudden onset of convulsions with high or rapidly rising fever. The convulsions are long, severe, usually generalised and often repeated. Following their cessation fever rapidly subsides and complete hemiplegia is noted. Epilepsy and mental deterioration are common sequelae.

Treatment. No treatment is of avail.

5. *The tetanies*. Most commonly tetany develops in association with rickets. This is known as infantile tetany and is one of the most frequent causes of convulsions between the ages of six months and two years.

Tetany may be due to temporary hypoparathyroidism or absence of these glands.

Prolonged steatorrhea such as occurs in celiac disease and in pancreatic insufficiency may lead to hypocalcemia and tetany.

Hyperventilation may also cause tetany. This occurs as a symptom in the postencephalitis syndrome, also rarely in hysteria.

Gastric tetany may develop from prolonged vomiting from any cause.

Signs and symptoms. Symptoms are characteristic. There are convulsions, carpopedal spasm, and laryngospasm. Convulsions are usually generalised and repeated. They are not followed by the stuporous period so usual after those from other causes. Nervous irritability can be elicited by Chvostek's sign, Trousseau's sign, peroneal sign and Erb's sign.

Treatment. Treatment is like that of the neonatal form. Calcium and vitamin D are required. In hypoparathyroid function, parathormone or dihydrotachysterol may be given.

The cause must be removed in hyperventilation form. Sodium chloride may be given in gastric tetany by subcutaneous or intravenous route.

6. *Hypoglycemia*.

The commonest cause is an overdose of insulin. Certain liver damaging poisons like chloroform, phosphorous and hydrazine also produce it. Von Gierke's disease or hyperinsulinism because of maternal diabetes or due to pancreatic adenoma may also cause it. There are some cases of spontaneous hypoglycemia without any explanation.

Signs and symptoms. Symptoms of an overdose of insulin are drowsiness, dizziness and ataxia. There is shock with rapid weak pulse, profuse perspiration, soft eye-balls, occasionally convulsions and coma may follow.

Treatment. Glucose is the specific remedy for hypoglycemia. It may be given by mouth, injection or enema.

7. *Cerebral poisonings*. Lead Encephalopathy. Lead poisoning causes convulsions in infants and children. These are severe, protracted and prone to repetition. Associated are vomiting often projectile, mental change, delirium, stupor or coma. There may be abdominal pain and constipation. Examination usually reveals pallor, elevation of blood pressure and choked disks. X-ray shows the lead line and epiphysis.

Treatment. Remove the cause. Phosphorus in the form of disodium phosphate may be useful.

8. *Tetanus*. The symptoms may be local as stiffness with or without pain. General symptoms as rigidity of jaw, risus sardonicus and boardlike consistency of the abdominal wall may be present.

Treatment. Prophylaxis. Active immunization by means of tetanus toxoid may be done. Specific treatment with anti-toxin should be adopted. Debridement should be performed and sedatives given.

9. *Cerebral edema*

It may be caused by lead poisoning or acute nephritis.

Treatment, is instituted against the cause.

10. *Congenital cerebral defects*. Diseases like microcephaly, hydrocephalus, tuberous sclerosis and spastic diplegia may cause convulsions.

11. *Epilepsy*. It may be congenital or acquired. A great variety of lesions have been found in the exposed cortices, neoplasms, scars, focal microgyria, cysts, congenital malformations etc.

Signs and symptoms. Recurrent major convulsions, minor motor attacks, or episodes of petit mal constitute the clinical picture. There may be associated mental or motor defect.

Treatment. Bromides and phenobarbital may be given. Dilantin sodium has given somewhat better results.

Failing favourable results from drugs, ketogenic diet should be tried.

Surgical treatment should be considered if the irritative focus can be localized.

Differential Diagnosis.

Periodically recurrent convulsions suggest idiopathic epilepsy, rarely hypoglycemia. Associated mental retardation with recurrence suggests microcephaly, cerebral agenesis, spastic diplegia, rarely tuberous sclerosis.

Failure to lose consciousness during the attack indicates tetanus or strychnine poisoning. Rapid return of mental alertness is suggestive of tetany.

Most attacks are generalized. When unilateral, vascular thrombosis, tumour or polio-encephalitis should be suspected but tetany and lead can be responsible. When focal, subdural haemorrhage, tumour or abscess is not unlikely.

Absence of fever will rule out infections. Its presence may indicate intracranial or meningeal infection or a non-specific one. It may also be present in tetany and in subarachnoid haemorrhage.

Choked disks is seen in tumour, lead poisoning and the cerebral edema.

Hypertension is present in intracranial tumours, lead poisoning and acute nephritis.

Normal spinal fluid rules out meningeal infections, the encephalitis, and lead encephalopathy. It is seen in subdural hematoma, polio-encephalitis and tetanus.

Clear spinal fluid with a moderate increase in cells and great increase in globulin strongly suggest lead encephalopathy. Clear fluid with increased cells and slight globulin increase suggests the meningo encephalitis group. Proportionate cellular and globulin increase is usual in tuberculous meningitis.

Turbid fluid means purulent meningitis.

Blood spinal fluid suggests intracranial hemorrhage of the newborn, ruptured intracranial aneurysm, sinus thrombosis or rupture of a subdural hematoma.

(*Medical Clinics of North America.*)

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Extracts.

SULPHANILYLGUANIDINE IN CHOLERA

BY SIR R. N. CHOPRA, C.I.E., M.A., M.D., SC. D. (Cantab), F.R.C.P. (Lond.) Col. I.M.S. (Retd.), A. J. H. DEMONTE, Dp. Bact. (Manchester), I.M.D., S. K. GUPTA, M.B., D.T.M. AND B. C. CHATTERJI, M.B., D.P.H., D.T.M.

(*From the Calcutta School of Tropical Medicine.*)

In the present study, an attempt has been made to evaluate the effect of sulphanilylguanidine in the treatment of cholera.

Sulphanilylguanidine for the sake of brevity called sulphaguanidine is a sulfonamide derivative and is distinguished from allied compounds by reason of its solubility in water and low absorbability from the gastro-intestinal tract. This causes it to remain in the intestines and to exert strong antibacterial effects. It is, therefore, likely to be beneficial in enteric infections such as acute bacillary dysentery, enteric fever, cholera and similar conditions.

Toxicity and medicinal use. Marshall and Litchfield (1939) found that sulphaguanidine was less toxic than sulphapyridine in dogs. The penetration and distribution of sulphaguanidine in the tissues appear to be very similar to that of sulphanilamide and sulphapyridine.

Clinically sulphaguanidine has been found efficacious in bacillary dysentery.

Clinical value in cholera. On the strength of the above properties sulphaguanidine was tried in the cholera ward of the Campbell Hospital.

The results in cholera treated with small doses of sulphaguanidine combined with saline, showed very little superiority over cases treated with intravenous saline. Cases treated with bigger doses of sulphaguanidine combined with saline therapy gave definitely better results, and the case mortality was reduced to 3.21 per cent. It is also interesting to note that of the three series of cases, studied, the series treated with larger doses of sulphaguanidine required 3.32 pints of saline per patient whereas the average requirements of saline for patients treated with smaller doses of sulphaguanidine or with saline alone were 4.8 and 4.27 pints respectively.

The effects of the larger doses of sulphaguanidine on the frequency of stools were also investigated. It was noted that cases having both sulphaguanidine and saline had 5.24 and 3.73 stools per head on an average on the first and second days of treatment against 7.81 and 4.09 stools per head for similar periods of treatment with saline alone.

The effect of the larger doses of sulphaguanidine and saline in the anuric stage of cholera demonstrated that out of 117 cases 39 passed urine within 24 hours, 12 passed urine within 48 hours, 4 passed urine within 72 hours, and 2 patients died after 48 hours.

Among 94 control cases (treated with saline alone), 81 were admitted with suppression of urine, out of which 12 passed no urine within 24 hours and the rest passed urine within 24 hours. Out of these 12 patients 6 passed urine within 48 hours and 2 passed urine within 72 hours, 4 died 72 hours after admission. It is thus obvious that larger doses of sulphaguanidine are definitely more effective than smaller doses.

It is realized that the doses of sulphaguanidine tried in both the series of cases were small compared to those that have been recommended subsequently for this drug. This was due to the fact that when the drug was received early in 1940, it was still in the experimental stage and proper dosage had not been worked out. Besides in an acute infection such as cholera, we thought it would be advisable to try small doses to begin with in the interest of the patients.

Much larger doses of the drug have lately been recommended in the treatment of bacillary dysentery. The drug is administered according to a dosage of 0.1 gm. per kg. of body weight for the initial dose followed by 0.5 gm. per kg. every four hours as maintenance doses. When the stools become fewer than 5 in 24 hours, 0.05 gm. per kg. is given every 8 hours. It is proposed to try this dosage during the next cholera epidemic.

Summary and conclusions. In a series of 218 cholera cases, sulphaguanidine 1 gm. initial dose, followed by 0.5 gm. six-hourly for 72 hours, has been found effective to reduce the mortality. The mortality in control saline-treated cases (94) was 6.38 per cent. whereas the mortality in sulphaguanidine treated cases was only 3.21 per cent. Sulphaguanidine is not toxic to human beings in these doses.

Sulphaguanidine treated cases passed fewer stools per day and required less intravenous saline.

The percentage of deaths among culturally-positive cases, treated with large doses of sulphanilylguanidine, was 3.84, whereas among control culturally-positive cases of cholera, treated with saline transfusions only, this was 8.97.

(*Indian Medical Gazette.*)

RECENT EXPERIENCES IN THE TREATMENT OF GONORRHOEA IN THE MALE

BY R. M. B. MACKENNA, M.A., M.D., F.R.C.P., Major R.A.M.C.:
Command Specialist in Dermatology, Northern Command.

About a year ago, when the author took charge of the division, cases of gonorrhoea were being treated on routine lines. Sulphapyridine was administered thrice daily, the average dosage being 3

grammes a day. Treatment was given for a week or ten days, during which time the men were kept in bed and on a light diet. The results were satisfactory, but not outstandingly so. The author introduced an "eight day course," of intensive treatment. The men were strictly confined to bed, received only milk diet, and were encouraged to drink as much barley water as they could. The course of treatment was as follows.

On the first day they received 6 grammes of sulphapyridine. This dose was repeated on the second day; on the third and fourth days they were given 4 grammes daily and, on the remaining days 3 grammes. The tablets were given thrice daily, and the course consisted of 32 grammes of sulphapyridine in eight days.

On this course, which was given to several hundred cases, there were no severe complications, but there was too high a proportion of generalised scarlatiniform eruptions associated with a brisk rise of temperature. For the majority of the patients, the chief disadvantage of the treatment was that when on the ninth day they had to get up they were weak from the combined effects of the action of the drug and the low diet.

Requirements in sulphapyridine treatment. The following desiderata are required in sulphapyridine treatment:

1. That the first dose should be the maximum which the average patient would tolerate.
2. The administration of the drug should be maintained throughout the whole of the twenty-four hours, so as to keep the concentration in the blood as high as possible.
3. That the patients should be kept on milk diet during the course of treatment.
4. The patients should be encouraged to drink as much of barley water as possible.
5. Care should be taken to avoid constipation which is an important predisposing factor in the development of the complications in sulphapyridine therapy.

Efforts were made to discover the optimum initial dose of the drug and to curtail the length of the course. The course consists in the administration of 22 grammes of sulphapyridine in forty-eight hours. The tablets are crushed and are given in milk every four hours during the day and night. Two important points should be noted namely, that the tablets are crushed, and that there is no interruption of the treatment during the night. The first dose is 5 grammes, the second 4 grammes, the third 3 grammes, the fourth 2 grammes, the remaining eight doses each of 1 gramme. He is given a milk diet, and mist. potassi citrate, and barley water. On the third day the discharge has almost ceased, but there may be a slight mucopurulent urethritis. He is kept for two or three days on irrigations of potassium permanganate 1 in 8000 or oxycyanide of

mercury of the same strength. Treatment is given thrice daily and the "grand lavage" is used, the irrigating fluid being allowed to flow in to the bladder. On the sixth day a smear is taken, a two glass urine test is made and a prostatic smear is also taken. Usually these tests are satisfactory. The patient is given two bottles of stout with his evening meal and the "morning smear" and the urine examinations are repeated on the seventh day and if these are satisfactory a provocative dose of the gonococcal vaccine containing three hundred million gonococci is given. On the eighth day the smears and urine examination are carried out. If these tests are satisfactory the anterior urethra is examined through a urethroscope. Only a diffuse reddening of the urethra is seen but no littiritis is present. A curved sound of suitable size is passed and the patient is kept under observation for further period of two days.

The patient returns to duty but three weeks later he is examined for signs of urethral discharge; the two glass urine test is made, a prostatic smear is taken and usually a curved sound is passed.

The same investigation is repeated during the following two weeks. Three months later another provocative injection of gonococcal vaccine is given, and all the usual tests—macroscopic, microscopical and urethroscopic—are carried out. Only if these are negative is he discharged as cured. Under this regime relapse rate was found to be only 5 per cent.

Complications of sulphapyridine therapy. Headache, lassitude nausea and vomiting are the only complications to be anticipated.

Pain in the lumbar region with haematuria is an important complication to be remembered. There is diminution of urine and retention of nitrogen. This is due to crystals of acetyl-sulphapyridine causing pyelonephritis. The drug is stopped, the pain is relieved by morphia and large amount of barley water and potassium citras are taken.

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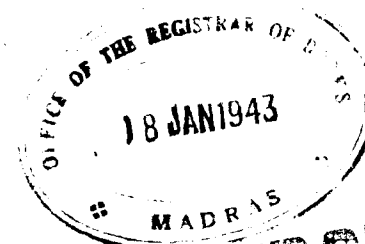
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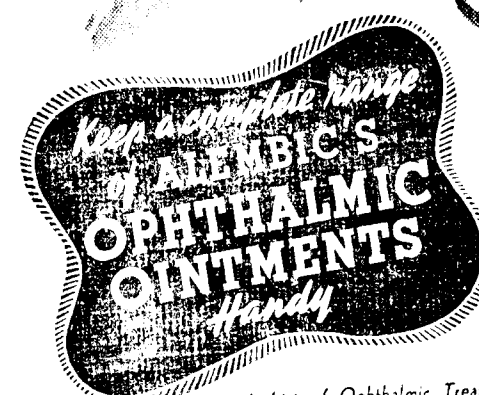
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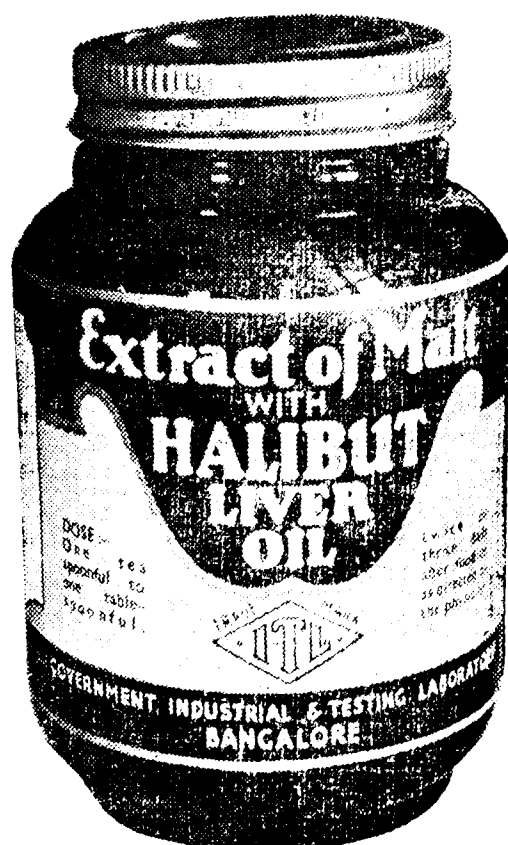
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SOME PROBLEMS OF BILIARY TRACT SURGERY

I. S. RAVDIN, M.D.

Harrison Professor of Surgery, Medical School University of Pennsylvania; Director, Harrison Department of Surgical Research, Medical School, University of Pennsylvania; Surgeon Hospital of the University of Pennsylvania.

The morbidity and mortality of many operative procedures have been reduced, as we have come to understand more clearly the deviations from normal physiological function which disease of certain organs brings about, and as we have also come to understand the processes by which normal, or more nearly normal, function may be re-established prior to anesthesia and operation.

This has been true in operations for hyperthyroidism, intestinal obstruction and prostatic hypertrophy where greater emphasis on pre-operative and post-operative care has led to a substantial reduction in mortality during the past decade or two. It is equally true of many surgical lesions of the biliary tract, for the past decade has provided a much clearer picture of normal physiological function of this system and a more comprehensive picture of the abnormalities of function which may occur as the result of disease. I should like to discuss with you to day a few of the problems presented by patients with gallstone disease, to present our methods of meeting them and then to leave it to you to determine whether greater attention to these factors may not provide a means of further lowering the morbidity and mortality of operations for the relief of gallstone disease and its complications.

One of the most serious problems in this field is the frequent inability to determine the extent of hepatic injury. The liver function tests have enjoyed a variable degree of approval by different investigators and by the same investigator at different times. The surgeon is interested in whether or not the liver is so seriously impaired that operation is attended by grave hazard. I doubt whether any one of the commonly used tests can, as a rule, give us more information than can be obtained from a good history and careful physical examination. The galactose tolerance test, hippuric acid conjugation, prothrombin determination, bromsulphalein

retention, the ratio of free and combined cholesterol and other tests now commonly used each measure but a single function, and a marked deviation from the normal in one test does not necessarily mean that the others will be equally impaired. In fact, in our experience the reverse is often true. Some general idea of the degree of impairment of function may be had from the results obtained from a number of different tests. However, we have not always been able to find close correlation between the histologic picture and the results of function tests, and on more than one occasion we have seen a liver which gave every indication of normal function prior to anesthesia and operation become incompetent following operation. If morbidity and mortality are to be kept low, the surgeon must not be lulled into a sense of false security, and too often dependence upon function tests has tended to do this.

A considerable number of the patients with gallstone disease have diabetes mellitus. It is our impression that this relationship is increasing rather than decreasing. It is important to remember that the diabetes often is not diagnosed until the patient is admitted to a hospital, and it is equally important to remember that the liver stores of glycogen may be seriously reduced in these patients because of long standing hepatitis. Since the hepatic glycogen is the sole direct source of the blood sugar, a marked decrease in the glycogen stores of the liver may create a real hazard. A dose of insulin, which in the non-biliary tract diabetic patient would have but slight effect on the blood sugar concentration, may have a very profound effect in the patient whose liver cannot rapidly mobilize glucose from the diminished hepatic glycogen stores to replenish the falling blood sugar concentration brought about by the insulin action.

There is no need rapidly to control the hyperglycemia in these patients, especially if they are above fifty years of age. The precipitation of insulin shock in the elderly diabetic patient, not previously treated with insulin, may be irreversible even though large amounts of glucose are subsequently given. It is far better slowly to prepare such patients, unless diabetic acidosis is also present when of course the therapy must be more active. These patients, as a group, require the care of some one familiar with diabetes on the one hand and the pathologic physiology of hepatic dysfunction on the other.

It is now 33 years since Babcock, of Chicago, called attention to the frequent relationship of gallstone and cardiac disease. For many years it was believed that organisms which had a specific affinity for the gall bladder also attacked the myocardial muscle. Schwartz and Herman believed that the cardiac disturbance was the result of fatty infiltration of the heart and that this was merely an expression of the disturbance of metabolism from which many gall bladder patients suffered.

Fitz-Hugh and Wolferth more recently reported certain changes in the electrocardiogram which many of these patients presented. There is often a depression and occasional inversion of the T wave and at times a slurring of the QRS interval. Weiss and Hamilton have confirmed the T wave changes and have substantiated the conclusions of Fitz-Hugh and Wolferth that the electrocardiographic changes disappear very soon after convalescence from adequate biliary tract surgery. It is their opinion that the changes are the result of toxic disturbances in the cardiac mechanism. Every change in the electrocardiogram in these patients does not necessarily indicate serious and irreversible cardiac damage.

Many patients with gallstone disease also have pain which simulates angina pectoris. Very often the patient is denied relief from the symptoms of gallstone disease because an internist believes the risk of operation is too great. We have had the good fortune to see a number of these patients, and careful study of them often reveals the fact that the pain is rarely due to true angina pectoris. Royster and Sanders have demonstrated in one of them, for the first time I believe, that reflexes arising in the common duct may give rise to pain and other sequelae which are at times nearly indistinguishable from angina pectoris.

The pain is often typically substernal and precordial and may be referred to the left upper extremity. It is frequently preceded by a feeling of impending death, and is followed by hyperesthesia and hyperalgesia of the area to which pain was previously referred. It differs from true angina pectoris in that the attacks are not related to effort, for they usually occur at night while resting in bed. The attacks may have been present over a period of years, yet the electrocardiographic changes may be negligible. Even with these differences, on more than one occasion we have seen competent cardiologists who were loathe definitely to state that the patient did not have angina pectoris. One of the patients upon whom we operated this year had been studied by a cardiologist in this part of the country who wrote to her family physician: "God save Mrs. from the hands of the surgeon."

Patients with gallstone disease at times do have true angina pectoris and serious cardiac disease, just as do non-gallstone patients, but all precordial pain, or even changes in the electrocardiogram, do not necessarily indicate serious cardiac disease. When heart disease is present its symptoms may be accentuated by the co-existing gallstone disease, the latter frequently giving rise to recurrent distention, to constipation and to disturbed sleep by nocturnal attacks of pain and indigestion. Even patients with serious heart disease, as a rule, withstand operation well, if they are properly prepared before operation, and the risk involved in the other patients is no greater than if the pseudo-angina had not existed.

A decade ago the most serious complication of operation on a patient with common duct obstruction was post-operative hemorrhage. Until Armand Quick demonstrated a prothrombin deficiency in these patients the pathologic physiology of this hemorrhagic tendency was not known. In 1934 Dam, of Copenhagen, and Almquist, of this country, found that the hemorrhagic disease of the gizzards of young chicks was due to a deficiency in the diet of an accessory foodstuff which Dam named vitamin K.

Dam later showed that these chicks had a prothrombin deficiency and that the response to vitamin K therapy was a return to normal of the prothrombin concentration. It was soon determined that vitamin K was fat soluble and that the prothrombin deficiency which took place in man might well be conditioned by the absence of bile salt from the intestine resulting in the failure of absorption of vitamin K. Before Quick had an opportunity to test this hypothesis, Smith, Warner and Brinkhous found it to be true.

It is now possible to prepare the jaundiced patient with bile salts and substances containing naturally occurring vitamin K₁ or K₂, or to use one of a variety of synthetic substitutes before operation. Approximately 80 per cent. of the patients so prepared for several days prior to operation will respond favorably, but from 18 to 20 per cent. have such severe liver injury that even though large amounts of bile salts and the most active preparations of K are administered, the prothrombin concentration does not respond satisfactorily and often does not respond at all. When this occurs, fresh blood or plasma should be used to control the tendency to hemorrhage. If operation can be delayed, diet may be used for some days in an effort to improve hepatic function. We have done this on several occasions with favorable results.

One of the most serious complications of operation on the patient with longstanding gallstone disease, with or without involvement of the common duct, is the degeneration or necrosis which may follow the use of hepatotoxic anesthetics. Ether, contrary to general opinion, may cause serious hepatic degeneration and even necrosis of the liver. Nitrous oxide may, under certain conditions of anoxia, lead to such serious injury that the histologic picture is nearly indistinguishable from the necrosis induced by chloroform. Even ethylene has been shown to effect the hepatic function, and no one has satisfactorily demonstrated that cyclopropane is entirely harmless.

Many of the severe reactions which biliary tract patients at times have following operation are without doubt due to varying degrees of degeneration and necrosis conditioned by the anesthetic agent and the method of its administration. These reactions are often called minor degrees of "liver shock." We are convinced that spinal anesthesia with procaine, in which major depression of the blood pressure are prevented by the preliminary use of long acting

vasoconstrictor drugs, is at present the safest anesthetic for these patients. Even with this method of anesthesia the patient should be prepared so as to minimize hepatic injury. Rarely indeed is operation an emergency.

For years we have been led to believe that a high concentration of hepatic glycogen would protect the liver from injury by hepatotoxic anesthetics. At first we fed patients a high carbohydrate diet, and then as the result of debatable reasoning, we gave these patients glucose intravenously for several days prior to operation. Many of us then sat back smugly confident that we had done our best. Some of us were even guilty of stating, as Banks and Sears have recently done, that hepatic regeneration would take place more rapidly following injury upon a carbohydrate diet than upon any other. It is high time that we critically review the evidence upon which these assumptions are based. I would not for a moment deny that the program we have been using has helped; but has it done the best that we are capable of? In the first place, 3000 c.c. of a 5 per cent. solution of glucose a day provides the patient with but 600 calories. If no food is given my mouth is it possible that glycogen can be maintained in the liver for any time? Can we materially affect the composition of the liver unless we supply the patient with a sufficient amount of food to meet energy requirements plus an additional amount for storage? The glucose so given will to a degree prevent the breakdown of body tissues to supply energy requirements, but that is all. The loss of weight of our patients during pre- and postoperative care is usually an expression of the extent to which we fail to meet their energy requirements. Of this there can be no doubt.

Evidence which we have obtained and which has since been confirmed and extended by several observers, points to the fact that protein and not carbohydrate directly protects the liver from injury by a variety of hepatotoxic agents. I can summarize our work by stating that a liver with a high lipid content and a low protein content is maximally susceptible to injury by these agents. This explains why the liver of undernutrition or starvation is so susceptible to damage. Conversely, the liver with a low content of fat and a large amount of available protein is maximally protected against injury. Even in the presence of considerable amounts of lipid, an adequate intake of a suitable protein for some time prior to anesthesia and operation will provide protection from such an injurious agent as chloroform.

We have known that the livers of patients with long standing biliary tract disease frequently contain large amounts of fat. One objective of pre-operative therapy must be to rid the liver of as much of this fat as possible. This can, in part, be achieved by the deposition of glycogen, for usually as glycogen is deposited in the liver fat

is displaced from it. This process is known as the fulfillment of the Rosenfeld hypothesis. Data which we have obtained demonstrate clearly that protein will do this much more rapidly than carbohydrate, for the addition of protein to the extent of 28 per cent of the total calories of an otherwise high carbohydrate diet will, in seven days, result in a decrease in the liver lipid concentration in the presence of ductal obstruction similar to that obtained on high carbohydrate-low protein diet in fourteen days. Protein, therefore, is useful from two points of view, its direct protective capacity, which is probably due to certain components of the protein molecule, and to its capacity to rid the liver of excess fat.

We are using in our patients, a diet in which the total calories consist essentially of 70-75 per cent of carbohydrate, 20-25 per cent of protein and not more than 5 per cent of fat. It is, we believe, the ideal pre-operative diet for these patients. It can be fed by tube if the patient will not eat. The carbohydrate in large part comes from the banana and the protein from Casein or cottage cheese or a protein digest such as Aminoids. The patients receive from 2500 to 4500 calories daily for from five to twenty-one days before operation. Biopsies from the liver of these patients operated on under spinal anesthesia demonstrate that we can by this method condition a liver to minimal injury.

If oral or tube feeding is impossible, then glucose and plasma can be given intravenously. The addition of plasma will provide a better hepatic composition than can be obtained from glucose alone, but even this combination will not provide as satisfactory a condition as can be obtained by oral or tube feeding. If sufficient food is not ingested by mouth, intravenous therapy should be used as an adjunct.

There are certain problems concerned with the operation which must be kept in mind. Injury to the ductal system can often be repaired but injury to the hepatic artery requiring ligation will inevitably lead to hyperthermia, a falling blood pressure, a rapid thready pulse, hypoglycemia and death. This is commonly known as "liver shock." While life is compatible with a very slow occlusion of the hepatic artery, its acute occlusion is invariably associated with death. The greatest care must, therefore, be exercised in dissections of the right free border of the gastro-hepatic omentum.

The decision of when to open the common duct is often difficult to make. It should always be opened if the patient is jaundiced at the time of operation, or if there is a history of previous jaundice. It should be opened if it is greatly dilated or if the cystic duct is short and large and the gallstones small. Finally, it should be opened if, after all considerations, any doubt as to the advisability of this additional procedure still persists. The time to open the duct is at the first operation.

When common duct drainage has been established, it is the height of folly to permit large amounts of bile to drain into a bottle at the side of the bed. No amount of glucose and salt given intravenously can compensate for the diversion of bile from the intestinal tract. Even though the bile may have a low bile salt content, it will help to bring intestinal function back to normal and the asthenic states so frequently observed during periods of excessive bile diversion rarely will be encountered if excessive bile loss is not permitted. Bile is not solely an excretory product; it plays an important part in the intestine in the activation of lipases, the emulsification and absorption of fats, and the absorption of the fat soluble vitamins such as vitamin K. We must constantly attempt to restore normal function rather than inhibit it.

Diet is just as, or even more important after operation as before it, for it is then that regeneration can most readily take place. We do not yet know what the ideal postoperative diet is, but it can be accepted without controversy that cellular regeneration cannot take place rapidly on a diet inadequate in protein. The major question at present is what protein will best facilitate repair. Data which workers in our laboratory are now obtaining strongly suggest that when liver repair is desired liver protein is the best substance to provide this.

Vitamin K therapy should be continued until wound healing is nearly complete. The fact that the prothrombin concentration was normal prior to operation should never be accepted as evidence that it will remain so after operation. It has been our experience that it usually falls, even when the physical trauma to the liver has been minimal. Occasionally a liver that responded well to vitamin K therapy prior to operation will fail just as completely to respond following operation. Such a liver has been seriously injured, and it may be necessary to resort to blood or plasma transfusions to prevent or control hemorrhage.

The common duct tube should never be removed until a cholangiogram has been made. Our complacency is disturbed from time to time by a graphic visualization of a stone we have failed to remove. It is best to discover this when it is still possible to attempt the solution of the stone through the tube which leads to it.

These are a few of the problems which we are daily meeting. Their control is often not difficult. There are, of course, many others such as those presented by acute infection and cystic duct obstruction, by stricture, acute cholangitis and malignancy which I have not had the time to review with you. Certain of the principles I have attempted to cover apply, however, equally well to these conditions.

Applying these principles in our own clinic has led to a very substantial reduction in mortality. From 1922 to 1929 the mortality

of operations for common duct obstruction was 22 per cent. from 1935 to 1938 11 per cent, and from 1938 to 1941 it was but 2.6 per cent. There have now been operated on, on my service, a consecutive group of 629 non-jaundiced patients subjected to cholecystectomy with or without common duct drainage with but a single death. This mortality is not due to great operative experience, for a very considerable number of these patients were operated on by my surgical fellows. It is, in the main, the result of having obtained a better picture of the pathologic physiology of biliary tract disease and of attempting to restore more nearly normal function prior to operation and to maintain and improve this subsequent to operation.

(*Illinois Medical Journal*, August 1941.)

ACUTE HEMATOGENOUS OSTEOMYELITIS

GUY W. LEADBETTER, M. D.

Associate in Surgery, George Washington University School of Medicine

Osteomyelitis is an acute infection involving the medulla of a bone primarily and the cortex and the periosteal tissue secondarily. This discussion is limited to acute hematogenous osteomyelitis, because osteomyelitis appearing as a complication of compound fractures or following operative procedure presents its own individual problem.

The bacteriologic etiology of this disease is well known and is limited nowadays largely to two groups of organisms, namely, staphylococci and streptococci. Many of the other bacteriologic causative agents have been greatly diminished in their incidence by the protective measures of preventive medicine. Trauma undoubtedly plays a large part in the production of the localization of the disease, but unquestionably in some instances focal infection which produces a bacteriemia or mild septicemia without trauma is equally important.

Characteristic are the sudden and acute onset with rapid, almost explosive overwhelming toxemia, unbearable localized pain, sometimes without external-localizing signs and symptoms, at other times with great swelling, redness, local heat, pulsating pain and excruciating tenderness to pressure. The differential diagnosis must be made from tuberculosis of the bone, acute hemorrhagic periostitis, purulent periostitis or acute cellulitis.

The anatomy of the bone presents the explanation for the origin of the localization of this disease in children. It appears more often

that not in the metaphysis in juxtaposition to the epiphyseal line. This site of onset is explained best by the fact that loops of arterial and venous vessels are to be found in the metaphyseal site of the epiphysis. Here in these expanded loops of vessels there is more opportunity for the formation of a bacterial nidus than in other parts of the shaft where circulation is more rapid. The spongy, soft, very vascular bone in this area presents an excellent medium for the rapid growth of bacteria and their consequent inroads on and the destruction of these tissues. Very rapidly the circulation of the infected area is much diminished. Early necrosis results. So quickly does the necrosis progress that it is rare indeed in the early, acute stages to find a definitely walled-off lesion. Hence, in the medulla of the bone the disease has a fertile field for rapid expansion and only after a defensive wall has formed and the internal pressure has increased greatly in the medullary cavity do the organisms and purulent material attempt to evacuate themselves by invading the cortex through the medium of the Haversian system.

When this invasion is completed, purulent material together with organisms appear beneath the periosteal layers and produce the elevation of the periosteum which is so commonly seen in the roentgenograms. Actual appearance of the elevated periosteum is relatively late in the course of the disease, and sometimes, although it may be the only sign of osteomyelitis, it must be clear that considerable destruction has already materialized. These facts explain why osteomyelitis is a dread disease.

It is clear that there are two phases of the disease which must form the objective of the spearhead of attack: (1) the septicemia, and (2) the localized necrosis of the bone. The first phase is represented by a bacteriemia or septicemia in all degrees of severity and more often than not this condition presents the factors of success or failure. It has been pointed out in many researches during the past two decades by Wadsworth, Hoppe Burnett, Parker, Pike and others that the various strains of staphylococci form exotoxins which produce destruction of leukocytes, hemolysis of the red cells, coagulation of the plasma, and necrosis of the tissues. It has also been shown by Burky and Dolman that the antitoxins produced by one strain of staphylococcus are capable of neutralizing the toxins of other strains. Robertson published a work in which he emphasized the value of determining the titers of the level of antitoxin in the patient's blood. In those cases of acute osteomyelitis with a favorable prognosis, the staphylococcal antitoxin appeared in tremendous amounts. In those cases with fatal determination, little or no antitoxin was found. The presence of mature and immature white cells, regardless of the leukocytic count, is an important index in determining prognosis and the value of administration of the staphylococcal antitoxin. More recently the appearance

By the same chain of reasoning the local recurrence of an osteomyelitis months or years after the original onset carries with it a negligible mortality since already the local and general defenses are in order and the recurrence is usually due to the momentary flaring up of a vitiated organism which has been harbored within bony structures for a considerable period of time. The presence of sequestra diagnosed by X-ray is the usual indication for operation.

It is not necessary here to discuss the particular technic of the operative treatment of acute osteomyelitis except to point out, as has been mentioned before, that acute and complete drainage of the entire necrotic area is essential for success; for example, the medullary cavity of any of the long bones can readily be drained by fenestration. In the particular instance of the os calcis, a somewhat definite problem presents itself, and adequate drainage is not obtainable by incision and drainage of one or the other side of this bone. The best results in osteomyelitis of the os calcis have been obtained by a splitting and bifurcation of the bone posteriorly, thereby allowing adequate drainage from all portions. This procedure is mentioned merely to emphasize the necessity of proper mechanical drainage.

It is clearly evident from the facts of comparatively recent research and from experience that hematogenous osteomyelitis is a serious disease, presenting not only the danger of loss of a functioning extremity but also a mortality rate which until recent months has been distressing. It is also clear that for the first time we have in our hands the instruments by which the disease can be successfully attacked. The use of antitoxin or sulfathiazole, or both, should be a part of the active and vigorous therapeutic measures necessary for success. Early operation should also be done with the establishment of adequate drainage of the localized infection. The type of early operative procedure here described is not shocking and does not injure the patient but gives considerable aid in allowing for the discharge of accumulated infected material and in most instances prevents increased destructive inroads of bone.

Finally, a word of warning is indicated, that we must not become too enthusiastic concerning the use of chemotherapy or antitoxin and utilize these agents as a sole procedure in treating osteomyelitis. More failure than success will result. The disease is twofold, a general septicemia and a localized necrosis. The adequate treatment of both conditions must be utilized if success is to crown our efforts.

(*Medical Annals of the District of Columbia, September 1941.*)



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Extracts.

TREATMENT OF FRESH COMPOUND FRACTURES AND WOUNDS

BY J. C. SCOTT, M.D., M.S., F.R.C.S.,

Accident Service, Radcliffe Infirmary, Oxford.

Wounds and compound fractures are cases of extreme surgical emergency. A few hours' delay in treatment may make it impossible to prevent infection in the wound with the resulting danger to life and limb and many months of delay in obtaining healing of the wound and union of fracture. The optimum time for treatment is usually accepted as being up to six hours.

People who first see these injuries are not sufficiently aware of the urgent necessity to arrange so that the patients receive adequate treatment as soon as possible. The only justifiable cause for delay is the patient's general condition, and, if this is such that transfer is not possible, then active measures to restore the general condition must be started at once.

The question of urgency of treatment is very intimately bound up with resuscitation. When any patient is admitted with a serious wound or compound fracture, resuscitation should be started at once without regard for his condition. The patient should be kept warm. The necessary sedative should be administered, and the limb should be splinted as it lies if this has not already been done. It must be stressed that gross compound fractures should not be pulled into line by the first person who sees them or immediately on admission. Early splintage is essential, but where there is contamination of a compound fracture no attempt should be made at reduction until the wound has been examined. When sedative and splintage are in order plasma drip or blood transfusion may be given for resuscitation.

Preparation of skin. This is to be prepared like preparation of the surgeons' hand. Soap and water are enough. The wound is covered with sterile gauze and the skin round about cleaned. The surface of the wound should also be washed with soap and water. Flushing of the wound with large quantities of saline is to be avoided.

Excision of wound. A tourniquet should never be maintained in place during this procedure. The excision should include the removal of all tissues which have been robbed of their blood supply. This rule should be applied to each layer in turn. Any surgeon who fails to do this will have cases of gas gangrene and spreading infection no matter how much he tries to lean on the aids to surgery—sulphanilamide, sera, rays etc. To follow this rule may mean in some cases very extensive incision of soft parts. It is essential that this should be ruthlessly carried out. The removal of bone should be conservative and only fragments that are completely detached from periosteum and muscle or are definitely contaminated should be removed. The fresh wound is the wound in which infection is not already established. To fail to excise a wound because it is more than six hours old is to deny the principle of excision.

Suturing. In civilian injuries, when the operation is carried out within six hours and the suturing can be done without any tension on the skin, it is advisable to suture the wound. Six hours is accepted maximum but the sooner the better. In wounds of more than six hours' duration, and those in which tension is necessary to bring the wound edges together, no sutures should be used. With the exception of ligatures and stitches to close a joint, no deep stitches should be used.

Applying the plaster. If the wound is not sutured and no infection is present dry gauze is used in the wound. Plaster is applied immediately over the gauze. If infection is already present vaselined gauze may be used. The limb should be immobilized, including one joint above and one below the site, of the wound or fracture. If padding is used under the plaster it must be highly absorbent.

The next question is whether or not windows should be cut over the wound. When a wound is left open no window should be cut as window may lead to infection. If the wound is stitched and remains uninfected there is no need to cut a window, but the window will do no harm.

Sera, Sulphonamides and X-rays. Antitetanic serum should be used as a prophylactic in all cases.

Anti gas gangrene serum is of no use as a prophylactic.

The prophylactic use of sulphonamides in post-operative treatment of fresh wounds is also to be deprecated, especially the routine use of sulphapyridine a common practice. If infection is present then the sulphonamides may be used.

The routine prophylactic use of X-ray treatment against gas gangrene is unnecessary. It is said that use of the excision-incision closed plaster technique makes the early diagnosis of gas gangrene difficult. Gas gangrene starts most commonly in three regions in the body—the calf, the thigh, and the shoulder. If gross muscle damage and gross contamination is present in these areas and the results of excision are doubtful the application of plaster may be postponed for 48 to 72 hours. If by that time no infection has developed, the plaster may be applied. If change in general condition of the patient is closely observed early gangrene can be diagnosed without inspection of the wound.

Anti gas gangrene serum is useful in treatment of gas gangrene but adequate doses must be given *i.e.* about 100,000 units in twenty-four hours in an adult. Soda bicarb, by mouth may be given to combat acidosis.

Local use of X-rays is said to be useful in the treatment of gas gangrene.

(B. M. J.)

PROGNOSIS OF ACUTE INTESTINAL OBSTRUCTION

BY R. V. HUDSON, R. SMITH AND F. R. SELBIE.

Hudson and his associates studied a consecutive series of 670 cases of abdominal obstruction. In 62 a segment of the intestine was not considered viable and a resection was carried out; 48 of these patients died, giving a fatality rate of 77.4 per cent. Of 300 patients with strangulated external hernia 30 required resection; 24 of these died, a fatality rate of 80 per cent. Of the remaining 270 patients having simple release only 18 died, giving a fatality rate of 6.7 per cent. In trying to determine the reason for this significant difference in the prognosis when the intestine changes from viable to nonviable the authors find that the majority survive the immediate hazards of operation and the next few days. They may die at any time up to six weeks after operation, the most dangerous periods being about the fifth and the 10th day. The symptoms are typical of an increasing mechanical obstruction—colicky pains, exaggerated peristalsis, vomiting and increasing distention. At necropsy the cause of death is found to be a plastic peritonitis of a variable degree, with much adhesion of intestine to intestine and intestine to abdominal wall, and an edematous intestine full of foul material, with multiple sites of obstruction. The author believes that some organisms probably pass through the intestinal wall before operation but not many unless gangrene and perforation are present; the main contamination of the peritoneum probably takes place at the main contamination of the intestine being opened. If the 670 patients are placed in two groups, according to whether the intestine was opened or not, there are 224 whose intestine was opened, of whom 137 died; but of 446 having only simple release 23 died. The prognosis for patients requiring opening of the intestine was necessarily much worse, but the difference is so great as to suggest that opening the intestine provides a new lethal factor sufficient to weigh the scales against the patient. Reduction in the deaths can be attained by earlier diagnosis and by more successful treatment and prevention of the lethal complications which result from delay in diagnosis. With a view to preventing such complications, experiments with intraperitoneal sulfanilamide in rabbits demonstrate that (1) sulfanilamide introduced into the peritoneal cavity was absorbed and caused no local inflammation, (2) contamination of the peritoneal cavity with contents of the cecum produced a plastic peritonitis with the formation of abscesses and adhesions and sometimes general peritonitis, (3) this plastic peritonitis could be prevented by introducing sulfanilamide into the peritoneal cavity at the time of the contamination but not by a single large dose of the drug orally, (4) *Bacillus coli* peritonitis followed resection of intestine with

great regularity and (5) prophylactic use of intraperitoneal sulfanilamide usually reduced and sometimes eliminated the inflammatory process, but (6) in the presence of a fulminating infection intraperitoneal sulfanilamide was valueless. So far 2 patients having intestinal resection for acute intestinal obstruction have been treated with sulfanilamide intraperitoneally. In neither of them was there any local or general postoperative obstruction or peritonitis.

(Lancet.)

METABOLIC THERAPY OF CANCER

BY PROFESSOR ERNST FREUND, F.R.S.M.

*Professor of Pathological Chemistry of University Vienna ;
Director, Pearson Cancer Research Foundation.*

The development of malignant tumours is due to abnormal cellular activity arising from

- (a) A Congenital defect
- (b) Infection
- (c) Chronic irritation.

Any one or all these factors influence the general metabolism and produce cancer. The author taking into consideration these aspects of cancer have evolved a metabolic therapy. These results may be summarised as follows:

(1) The normal blood serum is said to contain a specific fatty acid "normal acid" which destroys carcinoma and sarcoma cells and prevents their development. The various organs of the individual also possess a similar property to that of the serum, this being most potent in the thymus particularly in relation to carcinoma.

(2) The cancerous serum and organism contains other specific fatty acid ("Carcinoma acid" and "Sarcoma acid") which nourish the malignant cells and protect them from destruction by normal serum.

Carcinoma and sarcoma acids are antagonistic to each other.

(3) The abnormal acids are produced from the food in the intestine through some pathological change.

(4) The production of abnormal acid in the intestine constitutes general disposition to cancer, but can not in itself lead to cancer in the absence of a local disposition where both these factors are present cancer may arise through the following process:

The chronic irritation involves an excessive consumption of the normal acid by the cells at the point concerned. When the sufficient

supply of normal acid ceases the carcinoma acid and the irritated cells become pathological. The result is cancer. Therefore it is necessary that the general as well as local treatment must be carried out simultaneously.

The carcinoma or sarcoma acid is produced from certain food-stuffs in the intestine. This process is not caused by the tumour but it may exist for years before the appearance of the malignant growth and also persist after its radical removal. Based on the above principles the following metabolic therapy is used:

(a) Intestinal disinfection by means of volatile oils and bismuth salicylate.

(b) A strict diet. In the case of carcinoma animal fats are excluded and carbohydrates are limited. In the case of sarcoma proteins are restricted.

(c) Administration of normalising substances such as thymus extracts in high concentrations.

The author tried the above treatment on 40 inoperable, hopeless cases of cancer. Beneficial results were seen in a good number of these. This therapy is not to be called "a cancer cure" but is to be employed in all cases before and after surgical treatment, parallel with radiological treatment and in the prophylactic sense.

(The Medical Press and Circular.)

Medical Notes & News.

WEST GODAVARI DISTRICT BRANCH OF INDIAN MEDICAL ASSOCIATION, ELLORE.

The first business meeting of the above association for the year 1942-43 was held at Municipal Council Hall of Ellore on 6-12-1942. Tea was served, the host of the evening being Dr. C. Satyanarayana Sastry, M.B.B.S. After tea, Dr. V. Narasimharao, B.Sc., L.M.S., was proposed to the Presidential chair and he conducted the elections of the office bearers for 1942-43. The following office bearers were unanimously elected for the year 1942-43.

1. *President*:—Dr. V. Venkataratnam, M.B.B.S.
2. *Vice-President*:—Rao Saheb Dr. M. Narasinga Rao Naidu, L.M.P., L.S.M.F.
3. *Secretary*:—Dr. C. Satyanarayana Sastry, M.B.B.S.
4. *Representative for the Andhra Provincial Council*: Dr. V. Narasimha Rao, B.Sc., L.M.S.

5. Committee Members:

- (i.) Dr. Miss K. V. John, L.M.P.
- (ii.) Dr. C. P. Satyanarayan, L.M.P.
- (iii.) Dr. D. Rajasekhararao, M.B.B.S.
- (iv.) Dr. D. Subba Rao, M.B.B.S.
- (v.) Dr. K. Suryanarayana, L.M.P.

The following resolutions were unanimously passed:—

1. Resolved that the annual subscription from each member of the Association be raised from Rs. 4 to Rs. 5 per annum.
2. Resolution congratulating Dr. M. Narasinga Rao Naidu on his getting the Rao Saheb title.
3. Resolved to try by all means to amalgamate this Association with the West Godavari District Medical Association.
4. Resolved to request the Andhra Provincial Branch of the Indian Medical Association, Masulipatam, to sanction Rs. 50 only this year, for enlisting more members from the West Godavari District.

THE KISTNA DISTRICT MEDICAL ASSOCIATION,
MASULIPATAM.

Minutes of the monthly Meeting of the Kistna District Medical Association, Masulipatam, held on 17-12-1942, Thursday, in the Government Hospital, Bezwada.

23 Doctors and 1 Lady Doctor attended.

Tea was served then the Business Meeting began with Dr. N. Sujeer, President, in the chair.

Dr. M. V. Subbarao, Secretary, read the minutes of the last meeting held at the Government Hospital, Gudivada, on 28-11-1942, which was passed unanimously.

Read letter C-107/42-43, dated 22-11-1942, from the Honorary General Secretary of I. M. A. Calcutta.

Read letter dated 20-11-'42, from the President, Bengal Medical Relief Committee, and collected some voluntary funds.

Then the Medical Meeting began at 5.45 P. M.

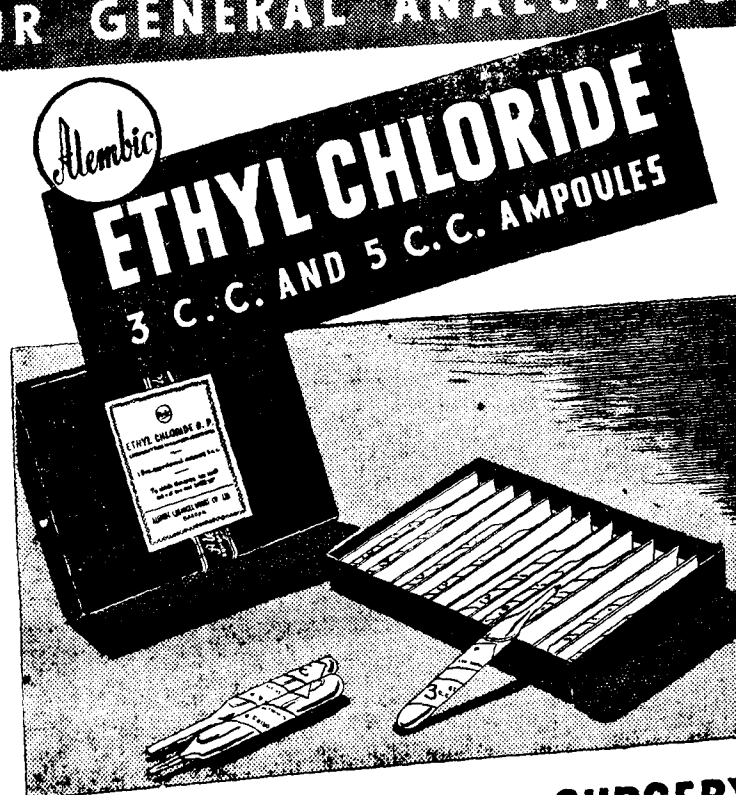
Dr. M. V. Subbarao, read two cases of Puerperal Tetanus.

Dr. N. Ramarao, read a case of Acute Abdomen.

Then the President in his concluding remarks discussed the differential diagnosis of the above cases.

The meeting concluded with vote of thanks by the Secretary.

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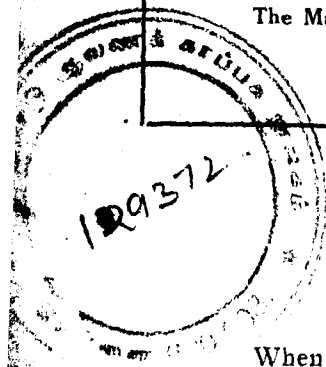
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