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Pulmonic Stenosis

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UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

Visitors Welcome

November 1 - 6, 1948

No. 220

Monday, November 1

9:00 - 9:50	Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.
9:00 - 10:50	Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; Interns' Quarters, U. H.
8:00 -	Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.
10:00 - 12:00	Neurology Ward Rounds; A. B. Baker and Staff; Station 50, U. H.
11:00 - 11:50	Roentgenology-Medicine Conference; Staff, Veterans' Hospital.
11:00 - 12:00	Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.
11:00 - 11:50	Physical Medicine Seminar; Anatomy of the Upper Extremities; Myron Lecklitner; E-101, U. H.
12:00 - 1:00	Physiology Seminar; M. B. Visscher; 214 M. H.
12:15 - 1:20	Obstetrics and Gynecology Journal Club; M-435, U. H.
12:30 - 1:20	Pathology Seminar; 104 I. A.
12:30 - 1:50	Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.
1:30 - 2:30	Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.
2:00 - 3:00	Surgery Problem Case Conference; C. Dennis and Staff; Small Class Room, General Hospital.
3:45 -	Pediatric Seminar; Tyrosine Metabolism; Mr. Robert Salmon; 6th Floor, Child Psychiatry, U. H.
4:00 - 6:00	School of Public Health Seminar; 113 MeS.
5:00 - 6:00	Urology-Roentgenology Conference; D. Creevy and H. M. Stauffer and Staffs; M-109, U. H.
5:00 - 5:50	Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

Tuesday, November 2

8:30 - 10:20 Surgery Reading Conference; Lyle Hay; Small Conference Room, Bldg. I, Veterans' Hospital.

9:00 - 9:50 Roentgenology Pediatrics Conference; L. G. Rigler, I. McQuarrie and Staff; Todd Amphitheater, U. H.

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Robert Hebbel; Veterans' Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; Pathology Staff; 102 I. A.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III, Veterans' Hospital.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

3:30 - 4:20 Clinical Pathological Conference; Staff; Veterans' Hospital.

4:00 - 5:30 Surgery-Physiology Conference; O. H. Wangensteen and M. B. Visscher; Eustis Amphitheater, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

5:00 - 5:50 Urology Pathological Conference; C. D. Creevy and Staff; Todd Amphitheater, U. H.

5:00 - 6:00 X-ray Conference; Dr. Rigler and Staff; Powell Hall Amphitheater.

Wednesday, November 3

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-515, U. H.

8:30 - 10:00 Orthopedic-Roentgenologic Conference; Edward T. Evans; Room 1AW, Veterans' Hospital.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker and Joe R. Brown; Veterans' Hospital.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; O. H. Wangensteen, C. J. Watson and Staff; Todd Amphitheater, U. H.

12:00 - 12:50 Radio Isotope Seminar; Neutron Beams (Fast and Slow) -- (a) Reactions; Cross-Section Determinations, (b) Protection; Mr. James Marvin; Rm. 216, Hospital Court, Temporary Bldg.

4:00 - 5:00 Infectious Disease Rounds; Medical Conference Room, Veterans' Hospital.

Thursday, November 4

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and E. M. Stauffer; M-109, U. H.

8:30 - 10:20 Surgery Grand Rounds; Lyle Hay and Staff; Veterans' Hospital.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Surgery-Radiology Conference; Daniel Fink and Lyle Hay; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

11:30 - 12:30 Clinical Pathology Conference; Steven Barron, C. Dennis, George Fahr, A. V. Stoesser and Staffs; Large Class Room, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; George Snively; 214 M. H.

1:00 - 1:50 Fracture Conference; A. A. Zierold and Staff; Minneapolis General Hospital.

4:00 - 5:00 Bacteriology and Immunology Seminar; Serological Studies in Polio; Mrs. H. Brumfield; 214 M. H.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

5:00 - 6:00 X-ray Seminar; Todd Amphitheater.

Friday, November 5

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:20 Medicine Grand Rounds; Staff; Veterans' Hospital.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boies and Staff; Out-Patient Department, U. H.

11:00 - 12:00 Surgery-Pediatric Conference; C. Dennis, A. V. Stoesser, and Staffs; Minneapolis General Hospital.

11:30 - 12:50 University of Minnesota Hospitals General Staff Meeting; Vocal Cord Paralysis; L. R. Boies and Harold Ulvestad; Powell Hall Amphitheater.

12:00 - 1:00 Surgery Clinical Pathological Conference; Clarence Dennis and Staff; Minneapolis General Hospital; Small Classroom.

1:00 - 1:50 Dermatology and Syphilology; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

Saturday, November 6

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; Station 21, U. H.

8:00 - 9:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor, West Wing, U. H.

8:00 - 9:00 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital, Small Classroom.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, U. H.

9:00 - 9:50 Surgery-Roentgenology Conference; O. H. Wangensteen, L. G. Rigler, H. M. Stauffer, and Staff; Todd Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-101, U. H.

9:00 - 12:00 Psychiatry Conference; VA Hospital Annex, Fort Snelling.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:00 - 11:50 Urology Seminar; Manipulation, Ureteral Stone; Gershom Thompson, Mayo Clinic; E-101, U. H.

11:00 - 12:00 Anatomy Seminar; The Rise of Anatomy in the United States; Shirley F. Miller; 226 I. A.

II. SURGICAL TREATMENT OF PULMONIC STENOSIS

Richard L. Varco
K. Alvin Merendino
Fletcher A. Miller

Introduction

In the spring of 1945 Blalock and Taussig[6] described a technique for treating certain cyanotic cardiac cripples which immediately captured and held the interest of medical and lay persons. Their imaginative reasoning and the dramatic results they were able to secure have so consolidated this position, that a remarkably widespread audience now recognizes the phrase "blue baby", and associated its treatment with these pioneers. A local interest in this field of thoracic surgery was apparent soon after the initial publication. No patients, however, were operated upon until numerous simulated procedures had been successfully performed in dogs. It was September, 1946, therefore, before the first case was tried at the University of Minnesota Hospitals. Since that date fifty-three persons have been submitted to surgery for relief of congenital pulmonic circulatory insufficiency with arteriovenous mixing.

This phrase "congenital pulmonic circulatory insufficiency with arteriovenous mixing", although a more inclusive and accurately descriptive term for these cases, is too awkward for convenience. Since at least 75% of this entire group is composed of cases with Fallot's tetrad, by common usage that phrase, or merely congenital pulmonic stenosis, appears to be preferred in other reports of similar material. At least three of our patients were, however, instances of tricusped stenosis or atresia, cardiac anomalies likewise associated with pulmonic circulatory insufficiency, and in which the Blalock-Taussig procedure is quite capable of providing genuine relief from an incapacitating illness. Other developmental defects that might similarly be benefitted are truncus arteriosus with bronchial arteries and a rudimentary pulmonary artery; a single ventricle with a rudimentary outlet chamber in which the pulmonary atresia or stenosis associated with transposition of the great vessels and an interventricular septal defect.[33] The common denominator for this group is the presence of an adequate pulmonary blood flow, together with some arterio-venous mixing; and the treatment conceived by Blalock and Taussig is the establishment of an adequate blood flow to the lung. This is accomplished by shunting a systemic artery into the pulmonary circuit distal to the site of obstruction.

Historically, Fallot's[17] writings in 1888 were not the first to call attention to the clinical and anatomical findings in the syndrome which now carries his name. Morgagni,[21] Sandifort,[29] Hunter,[15] Farre,[18] Gintrac,[19] and Peacock[26] had all reported cases typical of this condition. His contribution, and the eponym, stem rather from the detailed reports of collected cases; his accurate featuring of the four anatomical defects present in the process; as well as the recognition and emphasis of the fact (which still obtains) that three out of four patients with cyanosis and clubbing of the fingers since infancy, are victims of this tetralogy of defects: pulmonary stenosis or atresia, dextroposition of the aorta, a high interventricular septal defect, right ventricular hypertrophy. The peripheral cyanosis in these cases is compounded, therefore, of the effects of the pulmonic stenosis as well as the mixed venous and oxygenated blood from right and left ventricles flowing through the septal defect and out of the overriding aorta.

The true incidence of the condition is difficult and perhaps impossible to ascertain. That one clinic alone could find nearly 1,000 cases in the space of $3\frac{1}{2}$ years is an imposing statistic. It can also be predicted from autopsy percentages that among the approximately 3,250,000 live births in 1946, more children with congenital heart disease amenable to present surgical procedures (patent ductus acteriosus, aortic coarctation, and pulmonic circulatory insufficiency) were born to survive to the present, than have ever been operated upon in all the surgical clinics of this country. The back log, therefore, from other years of unrecog-

nized, untreated cases, looms large.

Another question frequently posed by all concerned with the problem is: Why operate upon these cyanotic children? What is their outlook without surgical care? Unfortunately, the answer is not wholly available since prescience is supplied as a talent only slightly more often to the man of medicine, than to any other. White and Sprague[38] report the case of a musician with Fallot's tetrad who survived until shortly before his sixtieth birthday. This is far from the usual patient's lot. Those with pulmonic stenosis, as might be predicted, lived, on the average, longer than those with atresia. In one collected series of 85 cases with pulmonic stenosis, the mean survival was twelve years, with a maximum longevity of fifty-nine years plus; among thirty persons with pulmonic atresia the mean survival was five years and the maximum was thirty years.[1,37] This gloomy outlook was made additionally disagreeable by the life of semi-invalidism, often the lot of these cardiac cripples because of a morbid fear generated by bouts of asphyxia after incautious episodes of physical exertion. The physical benefits and emotional relief brought to these patients by the ingenious procedure of Blalock and Taussig, must be difficult to imagine for those of us more normally endowed. These sentiments, even as they are so regularly voiced by parents or relatives, can be almost embarrassingly fevent.

Diagnosis

The embryological, physiological, and clinical features in cases of Fallot's tetrad have been described clearly by numerous writers,[6-13,15,16,23,30,33-36] To the experienced cardiologist the latter are virtually diagnostic when typical. They will hence be dealt with but briefly here.

1. Cyanosis is usually a prominent symptom. Frequently this has been noted since shortly after birth, and is aggravated by any undue exertion (crying, feeding, walking, etc.) Its onset is occasionally delayed or less apparent by virtue of a patent ductus, only to manifest itself noticeably with closure of the latter. Suffusion of the conjunctival vessels and plethoric mucous membranes are common features.

2. Dyspnoea is in some degree usually present. In our experience it has not always been correlated progressively with the degree of cyanosis, contrary to the experience of others.[15] It should be recalled that in pure pulmonic stenosis, cyanosis is minimal or lacking, but dyspnoea can be quite severe and disabling. Some of the least cyanotic children have had a sharply reduced exercise tolerance. As a means of improving their dyspnoea a large percentage of the children have learned to occupy a peculiar squatting position which appears to provide a measure of relief not obtainable by other household means. This self taught assumption of an odd stance, which has been noted in children from widely separated areas of our country, remains a physiological enigma. Taussig was apparently first to make this observation.

3. Murmurs are usually present, and systolic in time relationship. Blalock has expressed the feeling that the absence of such a sign is unfavorable from the surgical outlook. The exact origin of this feeling of his has been difficult to trace, but appears to stem from the thought that those without such a rumbling are liable to have an atretic rather than a stenotic pulmonary artery, which it may be impossible to use in an anastomosis.

4. Digital clubbing, customarily present, is but a sign of chronic pulmonary disease.

5. Laboratory examinations. The roentgenographic and fluoroscopic findings in our cases have been clearly defined by Stauffer.[31] The sabot-shaped heart with a blunted, elevated apex, clear pulmonary artery, and association with small, non-pulsatile pulmonary arteries are text-book clear --, when present.[25,32] As the child adjusts to his handicap these manifestations can become so altered as to obscure the picture considerably. Lung fields assume a greater vascularization, the cardiac silhouette may be so modified too by

these changes that it also loses the classical appearance. These latter instances have predominated in our patients, with the consequent effect of increasing the roentgenologist's diagnostic difficulties. Electrokymography has proved of little additional diagnostic help.

The electrocardiogram is not pathognomonic and the usual findings are those of a right axis deviation.

Electroenceohalograms in our cases have frequently shown foci of localized brain pathology, but were not diagnostic.

Polycythemia is rather regularly present, as is also a decreased blood oxygen saturation because of a lowered oxygen content in the presence of an augmented oxygen capacity. In several cases studied here the oxygen saturation values have decreased sharply with exercise and only slowly risen toward normal after inhalations of 100% oxygen. No blood volume studies have been carried out in this series. Nelson et al[23] have found that it was regularly increased, principally through the enlargement of the red blood cell mass.

Angiocardiography and heart catheterization techniques have not been available in this hospital for routine diagnostic use in the work-up of this group of cases. Their use by others[3,4,5,12,32] has been associated with considerable success; at times has been invaluable; and, when combined with fluoroscopy may well represent the most accurate method now available for predicting the location and number of cardiac anomalies present.

6. Exploration, for diagnostic as well as therapeutic purposes, is available for a select group of cases in which it is impossible to decide by other means the need for a shunt procedure. Measurement at that time of the pulmonary arterial pressure will provide information confirming the wisdom or undesirability of making an anastomosis between the systemic and pulmonary systems. A discussion of the differential diagnosis of this lesion from simulating congenital cardiac anomalies is considered outside the province of this paper.

Surgical Care

1. Preoperative: All patients are admitted several days before the contemplated surgical procedure. 50,000 units of penicillin every three hours is routinely administered intra-muscularly to each patient not sensitive to the drug, for reduction of the bronchial bacterial flora and control of any existing bacteremia since many of these children have a chronic cough, a slightly elevated temperature, and pulmonary findings suggestive of a bronchitis. An attempt is made to alter the findings in nose and throat cultures, where pathogens are found. This has not always been possible and failure to do so has led to postponement, but not cancellation of the operation. Frequently these children have poor dental conditions, and badly infected tonsils. If at all practicable these are corrected before the major surgery. Close attention is regularly paid to the establishment and the maintenance of hydration in these children, particularly where a high hemocrit-hemoglobin value exists, for here the danger of spontaneous cerebral thrombosis is considerable at any time, and increases with any post-operative dessication of the patient. These patients are urged to drink liberally of fluids until a few hours before surgery.

2. Operative: For premedication these children are ordinarily given morphine sulphate and atropine. Heavy atropinization is the rule to secure vagus suppression and thereby reduce the incidence of cardiac irregularities. Other workers[9] have studied the grave significance of arrhythmias together with the strong inclination congenital hearts have to develop that status. A wholly effective therapy awaits further information and work. For those with low oxygen saturation values and markedly reduced exercise tolerances, rectal pentothal is administered in the ward and the patient then comes to the anesthesia room in a somnolent, unexcited, state. The problem of inserting the intratracheal tube without an episode dramatized by intensely cyanotic struggling, is thereby avoided. After trying oxygen-ether, cycloprane, and other mixtures, the pre-

ferred anesthetic at present appears to be Baird's solution[2] of pentothal-curare supplemented with a nitrous oxide-oxygen mixture through an intratracheal tube. In the smaller child (under four), or older if underdeveloped physically for their age, there arises a real danger of laryngeal edema, secondary to the intratracheal tube. In one such case this complication required a tracheotomy. The intravenous anesthetic is provided through a saphenous v in cannula or needle, which is regularly placed there for all cases. Through this the patient can and does receive throughout the operation a continuous drip of 5% glucose in distilled water, plasma, or whole blood. Five to ten cubic centimeters of 1% novocain solution at a time, and such other medications as prove necessary are injected as required. A cleanly patent, readily available, intravenous cannula, is mandatory in these cases.

The operations of Blalock-Taussig[6,10,11,12], or Potts-Smith,[27,28] Gibson propose to increase the total pulmonic blood flow. A thoracotomy is made, therefore, on the right or left hand side, a segment of pulmonary artery identified, isolated, opened and united with a suture anastomosis to the cut end of a systemic artery. The subclavian is the vessel of choice, but if necessary for technical reasons either the innominate or common carotid can be used. Approximately 25% of the cases have a right aortic arch which is readily recognizable during fluoroscopy when the patient swallows some barium.[31] The more detailed technical features of the procedure are beyond the scope and interest of this paper and have been reported elsewhere.[6,13,14,24] In the first cases (15) the proposal of Blalock[9] that the approach to the shunt be on the side opposite to the arch, was accepted. Technically, this is more difficult, particularly for the uninitiated, and so while acquiring some measure of know-how the next cases (27) were approached on the side of the arch. Laterally (13 cases) we have returned to Blalock's technique because it is possible to revise the architecture and fabricate a better vascular joint on the side where the subclavian artery takes off from the innominate. Throughout the course of the dissection novocain is used locally in liberal amounts while blocking the vagus nerve high in the thorax. In the event of any cardiac irregularity of a noticeable degree the patients are given additional amounts of atropine and/or novocain intravenously. All anastomoses are made with a running 000000 braided silk suture on an atraumatic Kalt needle developed for us by the Ethicon Suture Laboratory. The Potts-Smith-Gibson procedure has not been used in this series, but has been tried at some length in the laboratory.

3. Post-operative - The patient is returned to his bed under an oxygen mask and remains in an oxygen tent for 24 to 48 hours or until his pulse, respirations, and color are stable while he is outside this more favorable environment. Penicillin is continued in large doses for approximately ten days, even though the patient is afebrile, and longer if any sign of infection is apparent to avoid occupancy of the suture line by bacterial colonies. For the first few days hydration is largely by the intravenous route, relying on a close record of body weight, urine output, its specific gravity, and the child's clinical appearance, for the calculation of his needs for water and salt. Since all cases are closed without intrapleural drainage, each case must be checked by percussion and auscultation twice daily and by chest x-rays when indicated. An effusion is aspirated if estimated to be in excess of a few hundred cubic centimeters, whenever it might be the cause of an increased respiratory or pulse rate, if it fails to absorb, or is considered to be very bloody. Chylothorax has occurred, always on the left side, and each time has responded to repeated aspirations. Atelectasis, pneumonia, thrombophlebitis are some other of the more common postoperative complications to be looked for so that they may be treated at an early stage. Laryngeal edema, of varying severity, secondary to the intratracheal tube, has been noted in a number of patients. If detected in the incipient stage when stridor is minimal, elevation of the larynx above heart level by sitting the patient up, the use of adequate room humidification,

and the inhalation of oxygen-helium mixtures, will uniformly avert the necessity for more radical procedures.

Digitalization has not been carried out preoperatively and but one child has had it postoperatively for a tachycardia and a mild, transient, decompensation which cleared within a few days. The liver edge, pulmonary fields, and peripheral veins are regularly checked during bedside rounds in the recovery period, for any signs suggestive of cardiac failure.

The anticoagulants, heparin and dicumarol, have not been given before or during surgery. One patient received them after operation, but appeared to develop a thrombosis at the suture line despite this therapy.

During the recovery period two signs of favorable prognostic import have been recognized. Frequently, the child who is to have a good eventual result will show a triangular circum oro-nasal pallor. This appears within 24 to 36 hours as a quite definite area of blanching. Also, it has been noted that a cyanotic reddish hue will be apparent at the periphery of the nail pulp. This rim increases in breadth as the child continues to realize benefits from his operation. The suffusion of the eyeballs, labial and mucosal cyanosis are slower to disappear and harder to evaluate accurately from a memory which can easily be deceived by wishful thinking.

Complications

A listing of these has been made in tabular form. The most common has been pleural effusion, which probably develops to a limited degree in all instances by virtue of a traumatic pleuritis secondary to the surgery. All accumulations are closely checked by physical examination, changes in pulse and respiratory rates, and radiographs. A failure to recognize the seriousness and size of an effusion until respiratory decompensation had occurred, was the unfortunate cause of death in one child, who had merely been explored. When the volume is estimated to exceed about 200 cubic centimeters, to be causing distress, or continues to grow during the latter phases of convalescence, aspiration through a closed system is performed. At that time, an attempt is made to remove all obtainable fluids. With the exception of those cases with chylothorax two or three taps invariably sufficed; with these more prolonged treatment was required. In lymph effusions, attention must be closely directed to the matters of nutrition and fluid balance, for considerable quantities of protein, fat, and water can be siphoned off through repeated thoracenteses. The patients dietary intake of these requirements along with the daily weight was therefore charted. When required, supplements to the caloric and liquid needs were provided through parenteral routes. Thirty-one clinical cases of pleural effusion were noted, ten required aspirations. Three patients had chylothorax. In none of the entire series was an empyema found; and in all complete expansion of the lung eventually took place.

Thrombosis at the anastomotic site was suspected of having occurred whenever a continuous murmur failed to be audible during the convalescence and whenever significant clinical improvement did not occur. In all instances this complication was ascribed to technical difficulties, errors, or bungling. Six patients suffered this complication, and in two it was associated with a chylothorax.

A young girl developed what appeared clinically to be a cerebral thrombosis and hemiplegia. The recovery from this was distressingly slow and although considerable paresis remained at the time of discharge, hope was held that she would continue to improve. Two other patients developed severe anoxia during surgery, failed to regain consciousness despite a broadly patent anastomosis in one at least, and ultimately died. A third patient was lethargic and comatose for several days but finally recovered completely. To avoid this complication, the lung during surgery is maintained partially inflated at all times. The difficulty this causes the surgeon is acceptable and has seemed to reduce the incidence of

cerebral anoxia.

Some degree of Horner's syndrome was present in eight patients; in all it was a transient phenomena. Its appearance was more frequent when the incision was made into the left chest. The exact anatomical explanation of this is not readily apparent since on each side the sympathetic chain is closely approximated to the area under dissection.

Pneumonic consolidations or pneumonitis was recognized in three patients. In each the recovery was complete and gratifyingly prompt. Among the single instances of other major complications was, as noted earlier, a case with severe laryngeal obstruction requiring tracheotomy. This condition improved in a few days and permitted an early withdrawal of the tracheal cannula. No residual vocal sequelae existed. One child developed gangrene of the distal forearm after subclavian artery ligation. Vascular hypoplasia and anomalous brachial vessels were present in this child. Death subsequently took place and was due to encephalomalcia. This is the sole report of brachial vascular insufficiency we have been able to find in reports from other surgeons doing this operation. The likelihood of its recurrence seems low, therefore, and upon two occasions after an elapse of time, both subclavians have been divided in the same person. Digitalization was deemed necessary to control incipient post-operative heart failure and tachycardia in one child.

Minor wound infections and gastric distention have occurred periodically. The former has never been more than a cutaneous stitch abscess; the latter invariably responds to a short period of gastric siphonage. The late cosmetic appearance of the chest wall incision has been annoying. Keloid formation has been most unsightly at times, and maneuvers designed to control this have, to date, proven of questionable value. Thrombophlebitis about the cannula site, has been noted from time to time. It appears to be a local saphenous chemical phlebitis and responds to the simple measures of local heat, elevation, and immobilization.

Results

Several summarical tables have been prepared for the reporting of this material. To date 58 operations have been performed upon 53 people with eight deaths, a mortality of 15%. This includes the death of anyone who has ever been operated upon and subsequently died in or outside of the hospital. In one instance this was six weeks postoperative, in another it was after 16 months of good improvement from her anastomosis. The cause of death in the latter was a brain abscess and meningitis, to which these individuals are said to be particularly prone. The anastomotic sight was well healed and free of endarteritis. In the last 42 cases there have been two deaths. This has been compounded partially of good fortune, but most substantially by learning to recognize and attend to the small things which go to make up experience. This recognition and learning has been the individual and cooperative accomplishment of the several members making up the team - i.e., cardiologists, pediatricians, roentgenologist, anesthetists, floor nurses, and surgeons. The role of each is of equal importance to the success of the procedure. By so working together the hazards for the patient become more tolerable. Of the 45 out of 53 patients who survived surgery, 40 are improved and for the remaining five the possibility still exists of help through reoperation. Information about 32 persons of the entire group of 45 has been received recently. One feels normal, 28 are markedly improved, one moderately improved, two believe there has been no improvement. With knowledge gained from these first 50 odd cases, it should be possible to increase the present 75% change for survival and improvement which has just been listed. Of the eight deaths two had severe encephalomalacia; two had cardiac arrest during surgery; one had a delayed erosion of the pulmonary artery and bronchous by a foreign body; one massive pleural effusion was untreated; one died of status asthmaticus and hemothorax; one died of cerebral abscess and meningitis.

Discussion

Studies of pulmonary oxygenation were made using the Millikan-Smaller oximeter as modified by Hemingway. These have been reported and since then others have investigated this problem with the use of a similar instrument.[20, 22] The original theses of Blalock and Taussig that an increase in pulmonic blood flow was required in this group of cases and that the union of a systemic artery to the pulmonary circuit would accomplish this, have been confirmed. When the shunt was opened at the time of recording continuously the oxygen saturation, a prompt jump in this value was secured. It also became apparent, from the shortened saturation time after the inhalation of 100% oxygen, that an increase had occurred in that fraction of the total cardiac output sent through the pulmonary circuit. Too, the effect of widespread pulmonary atelectasis on the oximeter tracing, was clearly visible. These anoxic dips, if prolonged or repeated could readily initiate cerebral and cardiac disasters.

Pulmonary artery pressure measurements were made during surgery in most of the early cases and in all in whom any doubt existed as to the diagnosis. The artery is usually soft and easily collapsed, with pulsatile characteristics absent. Occasionally it has been hypoplastic, in none has it been absent, but in one it was retro-bronchial on the left side. How high the pressure can be and still be benefited by a shunt remains to be determined. In one case it was 35 centimeters of citrate, and that child had an excellent outcome.

Pre- and post-operative arterial oxygen saturation determinations are obtained with the blood drawn under basal or anesthetic conditions. Those with the best results from surgery have uniformly had an increase in this value. The correlation, however, has not been absolute, for in three cases the post surgical value was about the same as, or even lower than the earlier sample, whereas the children were clinically considerably benefited. We believe that this is partially explicable by the difficulty in securing basal conditions, together with the degree of aortic overriding, size of the septal defect, and its angle of deflection for the transventricular stream of blood.

Electroencephalographic tracings were taken on 18 tetralogy patients. These have been interpreted by Dr. James Bosma. Eleven were considered as normal; the remainder showed localized pathological sites. This incidence was felt to be abnormally high. The graphs were those of focal brain damage, but without a typical pattern. Its continued use as a laboratory screening device was recommended.

Unfortunately, an increased susceptibility to bacterial endarteritis because of the repetitious trauma of blood surging through the anastomotic site, appears predictable for the survivors. Taussig[36] has recently reported upon eight such instances. The success in controlling such an episode with current and future antibiotics, partially allays the grimness of this prospect.

Any increased work load imposed by the systemic-pulmonary arterial leak has not made itself apparent and recordable by a continuous postoperative cardiac deterioration. Any heart enlargements noted to date have been contained within physiological limits and clearly short of recognizable failure.

Psychometric studies have been carried through by Miss Audrey Arkola on many of these children. On the average, their intelligence has been normal or somewhat better. Many had capacities well above the mean. It has been difficult to quantitate any beneficial effects from surgery, but with the passage of time and the opportunity to re-evaluate them periodically, it is hoped that a sounder appraisal can be made. There can be no question of the Hyde-Jekyl transformation in the reaction to people and situations. As a group they are a conglomeration of behavior problems, since the invocation of any form of disciplinary action commonly precipitates a tantrum, cyanosis, and even coma. Their physical capacities limit much group

activity and so adjustments are poorer--the child is often shy. Above all hovers a fear of oxygen want and the knowledge of how easily it occurs. The privilege of observing these handicaps fall away is one of medicine's genuine pleasures. Invariably, after a successful operation the entire personality recuperates along with the body of the child.

Any discussion of experiences with this problem at the University of Minnesota Hospitals would be incomplete without specific reference to the contributions of Dr. John R. Paine. He initiated surgical interest locally, guided all of the technical training period in the animal laboratory, and did more than any other one of us to harmonize the participating departments into a workable team. His thoughts and ideas have been written into the final results.

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TABLE I

AGE AND SEX DISTRIBUTION

	Number	Extremes of Age	Average Age
Males	25	$1\frac{1}{2}$ - 25	8 years
Females	27	1 - 20	7 years

Relationship of Aortic Arch

	Number	Percentage	Male	Female
Right aortic arch	13	25	7	6
Left aortic arch	39	75	18	21

TABLE II

TYPES OF OPERATIONS

	Number of Operations	Results		
		Good	Poor	Deaths
A. Left Aortic Arch with Left Approach	20	18	1	2
1. End to side anastomosis subclavian to pulmonary artery	19	17	1	1
2. End to end anastomosis subclavian to superior branch pulmonary artery	0	0	0	0
3. Thoracotomy	1	0	0	1
B. Left Aortic Arch with Right Approach	21	13	4	4
1. End to side anastomosis subclavian to pulmonary artery	12	11	1	0
2. End to end anastomosis subclavian to superior branch pulmonary artery	2	0	0	2
3. End to side anastomosis subclavian to superior branch pulmonary artery	1	1	0	0
4. End to side anastomosis innominate to pulmonary artery	2	1	0	1
5. Thoracotomy	4	0	3	1
C. Right Aortic Arch with Right Approach	6	3	2	1
1. End to side anastomosis subclavian to pulmonary artery	4	3	0	1
2. End to end anastomosis subclavian to superior branch pulmonary artery	1	0	1	0
3. Thoracotomies	1	0	1	0
D. Right Aortic Arch with Left Approach	11	5	4	1
1. End to side anastomosis subclavian to pulmonary artery	8	5	2	1
2. End to side anastomosis innominate to pulmonary artery	1	1	0	0
3. Thoracotomy	2	0	2	0
TOTAL	58	40	11	8

TABLE III

SUMMARY TYPES OF OPERATIONS

Type of Operation	Number	Good	Poor	Deaths
End to side subclavian to pulmonary artery	43	37	4	2
End to end subclavian to superior branch pulmonary artery	3			3
End to side innominate artery to pulmonary artery	3	2		1
Thoracotomy	8		6	2
End to end subclavian to pulmonary artery	1	1	0	0
TOTAL	58	40	10	8

TABLE IV

RESULTS AND MORTALITY

Total patients	53
Total operations	58
Total deaths*	8
A. Vascular anastomoses completed	50
Patients improved	40
Patients unimproved but living	5
Died**	6
B. Thoracotomies with exploration with anastomosis	8
Died	2
C. Re-operations	5
Patients improved	4
Patients slightly improved but no anastomosis made	1
Died	0
D. Total all patients	
Improved	75%
Unimproved	10%
Mortality	15%

* One case was markedly improved by operation, but died sixteen months post-operatively of meningitis and a brain abscess. The site of the vascular anastomosis was functioning, well healed, and without vegetation.

** There have been two deaths in the last forty-two cases.

TABLE V

COMPLICATIONS

		End Results		
	Number	Good	Poor	Died
Pleural effusion	31	26	1	4
Chylothorax	3	1	2	0
Thrombosis of anastomosis	6	0	5	1
Horner's syndrome	8	6	1	1
Pneumonitis	3	3	0	0
TOTAL	50	38	9	6

In addition to the above there has been one case each of the following: laryngeal edema requiring tracheotomy, severe abdominal pain, high pulse suggestive failure requiring digitalization, and gangrene of arm.

TABLE VI

POOR RESULTS

Patient	Age	Operation	Complications	Cause of Poor Results
1.	6	End to side anastomosis left subclavian to pulmonary artery - right arch	Chylothorax	Probable thrombosis of anastomosis
2.	12	Bilateral thoracotomies - right arch		Vessels too short for anastomosis
3.	7	End to side anastomosis right subclavian to pulmonary artery - left arch	Right hemiplegia	Probable thrombosis at site of anastomosis
4.	8	End to side anastomosis left subclavian to pulmonary artery - left arch	Chylothorax	Probable thrombosis at site of anastomosis
5.	6½	End to end anastomosis right subclavian artery to upper branch right pulmonary artery - right arch	Pleural effusion, Horner-s syndrome	Probable thrombosis at site of anastomosis

TABLE VII

RE-OPERATIONS

Name	Age Original Operation	Aortic Arch	Original Operation	Reason for Re-operation	Time in Months Between Operations	End Result
1.	11	Left	Right thoracotomy	Vessels too short for anastomosis	7	Good
2.	12	Left	Right thoracotomy	Vessels too short for anastomosis	$2\frac{1}{2}$	Good
3.	13	Right	Left thoracotomy	Vessels too short for anastomosis	3	Good
4. .*	12	Right	Left thoracotomy	Vessels too short for anastomosis	4	Poor (vessels too short on rt. side also)
5.	3	Right	End to side anastomosis left subclavian to pulmonary artery	Anastomosis thrombosed	17	Good

*In spite of inability to complete anastomosis, there has been some subjective improvement.

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TABLE VIII

ANALYSIS OF SURGICAL DEATHS

Name	Age in Years	Operative Procedure	Post-op. Interval	Complications	Autopsy
1.	8	Right thoracotomy	4 days	Massive pleural effusion	1. Atresia tricuspid and pulmonary valves. 2. Interatrial septal defect. 3. Rudimentary right ventricle with no outlets. 4. Massive right pleural effusion. 5. Atelectasis - right lung.

TABLE VIII (Cont.)

Name	Age in Years	Operative Procedure	Post-op. Interval	Complications	Autopsy
2.	1½	Left thoracotomy with vessel dissection	0	Repeated cardiac standstill during operation	1. Pulmonary atresia 2. Interatrial septal defect 3. Tricuspid stenosis 4. Right ventricle hypertrophy.
3.	1	End to side anastomosis Right subclavian to pulmonary artery	3 days	Anoxia during	1. Tetralogy of Fallot 2. Thrombosed anastomosis 3. Arterial occlusion and gangrene of right lower arm 4. Encephalomalcia.
4.	10	End to side anastomosis Right subclavian to pulmonary artery	4½ wks.	Arterial bronchial fistula	1. Tetralogy of Fallot 2. Patent anastomosis 3. Erosion and rupture left pulmonary artery with resultant erosion into left main bronchus. Due to retained cotton pledget.
5.	10	Right end to side anastomosis innominate to pulmonary artery	3 days	Anoxia during operation	1. Tetralogy of Fallot 2. Patent anastomosis 3. Pulmonary edema 4. Marked encephalomalacia.
6.	25	End to end anastomosis Right subclavian to superior branch pulmonary artery	1 day	Massive hemothorax status asthmaticus	1. Tetralogy of Fallot 2. Patent anastomosis 3. Bilateral hemothorax 4. Pulmonary congestion and edema, emphysema 5. Venous infarction of brain.
7.	5	End to end anastomosis Right subclavian to superior branch pulmonary artery	0	Cardiac standstill during operation	No autopsy
8.	20	End to side anastomosis left artery to pulmonary artery	16 mo.	None during surgery	1. Tetralogy of Fallot 2. Patent anastomosis with no end arteritis 3. Brain abscess 4. Meningitis.

TABLE IX

SUBJECTIVE RESPONSE AS JUDGED BY PARENTS

	Number of Replies	Apparently Normal	Markedly Improved	Moderate Improvement	Unimproved
General Condition	32	1	28	1	2
Cyanosis	31	4	17	8	2
Weight	24	0	14	8	2
Colds	28	0	20	3	5
Behavior	29	0	23	3	3
Exercise Tolerance	32	5	22	3	2

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TABLE X

PULMONARY ARTERY PRESSURES (in CM, H_2O)

Extremes 5-35 Average 20.7

Pressure in Cm.citrate	Number of Cases	Results Good	Poor	Death
Under 15	8	6	1	1
15-25	14	10	1	3
25-30	2	2	0	0
30-35	4	4	0	0

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TABLE XI

FORM LETTER

Dear Mr. and Mrs. --

We are seeking to learn how your child has fared from (his-her) operation for relief of (his-her) heart condition. Your interest and help in filling out the questionnaire will be of real benefit to us in future candidates for this operation. We would like to know how - - - - - - - - - is progressing following the operation which was performed in this hospital to treat (his-her) heart condition. Would you please help us by completing the accompanying form and returning it to us as quickly as possible?

1. The child is:
   ___A. Completely cured following the operation.
   ___B. Markedly improved " " "

___C. Slightly improved following the operation.
___D. The same as before the operation.
___E. Worse than before the operation.

2. The blue discoloration which was present before the operation:
___A. Has completely disappeared.
___B. " " " , except during exertion.
___C. Is still present to a slight extent at all times but has decreased a great deal since the operation.
___D. Is only slightly less than before the operation.
___E. Is the same as before the operation.
___F. Is worse than before the operation.

3. The child:
___A. Can walk quickly up one ordinary flight of stairs without stopping and without any ill effects.
___B. Can walk quickly up one ordinary flight of stairs without stopping but gets very tired and short of breath.
___C. Can not walk up one flight of stairs without stopping.

4. Approximately how many blocks can the child walk without shortness of breath or other signs of distress?

How much better or worse is this than before operation?

5. Is the child able to keep up with the other children his age:
___A. Completely.
___B. Must stop and rest a little more often than the others.
___C. Must stop and rest much oftener than the others.
___D. Must markedly limit his activities.
___E. Is not able to play with the other children at all.
___F. Is completely bedridden.

6. How does number 5 compare with his condition before the operation:
___A. Better
___B. Only slightly better
___C. The same
___D. Worse.

7. Has there been any change in the child's behavior and disposition?____
What have these changes been (if present)?

8. How much weight has the child gained since the operation?

9. How is the child's appetite? ___A. Good, ___B. Fair, ___C. Poor.

10. How has the child fared in regard to colds?
___A. Very few colds since operation.
___B. Number of colds less since operation.
___C. No improvement in number of colds since operation.
___D. More colds than before operation.

11. Knowing what you know now, would you recommend the operation for other children in a similar condition as yours was? ___Yes, ___No.

12. We would like a brief statement from you summarizing your reaction to the operation and the results it obtained in your child.

Thank you very much.

References

1. Abbot, M.E.
Atlas of Congenital Heart Disease.
New York Heart Assoc., '36.
2. Baird, Joseph
Pentothal Curare Mixture.
Anes. 8:75-79, '47.
3. Bing, R.J., Vandam, L.D., and Gray, F.D.
Physiological Studies in Congenital Heart Disease.
I Proc.Bull.Johns Hopkins Hospital, 80:107-120, '47.
4. Bing, R. J., Vandam, L.D., and Gray, F.D.
Physiological Studies in Congenital Heart Disease. II. Results of Preoperative Studies in Patients with' Tetralogy of Fallot.
Bull.Johns Hopkins Hosp. 80:121-141, '47.
5. Bing, R.J., Vandam, L.D., and Gray, F.D.
Physiological Studies in Congenital Heart Disease. III. Results Obtained in Five Cases of Eisenmengers Complex.
Bull. Johns Hopkins Hosp. 80:323-347, '47.
6. Blalock, A. and Taussig, H. B.
The Surgical Treatment of Malformations of the Heart in which there is Pulmonary Stenosis or Pulmonary Atresia.
J.A.M.A. 128:189-202, '45.
7. Blalock A.
Physiopathology and Surgical Treatment of Congenital Cardiovascular Defects.
Harvey Lecture, Bull. N.Y. Acad.Med., 22:57-80, '46.
8. Blalock, A.
Effects of an Artificial Ductus Arteriosus on Experimental Cyanosis and Anoxemia.
Arch.Surg. 52:247, '46.
9. Blalock, A.
The Surgical Treatment of Congenital Pulmonic Stenosis.
Ann.Surg.124:879-887, '46.
10. Blalock, A.
Use of Shunt or By-Pass Operations in Treatment of Certain Circulatory Disorders, Including Portal Hypertension and Pulmonic Stenosis.
Ann.Surg. 125:129-141, '47.
11. Blalock, A.
The Technique of Creation of an Artificial Ductus Arteriosus in the Treatment of Pulmonic Stenosis.
J.Thoracic Surg. 16:244-255, '47.
12. Blalock, A.
Surgical Procedures Employed and Anatomical Variations Encountered in the Treatment of Congenital Pulmonic Stenosis.
Surg.Gyn.& Obst. 87:385-409, '48.
13. Burchell, H. B.
Basis of Cyanosis in Tetralogy of Fallot.
Proc. Staff Meet. of Mayo Clin., 22:162-165, '47.
14. Clagett, O. T.
Surgical Considerations in Tetralogy of Fallot.
Proc.Staff.Meet., Mayo Clin. 22:180-182, '47.
15. Dry, T. J.
Classic Clinical Features of Tetralogy of Fallot.
Proc.Staff Meet., Mayo Clin. 22: 173-174, '47.
16. Edwards, J. E., Bulbulian, A. H., Rogers, H. M.
Pathologic and Embryologic Considerations in Tetralogy of Fallot.
Proc.Staff Meet., Mayo Clin. 22: 166-172, '47.
17. Fallot, A.
Contribution a' l'anatomie Pathologigue de la Maladie Bleue,
Marseille Med. 25:77-93, 138-158, 207-223, 270-286, 341-354, 403-420, 1888.
18. Farre, J. R.
Pathological Researches. Essay I. On Malformations of the Human Heart. Illustrated by Numerous Cases, London, 1814.
19. Gintrac, E.
Observations et recherches sur la cyanose, ou maladie bleue.
Paris, 1824.
20. Gullickson, G., Elam, J. O., Hammond, M., Paine, J. R. and Varco, R. L.
Oxygenation Studies in Congenital Pulmonary Stenosis.
Am.Heart J. 35:940-947, '48.
21. Morgagni, G. B.
DeSedibus, et Causis Morborum per anatomen indigatis, libri quinque.
Venice, 1761.

22. Montgomery, G. E., Geraci, J. E., Parker, R. L., and Wood, E. H.
Arterial Oxygen Saturation in Cyanotic Types of Congenital Heart Disease.
Proc. Staff Meet., Mayo Clin., 23: 169-175, '48.

23. Nelson, W., Mayerson, H. S., Clark, J. H. and Lyons, C.
Studies of Blood Volume in the Tetralogy of Fallot and in other Types of Congenital Heart Disease.
J.C.I. 26:860-868, '47.

24. Paine, J. R., and Varco, R. L.
Experiences in the Surgical Treatment of Pulmonic Stenosis.
Surgery 24:355-370, '48.

25. Papp, C.
Le Diagnostic Radiologigne de la Tëtrade De Fallot.
Arch.d.mal.du coeur 24:249-258, '31.

26. Peacock, T. B.
Cases of Malformation of the Heart.
London, 1864.

27. Potts, W. J., Smith, S. and Gibson, S.
Anastomosis of the Aorta to a Pulmonary Artery in Certain Types of Congenital Heart Disease.
J.A.M.A. 132:627-632, '46.

28. Potts, W. J.
Aortic-Pulmonary Anastomosis for Pulmonary Stenosis.
J.Thoracic Surg. 17:223-231, '48.

29. Sandifort, E.
De Rarissmo Cordis Vitio.
Obs.Anat.Pathol., Surguni Batavorum, 1777.

30. Spitzer, A.
Über den Bauplan des normalen und missbildeten Herzens.
Versuch einer phylogenetischen Theorie, Virchows Arch.f.path.Anat. 243:81-272, '23.

31. Stauffer, Herbert M.
Roentgenology of Congenital Heart Disease.
Staff Bull. University of Minnesota Hospitals, June 11, 1948, Vol.XIX, No. 30, 486-506.

32. Sussman, M. L.
The Cardiovascular System,
Clinical Radiol. pp.3-106, Phila., F.A.Davis Co., '47.

33. Taussig, H. G.
Congenital Malformations of the Heart.
N.Y.Commonwealth Fund., '47.
E.L.Hildreth and Co. Inc., '47.

34. Taussig, H. G. and Blalock, A.
Pulmonary Circulation, Cyanosis, Polycythemia.
Am.Heart J. 33:413-419, '47.

35. Taussig, H. G.
Diagnosis of Tetralogy of Fallot and Indications for Operation.
J.Thoracic Surg. 16:241-243, '47.

36. Taussig, H. G.
Analysis of Malformation of the Heart Amenable to a Blalock-Taussig Operation.
Am.Heart J. 36:321-333, '48.

37. White, Paul D.
Heart Disease.
William Wilkins Co. '44.

38. White, P. D. and Sprague, H. B.
The Tetralogy of Fallot: Report of a Case in a Noted Musician, Who Lived to His Sixtieth Year.
J.A.M.A. 92:787-791, '29.

39. Ziegler, R. F.
The Cardiac Mechanism during Anesthesia and Operation in Patients with Congenital Heart Disease and Cyanosis.
Bull. Johns Hopkins Hospital 83: 237-274, '48.

III. MEDICAL SCHOOL NEWS

Lowered Maternal Mortality Revealed in Litzenberg Lecture

Alumni and friends of the University Medical School attended the first of what is hoped to be a series of lectures in honor of Jennings Crawford Litzenberg in the Medical Science Building on Friday, October 22.

Dr. John L. McKelvey, Professor of Obstetrics and Gynecology at the University of Minnesota, was the speaker at the annual meeting of the Minnesota Medical Foundation.

Dr. Everett D. Plass, University of Iowa Medical School, who was scheduled to speak, was unable to attend because of a critical illness in his family. The lecture given by Dr. McKelvey was the first of what is hoped to be a series of lectures honoring the name of Dr. Jennings Crawford Litzenberg, former Professor of Obstetrics and Gynecology at the University of Minnesota.

In view of the particular interest of Dr. Litzenberg during his life-time, the subject chosen was "The Changing Maternal Mortality in the United States and Especially in Minnesota." Old statistical material was shown in order to demonstrate that as recently as 1920 the United States maternity mortality rate was 8 deaths per 1,000 live births, whereas in Minnesota the figure was 6.2. These were among the highest figures reported in the world. Since that time, there has been a steady decline in both the national and state rates for reasons that appear to be clearly multiple.

Dr. McKelvey stated that the most important factor lay in the gradual infiltration of medical graduates with somewhat better training due to better facilities in the Medical Schools of the country.

The introduction of new techniques in obstetrics seems to have speeded this improvement so that following 1930 the decrease in mortality was much more rapid.

A detailed study in Minnesota of individual cases dying of puerperal causes in 1941 and 1942 showed that, even with a liberal interpretation, about 75 per cent of maternal deaths were preventable. Since the Minnesota rate that year was 2 per 1,000 live births, it was suggested that a irreducible minimum of 0.5 per 1,000 live births was the most that could possibly be achieved.

The following table shows the mortality experience for the United States through 1945 and for the state of Minnesota through 1947:

Year	U.S.	Minn.
1920	8.0	6.2
1939	4.0	2.7
1940	3.8	2.2
1941	3.2	2.0
1942	2.6	1.6
1943	2.5	1.5
1944	2.3	1.3
1945	2.1	1.4
1946		1.0
1947		0.6

It should be noted that the 1947 rate has practically reached that point which seemed to be irreducible.

It is obvious that this excellent record reports an extremely high level of efficiency in maternal care in Minnesota. Indeed, no comparable efficiency has ever been reported elsewhere in the world. Since the vast majority of women are cared for by the general practitioner, it is evident that to this group belongs the chief credit for such remarkable maternal safety.

Dr. McKelvey pointed out that Dr. Litzenberg's guidance and the teaching of obstetrics has been a major factor in achieving this end. He said that this was the most suitable memorial of all and one that "Litz" would cherish.

Letters from Colleagues of Dr. Litzenberg

"It pleases me no end to send you a little note apropos of 'Litz,' and to make it quite personal and not too lugubrious, permit me to put it in this form: Leaving a farm in southern Minnesota, Olmstead County, in the fall of 1898, I had my first introduction to the gentleman who, in the fullness of time, was to introduce to Minnesota and the world so many of its future exemplary citizens. 'Litz' had the way of looking extremely serious, but concealing a subtlety of humor and understanding that lent superb confidence to all prospective parents. What more could an obstetrician ask for in the way of personality equipment? However, it wasn't in his role of an obstetrician that I met him, but rather as a first or second assistant of the late 'Doc' Cook over at the old gymnasium. Here I was, hard as nails and fresh from the plow and cornfield. In those days, farm boys never ran unless there was something to chase -- usually some wandering livestock.

"Nevertheless, I was made to join the herd of faltering freshmen in scanty attire, told to go down to the running track in the old gymnasium and do the essential number of laps at a sharp pace, the while 'Doc' Cook's first assistant stood by and shouted, 'Hey, there, draw in your stomach.' You may judge from this how prophetic this abjuration connoted the major interest of this budding medical student who, possibly unknown to himself, was centering his anatomical interests upon protuberant abdomens -- the sign both of fertility and nutritional adequacy. God loved 'Litz,' and all through his wholesome and productive life, all God's followers found it easy to do likewise."

"Dr. Jennings Litzenberg was always known as 'Litz' among his many friends, and he liked to have us call him 'Litz.' This is evidence of his kindly democratic spirit. He was deeply interested in his profession and in the welfare of his patients. No man on the medical faculty devoted as much attention to his students as did Litz.' He made his teaching interesting, simple and practical; and left a lasting and favorable impression on almost every student. 'Litz' leaves us a memory of a kindly, generous spirit devoted to his profession and to the welfare of the community."

-- E. T. Bell, M.D.

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Phi Delta Epsilon Lecture

Dr. Franklin Hollander, Clinical Physiologist and Head of the Gastroenterological Research Division of the Mount Sinai Hospital, New York City, will deliver the second annual Phi Delta Epsilon lecture in the Medical Sciences Amphitheater on Wednesday, November 3, at 8:00 p.m. on "Studies in Gastric Secretion." Dr. Hollander has worked extensively in the field of gastric physiology and has pioneered in the studies of acid formation. He will be introduced by Dr. Owen Wangensteen.

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Kellogg Foundation Lectures

Dr. L. O. Underdahl	Current Concepts of Diabetes Mellitus and Hypoglycemia	Monday, November 1, 3:00 p.m., Rm. 229, Center for Continuation Study.
Dr. Franklin Hollander	Gastrointestinal Physiology	Tuesday, November 2, 2:00 p.m., Eustis Amphitheater, U. H.