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UNIVERSITY OF MINNESOTA

Medical Bulletin

OFFICIAL PUBLICATION OF THE
UNIVERSITY OF MINNESOTA HOSPITALS
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IN THIS ISSUE:

University of Minnesota Medical Bulletin

University of Minnesota Medical School

University Hospitals

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Medical Bulletin

OFFICIAL PUBLICATION OF THE UNIVERSITY OF MINNESOTA HOSPITALS, MINNESOTA MEDICAL FOUNDATION, AND MINNESOTA MEDICAL ALUMNI ASSOCIATION

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CONTENTS

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Staff Meeting Report

Deficient Abdominal Gas Pattern in Infants*

Samuel B. Feinberg, M.D.,[1] Alexander R. Margulis, M.D.,[2] and Charles M. Nice, M.D.[3]

Not infrequently, unnecessary surgery has resulted from the misconception that a gasless abdomen in an infant indicates mechanical obstruction, especially if the infant is vomiting. However, the condition may be produced by 3 distinct mechanisms: (1) mechanical obstruction, (2) mechanical factors associated with dehydration, and (3) unrelated causes of dehydration.

Since intestinal motility begins as early as the twelfth fetal week and air swallowed at birth pervades the entire gastrointestinal tract within two to six hours, the accumulation of gas is normal in infants. The absence of gas has been demonstrated radiographically in newborn infants with severe respiratory distress due to massive atelectasis or pneumothorax but without signs of abdominal disease. With expansion of the lung, the gas pattern becomes normal.

Mechanical Obstruction

The lesion of intrinsic high intestinal obstruction is an obstructive diaphragm or complete closure resulting from disturbed canalization of the primitive gut between the fourth and tenth weeks of fetal life. Complete stenoses are usually single and located in the duodenum, whereas atresias are usually multiple and situated in the ileum.

A common cause of extrinsic duodenal obstruction is intestinal malrotation, frequently associated with a common omentum and obstructing peritoneal bands. The duodenal loop often extends caudally and may pass through the mesentery of the ileum. Volvulus involving the entire midgut may be a consequence of a narrow mesenteric root at the duodenal-jejunal junction, leading to circulatory embarrassment and venous stasis. Not uncommonly, an annular pancreas encircling the duodenum may cause obstruction.

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*This is an abstract of a report given at the Staff Meeting of the University of Minnesota Hospitals on November 25, 1955. A copy of the complete report, including tables and references, may be obtained by writing to the Editor, UNIVERSITY OF MINNESOTA MEDICAL BULLETIN, 1342 Mayo Memorial, Minneapolis 14, Minn.

[1]Clinical Assistant Professor, Department of Radiology.
[2]Instructor, Department of Radiology.
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Exemplifying distention, obstruction, and gaslessness from these causes are the cases of (1) J.C., who at autopsy was found to have stenosis of a 1.5-cm. segment which ended at the ampulla of Vater; (2) B.F., in whom, during operation, a 0.5-cm. band of annular pancreas was found at the junction of the second and third portions of the duodenum; and (3) B.S.S., in whom operation disclosed a twisted transverse mesocolon with bands affixing the cecum and appendix across the second and third portions of the duodenum.

Combined Mechanical and Dehydration Factors

Complete anatomic obstruction with hypertrophic pyloric stenosis is rare. To correlate the degree of intestinal gaslessness and dehydration in this condition, surgically proved cases seen at University Hospitals during 1947-55 were reviewed.

Among 40 patients who had had preoperative roentgen studies, 20 were severely dehydrated, 5 had gastric dilatation and diminished gas, one had normal infantile meteorism, and 14 had elevated and 2 had normal carbon-dioxide combining power; one of the preceding had a normal gas pattern after hydration.

Examples of the development of severe or moderate dehydration and complete or partial gaslessness after prolonged vomiting for two or three weeks are 3-week-old ., 6-week-old ., and 8-week-old ., in each of whom a palpable mass in the right upper quadrant and visible peristalsis appeared.

At operation, no anatomic differences were found to explain the greater severity of obstruction in cases of gasless abdomen. Clinically, the 3 groups differed only in the amount of hydration. In 2 dehydrated infants, the amount of gas in the small intestine increased after administration of fluids. If, however, mechanical obstruction alone were the cause of both gaslessness and dehydration, correction of the latter probably should not change the amount of gas.

Mechanical causes of dehydration were apparent in the cases of and . .. had a tracheoesophageal fistula repaired and a gastrostomy performed. Failure to function, impaired feeding, and dehydration and gaslessness resulted. a 7-day-old male, had had jaundice and vomiting since birth. High obstruction was suspected. At operation, however, sudden narrowing of the small intestine beyond the ligament of Treitz was found, and no obstruction existed. Final diagnosis was neuromuscular dysfunction.

The Gasless Abdomen in Dehydration

With severe infantile diarrhea of specific or nonspecific origin, objective and roentgenographic findings suggest generalized intestinal obstruction, although in gravely ill patients the abdomen is commonly scaphoid.

A normal male, , refused feedings after the first seventy-two hours, vomited, passed green stools, and became dehydrated and jaundiced. A scout film showed a deficiency of gas. Cultures were positive for coagulase-negative staphylococci. At autopsy, the glandular epithelial changes were compatible with a diagnosis of Ritter's disease.

A premature male, ., was normal until the fourth day, when purulent yellow stools were passed. Ten days later, the abdomen was gasless caudad to the duodenum, and cultures were positive for *Pseudomonas æruginosa.* Superficial ulcers of the colon and enlarged mesenteric lymph nodes were observed at autopsy.

Primary adrenal insufficiency with dehydration and electrolyte imbalance and adrenal exhaustion resulting from vomiting, diarrhea, and dehydration produce a deficient gas pattern and signs simulative of the high intestinal obstruction observed with hypertrophic pyloric stenosis. Clinical and roentgenographic differentiation is difficult, but laboratory determinations are helpful.

Paradoxically, vomiting, which causes alkalosis, and severe diarrhea, which induces acidosis, produce the same roentgenographic picture. In both conditions, however, the factors of dehydration and loss of potassium are common.

Staff Meeting Report

Treatment of Grade 4 Hemorrhoids*

William T. Smith, M.D.[1]

Grade 4 hemorrhoids may be defined as internal rectal varicosities that are permanently prolapsed externally through the anal canal. Attempts at manual replacement by the patient or physician may be impossible or, if the hemorrhoids can be replaced, they promptly prolapse again. They are, in essence, internal piles that are always on the outside. In practically every instance the prolapsed hemorrhoidal tissue is accompanied by eversion or sliding down of the lining of the anal canal and protrusion of redundant rectal mucosa. The tonus of the sphincter mechanism, particularly the external, is usually quite relaxed. This represents a fatigue of the striated muscle bundles secondary to chronic stretching and dilation of the anal canal and sphincter muscle by the mass of protruding mucosa and hemorrhoidal tissue. When the condition is corrected, the tonus will return and anal continence will be re-established. Permanent incontinence is rare.

Simple prolapse of redundant mucosa may be severe enough to evert the anal canal and be mistaken by the uninitiated for grade 4 hemorrhoids. Grade 4 hemorrhoids are rarely confused with rectal procidentia or complete prolapse of all the walls of the rectum. Occasionally hyperplastic plaques are noted on the prolapsed tissue. These may appear grayish white and thickened and feel indurated when palpated. They may be suggestive of cancer and should always be sectioned after excision although the incidence of malignancy is low. One of the primary functions of the squamous epithelium lining the anal canal is to provide the shut-off mechanism with a relatively dry, elastic surface for coaption. The length of the anal canal varies markedly. In some instances the squamous epithelium may not extend more than 1 cm. from the anus, or it may measure 4 or 5 cm. and be surrounded by heavy sphincter musculature. The anal canal normally extends from the anal verge caudad to the mucocutaneous junction cephalad. Embryologically it arises from an invagination of the anal plate, an ectodermal structure which unites with the rectal

*This is a report given at the Staff Meeting of the University of Minnesota Hospitals on December 2, 1955.

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pouch early in fetal life.

The principles involved in treating grade 4 hemorrhoids of the type mentioned are: 1) Excise all hemorrhoidal tissue. 2) Excise all redundant rectal mucosa. 3) Leave enough rectal mucosa to line the distal rectum. 4) Provide the anal canal with a squamous type epithelial lining.

Grade 4 hemorrhoids may be encountered as the final stage in a slowly progressive process in which the anal lining, hemorrhoids, and redundant mucosa are constantly prolapsed externally in one or more quadrants or as a relatively acute process in which there is a massive thrombosis in one or all quadrants of large interno-ano-external hemorrhoids, all components being externally prolapsed.

A surgical method of treating these in the past was the Whitehead operation. In this procedure all the prolapsed internal hemorrhoids were excised. The anal and external hemorrhoids were also excised and the remaining anal canal was sutured to the rectal mucosa. Unfortunately in many instances the actual technique of the operation was not understood and all prolapsing tissue was cut off flush with the anal orifice and the rectal mucosa sutured to the anal orifice. This left the anal canal lined with red, juicy rectal mucosa. The resulting deformity is commonly referred to as a Whitehead deformity.

Attempts to treat grade 4 hemorrhoids with injection of sclerosing agents, e. g., 5 per cent phenol in oil, Sylnasol, are attended with failure to replace permanently the prolapsed tissue into the rectum. Such injections are usually accompanied by much pain. Very often slough and hemorrhage occur. Needless to say injection has no place in treating grade 4 hemorrhoids except as palliation in debilitated patients. Application of the various routine techniques for hemorrhoidectomy is usually impossible because of the distorted anatomy. If these methods are used, again the result is an anal canal lined with mucous membrane plus numerous perianal skin tags. Patients with an anal canal lined with mucous membrane have a constantly discharging anus with wet, irritated and itching perianal skin and perineum. In addition, the outer edge of the anal canal is surrounded by a band or ring of thick, inelastic scar tissue which commonly cracks and fissures with bowel movements.

In our experience the most suitable method of treating grade 4 hemorrhoids is the technique of plastic hemorrhoidectomy. Actual skin flaps in one to four quadrants are constructed from the prolapsed everted anoderm and the anal canal is relined with this tissue. Dis-

section is begun by transverse incision at the junction of the prolapsed anoderm and mucosa, usually in the left lateral quadrant. The incision is extended anteriorly and posteriorly along the mucocutaneous junction to a point at midline posteriorly and midline anteriorly. The skin edges are then grasped at each end with Allis forceps, and a flap is constructed by excising the underlying hemorrhoidal tissue, clots, and scar tissue. A flap 3 to 5 cm. in length is usually quite adequate to reconstruct the anal canal. The remaining prolapsed hemorrhoidal tissue and redundant mucosa in this quadrant are carefully excised until the sphincter musculature is visible. The skin flap is then drawn into the anal aperture and sutured with interrupted chromic catgut to the edge of the rectal mucosa at a point where formerly the correct anorectal anatomical junction existed. Each stitch should include a good bite of the underlying circular muscle to insure good fixation of the flap. Usually two separate flaps are constructed on the left and two on the right. They must not be sutured to the rectal mucosa under too much tension or slough will result. Actually in many cases only one or two skin flaps are necessary and this is determined by the degree of prolapse in the various quadrants. After the operation is completed a roll of No. 100 Gelfoam gauze is left in the anal canal and a firm dressing applied.

Preoperative preparation of the bowel with antibiotics usually improves healing processes. Although it is highly desirable, it is not absolutely essential to prepare the bowel before this procedure is done. Postoperatively, sulfasuxidine or sulfathaladine is usually given orally; otherwise routine post-hemorrhoidectomy orders are followed. In order to avoid the separation of the skin flaps patients are warned not to strain at stool. Common complications peculiar to this procedure, other than those often encountered after hemorrhoidectomy, are sloughing of a skin flap or sloughing of all flaps, which is relatively rare, and stenosis at the reconstructed anorectal junction. Careful postoperative observation of the patient and early, frequent dilatation will prevent the latter. Once extensive sloughing has occurred little can be done except to keep the patient well dilated and to attempt to aid epithelization of the raw surfaces. Slough of a single flap is seldom very serious because of the presence of adequate islands of epithelium which will proliferate and eventually cover the defect.

Grade 4 hemorrhoids represent a not uncommon anorectal condition which requires special operative techniques for its correction. The plastic hemorrhoidectomy technique outlined will give a good result if properly applied.

Editorial

Some New Horizons for Clinical Medicine

Several recent advances in immunology and related fields hold promise of important and ultimately far-reaching clinical application. Among the more important of these "break-throughs" has been discovery of acquired tolerance to antigenic substances.

In 1949 Burnet and Fenner, on the basis of their theory of antibody formation, predicted that animals would be forever incapable of specific responses to antigens to which they were exposed during fetal life. They were unable, however, to add to the indirect experimental data (Owen, 1945; Rawles, 1948) available supporting this idea (Burnet, et al, 1950). Billingham, Brent, and Medawar (1953) supplied the first sound experimental evidence that the prediction was correct. They discovered that mice given living cells during fetal life did not slough or "reject" skin homografts from the donor of the cells even after the recipient animal had reached full maturity. As would be expected, control homografts from other genetically distinct donors were promptly rejected by the same animals. Retention of homografts by virtue of exposure to donor cells *in utero* has been termed "acquired tolerance."

The work of Medawar's group, and especially the experiments reported by Mitchison (1953) have provided convincing evidence that homograft rejection is based upon immunologic mechanisms very closely related to, if not identical with, bacterial type (tuberculin type or delayed type) hypersensitivity. The term "transplantation immunity" has been used to describe the process by which the host rejects homografts. It was natural then that these experiments immediately attracted wide interest among experimental immunologists as well as those interested in skin and organ grafting and in cancer biology.

Investigators in numerous laboratories in Great Britain and the United States are known to be engaged in attempts to understand and extend the phenomena of transplantation immunity and acquired tolerance. Important and practical steps forward have been made recently by the demonstration that acquired tolerance to homografts can be produced in rats by implanting cells of the prospective donor on the first day of life (Woodruff and Simpson, 1955), and that permanent and specific inability of rabbits to respond to purified protein antigens results from being exposed to the antigen on the first day

of life (Cinader and Dubert, 1955). Similar observations have been made independently in our laboratory (Smith and Bridges, 1955) which confirm the British work. Thus far no studies of acquired tolerance have been made in human beings; such investigations seem in order.

Actual production in mature animals of a state closely related to acquired tolerance recently has been shown in several ways. Felton (1949) found that mice injected with relatively large amounts of pneumococcal polysaccharide developed "immunological paralysis." Following this treatment they appeared to lose their capacity to form antibody to this specific antigen. This work has been confirmed (Morgan, et al, 1953; Stark, 1954, Dixon, et al, 1955) in several laboratories and extended to include protein antigens. (Hanan and Oyama, 1954; Dixon and Maurer, 1955; Johnson, et al, 1955.) Since our preliminary data suggest that acquired tolerance to a protein antigen is a quantitative phenomenon as is "immunological paralysis" produced in the mature animals, similar mechanisms may underlie these phenomena.

Kaliss and Snell (1951), interested in the immunological and genetic aspects of cancer growth, showed that normally resistant animals exposed to large amounts of lyophilized-killed tumor became highly susceptible to the tumor. Repeated inoculations of large amounts of fresh tumor have a similar effect (Snell, 1953). On the basis of strong evidence that rejection of such transplanted tumors, like normal homotransplanted skin, has an immunological basis (Medawar, 1954), it can be reasoned that these animals may have developed the equivalent of "immunological paralysis" and as a consequence were unable to prevent growth of the neoplasm. Therefore, "immunological paralysis," with its characteristics of a high degree of specificity, permanence, and dependence for induction on a large dose of antigenic material, has been shown for pure antigens, and for malignant mammalian tumors. As yet it has not been possible to produce this state with normal tissues in animals or man.

A last twist of this newly available line of information concerns ways of reversing tolerance or paralysis once produced. Chase and co-workers (1942) first demonstrated that bacterial-type (delayed or tuberculin type) hypersensitivity could be transferred from a sensitive animal to a non-sensitive one by way of intact transferred leukocytes. Transfer of "transplantation immunity" to tumors or homografted skin via leukocytes or reticuloendothelial cells has since been demonstrated (Billingham, et al, 1954; Mitchison, 1953). Recently it has

been shown that transfer of spleen cells from animals which have rejected skin grafts from a given donor to animals previously rendered tolerant to homografts from the same donor by *in utero* exposure, results forthwith in rejection of the previously tolerated graft (Billingham, et al, 1953). The capacity to reject homografts was restored by the transferred cells. While restoration of ability to respond to a specific antigenic protein in the immunologically paralyzed animal has not yet been demonstrated, there is indirect evidence to indicate that this will be possible (Mitchison, 1955; Harris, et al, 1954).

Another interesting development has been the discovery of a defect in the immune mechanism of certain human patients. In agammaglobulinemia a generalized immunological deficiency exists. In the congenital form of this disease, the immunological defect extends to all antigens thus far studied. The fact that these patients accept skin homotransplants from genetically unrelated donors suggests that in man as well as in animals homotransplantation failure has an immunologic basis. It seems possible that in this inborn error of protein metabolism disturbances in mechanisms of protein synthesis are similar to those responsible for "acquired tolerance" or "immunological paralysis" may be operating.

It seems probable that elucidation of the mechanism underlying any of these several phenomena will make homotransplantation a reality, but currently, prediction of clinical application for the aforementioned interrelated phenomena is fraught with uncertainty and inherently unwise. However, labeled clearly as pure speculation, it may be in order to mention a few of the implications in clinical fields.

If it is possible to achieve tolerance by exposing human infants to antigens or cells at birth, it would seem logical, for example, to attempt to prevent erythroblastosis by giving baby girls Rh antigens at birth so they would be permanently unable to make that antibody and thus would be unable to be sensitized by their ultimate offspring; or by giving the newborn infant cells from its mother, father, or other donors to provide him with a "buddy" who could supply homografts in cases of dire need. Were this last example to prove eventually possible or practical, the psychiatric, economic, and sociological impact involved may well be imagined.

Requiring less wild flight of imagination is the possibility that understanding the mechanism of acquired tolerance will lead to practical ways of producing, directing, and controlling immune paralysis to specific proteins or tissues in mature animals and human beings. Practical control of the immune response in this way will have im-

mense potential in clinical medicine. For example, controlled homotransplantation would not only allow provision of replacements for deficient or diseased organs (i. e., diabetes, Addison's disease, myxedema, chronic nephritis, etc.), but allow the surgeon in eradicating cancer to sacrifice organs now considered essential.

The newly acquired knowledge of the immune mechanism also has important applications in cancer biology and therapy. It seems entirely within the realm of possibility that understanding of the strange capacity of the animal or human patient with cancer to acquire tolerance for his malignantly transformed cells may ultimately result in development of techniques for reversing the uncontrolled growth. If, for example, a malignant neoplasm may be considered as a genetically closely related homograft, then production of "transplantation immunity" to the tumor in a genetically unrelated host might be transferred to the patient via leukocytes, with rejection of the homotransplant and lysis of the tumor.

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Alumni Association

Since June, 1954, DR. KARL R. LUNDEBERG, '25, has been Commissioner of Health for the city of Minneapolis. Dr. Lundeberg is a retired colonel in the U. S. Army Medical Corps, and he also serves as a visiting lecturer for the School of Public Health at the University of Minnesota.

DR. LEONARD S. LINNELL, '53, is in Tanganyika, East Africa, serving as a medical missionary in that area where he will remain until 1958.

DR. LEONARD W. LARSON, '21, Bismarck, North Dakota, is a member of the Board of Trustees of the American Medical Association, Past President of the North Dakota State Medical Society, and President of the Joint Blood Council, Inc. In 1953 he was awarded a medal for his services to the American Cancer Society. He also holds the certificate of highest merit of the American Society of Clinical Pathologists.

DR. GEORGE M. LANDROCK, '20, San Rafael, California, is *Assistant Clinical Professor of Surgery* at the University of California Medical School.

DR. ROBERT G. KVARNES, '37, Chevy Chase, Maryland, has been *Executive Director and Dean of Students* at the Washington (D. C.) School of Psychiatry since 1953.

DR. IRVIN KERLAN, '33, received a Super Service Medal given by the U. S. Department of Health, Education, and Welfare in 1954. He is a member of the Committee on Toxicology of the American Medical Association and of the Committee of Aging of the Department of Health, Education, and Welfare.

DR. VINCENT C. KELLEY, '45, *Associate Professor of Pediatrics* at the University of Utah, Salt Lake City, received the 1954 Mead Johnson Award for Pediatric Research.

DR. EARL A. LOOMIS, JR., '45, is *Associate Professor of Child Psychiatry* at the University of Pittsburgh Medical School.

DR. SHERMAN N. KIEFFER, '50, is *Instructor in Psychiatry* at Johns Hopkins College of Medicine and Chief of the Psychiatric Service at the U. S. Public Health Service Hospital in Baltimore.

DR. JAMES R. JUDE, '53, holds a *Cushing Fellowship in Surgery* at the Johns Hopkins Hospital in Baltimore.

IN MEMORIAM

Notice has been received of the deaths of the following members of the Minnesota Medical Alumni Association. We wish to extend our sympathy to members of their families.

DR. CARL M. PETERSON, '26, Chicago, Illinois
DR. CARLYLE TINGDALE, '37, Hibbing
DR. EUGENE MC CANN, '30, Minneapolis
DR. JOHN A. THABES, JR., '26, Brainerd
DR. SAMUEL MILLER, JR., '28, Jamestown, North Dakota
DR. PHILIP ARLING, '40, McMinnville, Oregon
DR. PERCIVAL H. BENNION, '02, Miles City, Montana
DR. FRANK T. BENOIT, '05, Winona
DR. ROBERT W. CAMPBELL, '05, Cass Lake
DR. FREDERICK W. ENGDAHL, '33, Dallas, Texas
DR. C. O. ESTREM, '07, Fergus Falls
DR. ALLOYS F. BRANTON, '19, Chattanooga, Tennessee
DR. BERNARD A. LUCKING, '41, Helena, Montana
DR. MICHAEL F. HAYES, '09, Nashwauk
DR. HENRY W. QUIST, SR., '07, Minneapolis
DR. RALPH H. CREIGHTON, '23, Minneapolis
DR. CHARLES E. DUTTON, '89, Minneapolis
DR. STEPHEN H. BAXTER, '02, Minneapolis
DR. EDGAR W. DANNER, '95, New York City, New York
DR. CHARLES A. DAWSON, '03, River Falls, Wisconsin
DR. HARRY J. GOWENLOCK, '04, Jamestown, North Dakota
DR. ASA J. H. HAMMOND, '96, Minneapolis
DR. HERBERT T. HANSON, '46, Salt Lake City, Utah
DR. ENOCH HAUGSETH, '02, Twin Valley
DR. VICTOR G. HAURY, '34, Wellsville, Kansas

Postgraduate Education

Obstetrics for General Physicians

The University of Minnesota will present a continuation course in Obstetrics for General Physicians at the Center for Continuation Study from January 5 to 7, 1956. Guest faculty will include DR. LANCE TOWNSEND, *Professor and Head, Department of Obstetrics and Gynecology,* University of Melbourne Faculty of Medicine, Melbourne, Australia. The course will be presented under the direction of DR. JOHN L. MC KELVEY, *Professor and Head, Department of Obstetrics and Gynecology.*

Neurology and Neurosurgery for General Physicians and Specialists

Neurology will be the subject of a continuation course to be presented by the University of Minnesota at the Center for Continuation Study from February 6 to 11, 1956. Intended primarily for physicians in general practice, the program will have appeal also for neurologists and neurosurgeons. The most commonly seen neurological symptoms and syndromes will be stressed. Guest faculty will include DR. WILLIAM M. MEACHAM, *Associate Clinical Professor of Surgery,* Vanderbilt University School of Medicine, Nashville; DR. MORRIS B. BENDER, *Director, Neurology Service,* Mount Sinai Hospital, and *Professor, Clinical Neurology,* New York University College of Medicine, New York City; DR. JOHN F. SULLIVAN, *Associate Professor and Head, Department of Neurology,* Tufts College Medical School, Boston; and DR. OLIVER H. LOWRY, *Professor and Head, Department of Pharmacology,* Washington University School of Medicine, St. Louis. The course will be presented under the direction of DOCTORS A. B. BAKER, *Professor and Director, Division of Neurology,* and WILLIAM T. PEYTON, *Professor and Director, Division of Neurosurgery.*

Notice

All physicians may receive a free subscription to "CA — A Bulletin of Cancer Progress," a publication of the American Cancer Society. Physicians wishing to receive this bimonthly digest of cancer information may write to the Minnesota Division, American Cancer Society, 295 North Snelling Avenue, St. Paul 4, Minnesota.

Coming Events

January 5–7	Continuation Course in Obstetrics for General Physicians
January 30–February 1	Continuation Course in Emergency Surgery for General Physicians
February 2–4 . . .	Continuation Course in Mental Deficiency for General Physicians, Pediatricians, Obstetricians, and Child Psychiatrists
February 6–11 . . .	Continuation Course in Neurology and Neurosurgery for General Physicians and Specialists
February 8	J. B. JOHNSTON LECTURE; *Dr. Oliver H. Lowry,* Professor and Head, Department of Pharmacology, Washington University School of Medicine, St. Louis, Missouri; Mayo Memorial Auditorium; 8:15 P.M.
February 13–15 . .	Continuation Course in Internal Medicine for Internists
February 16–18 . .	Continuation Course in Cancer Detection for General Physicians
February 27–29 . .	Continuation Course in Eye, Ear, Nose, and Throat for General Physicians

Faculty Publications

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WEEKLY CONFERENCES OF GENERAL INTEREST

Physicians Welcome

Monday,	9:00 to 10:50 A.M.	OBSTETRICS AND GYNECOLOGY Old Nursery, Station 57 University Hospitals
	4:00 to 6:00 P.M.	ANESTHESIOLOGY Todd Amphitheater, University Hospitals
Tuesday,	12:30 to 1:20 P.M.	PATHOLOGY 104 Jackson Hall
Thursday,	12:00 to 1:00 P.M.	PHYSIOLOGY 214 Millard Hall
Friday,	8:00 to 10:00 A.M.	NEUROLOGY Station 50, University Hospitals
	9:00 to 10:00 A.M.	MEDICINE Todd Amphitheater, University Hospitals
	1:30 to 2:30 P.M.	DERMATOLOGY Eustis Amphitheater, University Hospitals
Saturday,	7:45 to 9:00 A.M.	ORTHOPEDICS Powell Hall Amphitheater
	9:15 to 11:30 A.M.	SURGERY Todd Amphitheater, University Hospitals

For detailed information concerning all conferences, seminars and ward rounds at University Hospitals, Ancker Hospital, Minneapolis General Hospital and the Minneapolis Veterans Administration Hospital, write to the Editor of the BULLETIN, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.