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Right Heart Catheterization

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INDEX

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I. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL

CALENDAR OF EVENTS

November 10 - November 15, 1947

No. 176

Monday, November 10

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.

9:15 - 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 Physical Medicine Conference; Virus Encephalitides and the Anaphylactic Reaction; Berry Campbell; E-101, U.H.

11:00 - 11:50 Roentgenology-Medicine Conference; Staff; Veterans' Hospital.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and D. State; Eustis Amphitheater, U.H.

12:00 - 12:50 Physiology Seminar; The Use of the Electromyogram in Evaluating Muscle Contraction; G. N. Loofbourrow; 214 M.H.

12:15 - 1:20 Pediatrics Seminar; Self Selection of Feeding of Infants and Children; Carl G. Goebel; 6th Floor Seminar Room, U.H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; M-435, U.H.

12:30 - 1:20 Pathology Seminar; Perthes' Disease; D. J. Nelson; 104 I.A.

12:30 - 1:50 Surgery Grand Rounds; A. A. Zierold, Clarence Dennis and Staff; Minneapolis General Hospital.

4:00 - 5:00 School of Public Health Seminar; Healthful Living in Movies; Dean H. S. Diehl; 113 MeS.

8:00 p.m. Clinical Research Club; Lane Williams and Howard E. Horns; Eustis Amphitheater, U.H.

Tuesday, November 11

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

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

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and Nathanial Lufkin; 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. L. Visscher; Eustis Amphitheater, U.H.

5:00 - 5:50 Roentgenology Diagnosis Conference; D. L. Fink and Staff of Veterans' Hospital; M-515, U.H.

Wednesday, November 12

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

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

11:00 - 11:50 Pathology-Medicine-Surgery Conference; Mesenteric Infarction;E.T. Bell, O.H. Wangensteen, C.J. Watson, & Staff; Todd Amphitheater, U.H.

4:00 - 5:00 Infectious Disease Routes, Todd Amphitheater, General Hospital, Veterans' Hospital.

Thursday, November 13

8:15 - 9:00 Roentgenology-Surgical-Pathology Conference; Walter Walker and H. M. Stauffer; M-515, 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; Todd Amphitheater, 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 D. State; Eustis Amphitheater, U.H.

12:00 - 12:50 Physiological Chemistry Seminar; B-Glucuronidase; Its Relation to the Octio Estrogenic Hormones; George Moore; 214 M.H.

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

1:30 - 3:00 Pediatric Psychiatric Rounds; Reynold Jensen; 6th Floor West Wing, U.H.

4:00 - 4:50 Bacteriology Seminar; Infectious Hepatitis; H. Dekruif; 214, M.H.

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

5:00 - 5:50 Roentgenology Seminar; Conference on Thoracic Surgery Cases; Thomas J. Kinsella, Richard Varco, Nathan Jensen and Robert Utendorfer; M-515, U.H.

7:00 - 8:00 Urology-Roentgenology Conference; H. M. Stauffer and George Eaves; M-515, U.H.

Friday, November 14

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

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustis Amphitheater, 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, Peroral Endoscopy; G. Eyjolfsson; New Powell Hall Amphitheater.

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.

4:00 - 5:00 Pediatric-Surgery Conference; I. McQuarrie, O. H. Wangensteen and Staffs; 6th Floor, Child Psychiatry Clinic.

5:30 - 6:20 Surgery Literature Conference; Clarence Dennis and Staff; Minneapolis General Hospital.

Saturday, November 15

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

8:30 - 10:00 Psychiatry and Neurology Grand Rounds; Staff; Veterans' Hospital.

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

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-515, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; M-515, U.H.

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

11:00 - 12:20 Anatomy Seminar; Some Histochemical Approaches to Morphological Problems; J. F. Hartmann; Hodgkin's Disease of the Bone Marrow; R. Dorothy Sundberg; 226 I.A.

II. RIGHT HEART CATHETERIZATION

Craig Borden
Richard V. Ebert

CONTENTS

(I) History

Werner Forssmann, a roentgenologist, was the first to report catheterization of the right heart in 1929[1]. With the help of a surgical assistant who cut down on the median basilic vein he passed a ureteral catheter on himself into the superior vena cava and right atrium. In 1930, Klein was successful in 11 out of 18 attempts to catheterize the right atrium and obtained blood samples in 3 instances[2]. From 1931 to 1936 there were 9 reports in the foreign literature, all but one[7] dealing with the use of a catheter to inject radiopaque dyes for the visualization of the heart and pulmonary vessels[3,4,5,6,8,9,11,12]. During this period the only reference in the American literature on the procedure is by Conte and Costa[10].

The technique was perfected in 1941 by Cournand and Ranges at Bellvue Hospital[13]. They applied the technique to the measurement of cardiac output and demonstrated the safety and practicability of leaving the catheter in place long enough to complete physiological studies. The mean auricular pressure was recorded with a saline manometer. During the war years Cournand and his group applied the technique extensively to the study of the circulatory dynamics of traumatic shock in man[34]. In 1944, the use of an optical recording manometer to record the pressure curves in the right heart was instituted[14] and later a double lumen catheter was developed for recording pressures in the auricle and ventricle simultaneously[15]. Since then the pulmonary artery has been catheterized with safety and ease, and electrical devices for recording pressures have become available. A wired catheter has been used as an endocardial exploring electrode in electrocardiographic studies[16,17].

During the latter war years Sharpey - Schafer and McMichael applied the method to studies on congestive heart failure. Warren, Stead, and Merrill used the method in various physiological studies on the circulation and extended the use of the technique to the catheterization of the renal vein[18]. Bradley in 1945 adapted the technique to the measurement of hepatic blood flow by catheterization of the hepatic vein[19]. In the past year, Dexter and Burwell[30], and Bing and his associates[77] have brilliantly applied the technique to the diagnosis of congenital heart disease. Cournand[50] has recently published important studies on the dynamics of the pulmonary circulation, a topic on which we have heretofore had relatively little factual data.

By 1945 Cournand and his group had done over 1200 catheterizations of the heart without a fatality or serious complication. In the same year, a fatality was reported from Baltimore following the injection of diodrast through the catheter[20]. The patient had an aneurysm of the pulmonary artery and had received an injection of diodrast ten days previously, so that sensitization to the compound was probably more implicated than the catheterization per se. Ameuille, et al[12], 1936, injected dye through the catheter in over 60 cases without mishap. We have adhered strictly to Cournand's recommendations and have refrained from injecting any substance except saline through the catheter. Apparently there is a danger of air embolism in certain congenital defects of the heart with a right to left shunt. Although we use every precaution to exclude air from the drip system, it is our impression that small amounts of air entering the right heart are relatively innocuous if no shunt from right to left is present.

Heart catheterization opens new vistas in the study of the circulation in the intact man, and gives to the clinical investigator a sound physiological method to apply to that study. It requires a trained team of three or four and expensive equipment. However, because of its usefulness in the study of the heart's dynamic function in health and disease, and its usefulness in the accurate diagnosis of certain congenital defects amenable to surgery, it has become a routine procedure in larger medical centers. McMichael[78] considers heart catheterization as the greatest single advance in cardiology since Einthoven's string galvanometer.

(II) Methodology

A. Technique of Catheterization

We have followed closely the technique of Cournand and Ranges[13]. The patient is made confortable on the fluoroscopic table by an air mattress and soft pillow. No premedication is used as this seems to bother our patients more than the procedure. The median basilic vein of either arm can be used, but the cephalic vein is not suitable because of its variable course and right angled junction with the axillary vein. In selecting the right or left arm it is advisable to palpate the course of the basilic and axillary vein since thrombosis of the axillary vein is not uncommon in hospital patients who have had frequent venipunctures. We have observed complete obliteration of the lumen of the basilic vein following a single venipuncture. If catheterization is anticipated, no venipunctures should be done on the arm to be employed.

Under strict surgical asepsis and local novacaine anaesthesia, a small transverse skin incision is made in the anticubital area, the median basilic vein is isolated by blunt disection, and a silk loop is twisted distally on the vein and held with a hemostat. The catheter is inserted through a small "fishmouth" incision in the vein. Enough spasm occurs to prevent retrograde bleeding so that a proximal silk loop is usually not necessary although one is usually put in place as a precautionary measure. Since novacaine interfers with the color reaction of para-amino-hippurate, metycaine is used as an anaesthetic if renal blood flow is to be determined. In either case adrenalin is omitted from the anaesthetic agent to prevent local venospasm. Frequent injections of novacaine are

given throughout the procedure to prevent spasm and keep the patient comfortable, since the only attendent discomfort is at the site of the local incision. Spasm which is manifested by resistence to passing the catheter and some aching and discomfort in the arm or axilla has not proved troublesome and usually stops spontaneously.

The catheter is a 100 cm. no. 8-F or 9-F ureteral-type catheter which is made of a radio-opaque plastic and was designed by the U.S. Instrument and Catheter Company of Glen Falls, New York. The last 3 inches of the tip forms a slight curve. It is sterilized by autoclaving at the same pressure and for the same time as rubber gloves. During autoclaving the shape and angle of the catheter is maintained by tying it in place with cotton tape on a plywood form. Dexter has recommended heat hardening the catheter[30] but we have not found this necessary. During the procedure of catheterization the lumen of the catheter is kept patent by a slow saline drip of the standard type for intravenous use which is connected to the catheter by a three-way stopcock. To prevent clotting we add 2 cc. (20 mg) of heparin to 1000 cc. of physiological saline. Disposable tubing is used to avoid pyrogenic reactions. Care in filling the tubing eliminates air bubbles. The catheter is moistened externally with saline and inserted rapidly into the vein up to the axilla. Then, under direct fluoroscopic vision, it is passed into the subclavian vein, down the innominate vein into the inferior vena cava and right atrium.

Occasionally some difficulty is encountered at the junction of the subclavian with the innominate vein and the catheter enters the internal jugular vein or its progress is blocked altogether. By rotating the tip inferiorly, abducting the arm at the shoulder, or bending the neck to the right the difficulty is usually overcome. From the atrium we have also entered the coronary sinus and vein, azygos vein, the inferior vena cava and hepatic vein. Sosman published reproductions of x-ray films showing the catheter in these various positions[21]. On entering the atrium from the right arm the tip of the catheter points to the right and must be rotated to the left to pass through the tricuspid valve into the ventricle. The rotation should be made anteriorly rather than posteriorly to avoid the coronary sinus and inferior vena cava. The tip is ideally placed when entering the atrium from the left arm. Cournand has stressed the occurrence of extrasystoles when the catheter is in contact with the tricuspid valve. We have noted extrasystoles occurring particularly when the catheter tip touches the intraventricular septum. Some patients are aware of skipped beats but no serious disturbances of rhythm have occurred. Once in the ventricle, by rotating slightly anteriorly and allowing the catheter to follow the stream of blood through the outflow tract the right pulmonary artery is entered with ease. The only instance to date in which we have experienced difficulty in entering the pulmonary artery was in a case of pulmonic stenosis. It is usually difficult to rotate the catheter again to the left and enter the left pulmonary artery. The pulmonary artery can be entered to the point where withdrawal of blood yields capillary blood with an arterial oxygen content[30]. Dexter found in dogs that a main branch of the pulmonary artery had to be occluded for an hour to produce pulmonary infarction[30].

Cournand has left the catheter in place for as long as 24 hours without harm. In dogs purposely traumatized by repeated catheterizations and then sacrificed no damage to the heart, large veins, or endocardial surfaces was found[30]. In patients who have had cardiac catheterization and later died from unassociated disease, no damage to the heart valves was found. Although the likelihood of inducing a bacterial endocarditis is a remote possibility, we have routinely given 50,000 units of penicillin one hour before the procedure and every three hours for forty-eight hours following it.

When the catheter is withdrawn, Cournand recommends that the vein be left untied, the skin approximated by a tape "butterfly", and bleeding controlled with a pressure bandage. A local thrombus

forms and the vein recanalizes. We have frequently encountered brisk retrograde bleeding requiring a tie around the vein. We routinely suture the skin incision with silk.

For obtaining arterial blood samples and recording arterial blood pressure directly, a Cournand needle is placed in the femoral artery. The Cournand needle is a No. 19 needle with a blunt round tip and large semi-circular bevel. A slightly longer second needle with sharp cutting edge fits into the first. After the artery has been entered, the cutting needle is withdrawn and replaced by a stylet. The needle can be taped in place while repeated blood samples are withdrawn or pressure curves recorded. We have experienced some difficulty in keeping the needle in place and with clotting. The brachial artery and radial artery may also be used.

B. Determination of Cardiac Output

1. In 1870 Fick[23] predicted that the blood flow to the body could be determined if it was known how much of a given substance was removed from the blood per unit of time and if the difference in the arterial and venous content of the substance was known. According to the direct Fick principle the following equation gives the cardiac output (C O):

$$\text{Cardiac Output (cc/min.)} = \frac{\text{Oxygen Intake (cc/min.)}}{\text{Arterial } O_2 \text{ Content (vol \%)} - \text{Venous } O_2 \text{ Content (vol \%)}} \times 100$$

The results are frequently expressed as the cardiac index so that comparison between individuals can be made:

$$\text{Cardiac index} = \frac{\text{C O in liters per min.}}{\text{Sq. meters of body surface}}$$

The oxygen intake or oxygen consumption is the metabolic rate under the conditions of the experiment, usually basal. It can be determined by measuring the oxygen consumed directly with the Benedict-Roth Metabolimeter as in the ordinary BMR, the procedure which we use routinely, or samples of expired air collected in a Douglas bag can be analyzed by the Haldane method of gas analysis[24].

The arterial oxygen content can be measured directly on arterial blood according to the manometric method of Van Slyke and Neil[25]. Blood is collected with an oiled syringe containing a few drops of heparin in the tip. Mercury is drawn into the syringe and the tip is sealed with a small piece of rubber tube through which a little blood is expelled as a glass tip stopper is inserted. The sealed syringe is then placed on rotating drums. The mercury insures thorough mixing and aids in preventing clotting. Mixed venous blood is obtained through the catheter from the right ventricle or pulmonary artery and handled in a similar manner.

Carbon dioxide can also be used to determine the cardiac output by determining the carbon dioxide output in the expired air and measuring the difference between the carbon dioxide contents of venous and arterial blood. Because of the wide range of variation in both venous and arterial samples and the large percentage of error in the method of determining carbon dioxide content, this method of determining cardiac output has been abandoned.

2. In the past the difficulty with the Fick procedure in determining cardiac putput was to obtain true mixed venous blood. Venous blood from various organs and various parts of the body varies in its oxygen content as well as in other chemical constituents. Thus blood entering the atrium from the coronary sinus contains 5 volumes per cent oxygen as compared to a normal in the superior vena cava of approximately 14.8 volumes per cent. Renal vein blood entering the inferior vena cava approaches arterial blood in its oxygen saturation, whereas, hepatic vein

blood is very unsaturated. The validity of the method depends on obtaining a sample of venous blood after true mixing has occurred.

Holt and Knoefel[26] had shown that true mixing does not occur in the right auricle of dogs. Cournand, et al. concluded that in man if the tip of the catheter is correctly placed in the atrium near the tricuspid valve that mixed venous blood is usually obtained although sometimes samples were poorly mixed[27]. They obtained an average normal A-V oxygen difference of 4.5 volumes per cent. Bauman in 1930 by direct puncture of the ventricle had obtained an A-V difference of 5.1 volumes per cent[28]. Warren, Stead, and Brannon found a maximum variation of oxygen content in the auricle of 2.3 volumes per cent and in the ventricle of 1.8 volumes per cent[29]. Dexter, et al. have recently shown that the maximum variation in oxygen content in samples of blood taken from the pulmonary artery was 0.4 volumes per cent and that usually the values showed a uniformity that barely exceeded the error of the Van Slyke method for determining oxygen content[30]. Warren, et al. had found a maximum of 0.4 volume per cent variation in arterial blood from the femoral artery[29].

The greatest increase in oxygen from the superior vena cava to the right auricle is 1.9 volumes per cent, from the right auricle to the right ventricle, 0.9 volume per cent, and from the right ventricle to the pulmonary artery, 0.5 volume per cent[30]. The defined limit of these values for the variation of oxygen content in the different right heart chambers of the normal subject is important in the diagnosis of congenital defects in which a left to right shunt exists (see Sec. VI).

C. Pressure Measurements

1. Pressures in the pulmonary artery and right heart are optically recorded with a Hamilton manometer which is connected to the catheter by a short piece of lead tubing[31]. The Hamilton manometer is essentially a rigid tube closed at one end by a semi-elastic metal membrane to which a small mirror is attached. The mirror reflects a vertical light beam from a slit lamp source onto the recording camera. The manometer is filled with air-free citrate solution and calibrated with an ordinary mercury manometer. Changes of pressure within the system cause a slight motion of the membrane, mirror and reflected beam of light. The sensitivity of the membrane on our arterial manometer is approximately 0.13 mm. per mm. of Hg. at an optical distance of 2 meters. A somewhat more sensitive membrane is used to record venous pulses. The natural frequency of the manometer is about 150 cycles per second. After the long catheter is attached, the frequency drops to 30 or 50 cycles per second. While such a frequency is not ideal, it is adequate to record the changes occurring in most pulse waves.

There is still controversy concerning the level of the base line. The zero level varies from individual to individual and with the different heart chambers. We have taken the level of the auricle to be 10 cm. above the skin of the back with the patient in the supine position and used this as the zero level in all pressure measurements[32].

Optical recording permits the simultaneous recording of pressure pulses along with the electrocardiogram and respiratory cycle. Characteristics of the contours of the various pulse tracings can be studied and the relationship between the mechanical and electrical events in the cardiac cycle timed. By planimetric integration the mean blood pressure can be determined from the femoral or brachial pulse pressure curves. The mean blood pressure is necessary in calculating the peripheral resistence (see Sec. IV, paragraph 3).

a. Certain difficulties arise in interpreting pressure curves. Motion or "whip" of the catheter tip induces artifacts in the tracings particularly in systolic rise of the pulmonary arterial pulse. Planimetry has not been possible on most of our

pulmonary arterial pressure curves so far, and ventricular systolic pressure has to be used to determine the pulmonary artery systolic pressure except where pulmonic stenosis is apparent from the contour. Artifacts in ventricular curves are minimal. In measuring ventricular curves Bloomfield, et al.[33] recommended that the highest average level reached with ventricular systolic ejection be taken as the ventricular systolic pressure, P_S. Two measurements are taken in diastole: 1, at the lowest point following closure of the pulmonic valve, P_{D1}, and 2, at the end of auricular systole or when this is not apparent, just before the onset of ventricular systolic rise P_{D2}. P_S minus P_{D2} is designated as the ventricular pulse pressure, P_p. We have taken the level plateau of diastasis as the diastolic pressure and as most representative of the diastolic filling pressure of the ventricle.

2. During the war, strain gauge pressure transmitters were used to measure the stresses imposed by flight on the control surfaces of aircraft. At the Aero-Medical Laboratory, Mayo Foundation, Lambert and Wood adapted the strain gauge pressure transmitter to measure the blood pressure of subjects in the human centrifuge[34]. The strain gauge manometer is a small compact unit containing a plastic cylinder filled with saline and closed by a plastic membrane which is attached to the cantilever suspension of a balanced Wheatstone bridge. Change in hydrostatic pressure causes motion of the suspension and alters the resistance of Wheatstone bridge allowing a current to flow[35]. The bridge may be wired directly to the galvanometer of the ordinary cardiograph. Electrical recording has the advantage of mobility, linear calibration with a mercury manometer, and easy control of the base line. Power is furnished by small direct current batteries eliminating AC interference. Cournand used a device requiring an elaborate amplification unit[36]. Recent models manufactured by the Statham Laboratories of Los Angeles need no amplification and have a natural frequency of 100 cycles per second with attached lead tube and 19 gauge needle[35]. Pressure curves compare very favorably with those obtained with the Hamilton Manometer[35,37].

(III) Studies on Normal Human Subjects

A. Cardiac Output

Since the turn of the century physiologisists have been attempting to determine the normal basal cardiac output of the intact man. Many methods have been proposed to measure the cardiac output, those employing the indirect Fick principle by the injection of dyes or the inhalation of acetylene were used most extensively. With the advent of right heart catheterization utilizing the direct Fick principle, the normal cardiac output for the average adult male under basal conditions has been determined as 5.6 liters per minute or 3.12 liters per minute per square meter of body surface[27,38]. The following table gives the values obtained by Cournand and Stead. These values are 26% higher than those obtained by previous methods[39].

AUTHOR:	COURNAND 13 Normal Males	Standard Deviation	STEAD 18 Normal Males
Surface Area (Sq. Meters)	1.77	± 0.19	
Oxygen Consumption (cc/min/sq. m)	138	± 13.7	128
A-V Oxygen Diff. (vol. %)	4.5	± 0.7	4.0
Cardiac Index (L/min.)	3.12	± 0.4	3.3

The establishment of normal values and the calibration of the ballistocardiograph with the direct Fick principle has made the ballistocardiograph a useful instrument in determining rapid changes in cardiac output except when aortic insufficiency is present[40,41].

2. Anxiety increases the cardiac output out of proportion to the oxygen consumption. In 5 subjects with anxiety Stead[42] found an average A-V difference of 3.1 and a cardiac index of 5.5. When the subject was tilted to 70 degrees, the A-V difference increased and the cardiac index decreased from 23 to 25%[42,43]. Syncope of the vasovagal type that was first described by Sir Thomas Lewis is frequently seen in blood donors after the removal of small amounts of blood. Following venesection of 500 to 900 cc. of blood the right auricular pressure falls and according to British investigators in McMichael's laboratory, the cardiac output decreases in a linear relationship[44]. If fainting ensues, it is characterized chiefly by a loss of peripheral resistance. Forearm flow measured by the plethysmograph is increased and since the skin is pale the site of the vasodilitation must be in the muscles. This effect can be abolished by blocking the autonomic nerves to the forearm.

3. Stead and his group have been unable to demonstrate any consistent relationship between the right auricular pressure or filling pressure of the heart and the cardiac output[45]. Thus in observations on the syncope of blood donors he was unable to find any definite change in cardiac output as the right auricular pressure fell during venesection and the peripheral resistance markedly decreased with the onset of acute circulatory collapse[46]. This does not apply of course, if bleeding is carried to the point of a significant reduction in blood volume and shock (Section IV). Moreover, Stead was unable to confirm McMichael's observations that mechanical lowering of the right atrial pressure with sphygmomanometer cuffs caused a decrease in cardiac outuut. Application of cuffs on the extremities, though lowering the right auricular pressure, caused no change in cardiac output. Releasing the cuffs caused an immediate rise in cardiac output presumably on a reflex basis due to the reactive hyperemia. A similar phenomena is observed after the release of an arteriovenous fistula[46].

Raising the auricular pressure by rapid saline infusion caused no change in cardiac output. Paredrinol, a vasoconstrictor drug without central action, raises the auricular pressure without changing the cardiac output. Anxiety increases cardiac output without changing the auricular pressure. From these and similar observations Stead concluded that the cardiac output was normally under reflex ventricular stimulation and that the auricular pressure had a reverse sufficient to insure adequate filling with any change of cardiac output demanded by activities of the body.

B. Pressures

1. R A P. Measurement of the right auricular pressure R A P with a saline manometer given an average normal mean pressure of 33 mm. of water with a normal range of from 0 to 85 mm. of water[47]. With a Hamilton manometer the mean pressure is from -2 to +2 mm. of Hg. with a maximum variation of from 4 to 8 mm. of Hg. in any one cycle. Auricular pressure curves have three characteristics: (1) An initial rise due to auricular systole, (2) a sharp rise ascribed by Wiggers[48] to the impact of the closing tricuspid valve on the auricular blood mass, (3) a fall due to the descent of the base during ventricular ejection, and (4) a fall due to the opening of the tricuspid valve and the onset of ventricular filling[33].

2. R V P. The maximum systolic pressure in the ventricle of normal subjects is between 18 and 30 mm. Hg[33]. Diastolic pressure at the lowest point in diastole (P_{D1}) is between -7 to +2 mm. of Hg., and between -0.5 to +4.5 mm. Hg. at the end of diastole (PD_2). A remarkably constant

NORMAL PRESSURES IN THE RIGHT HEART IN MM. OF HG.			
Chamber		Range	Average
R A P		- 2 to + 2	
R V P	P_S	18 to 30	25
	P_{D1}	- 7 to + 2	
	P_{D2}	- 0.5 to + 4	
	P_p	20.6 to 26.5	22.5
P A P	S	18 to 30	
	D	8 to 12	
	Mean		15

finding is the ventricular pulse pressure (P_p) which is within the narrow limits of 20.5 to 26.5 mm. Hg. averaging 22.5 mm. Hg.

3. P A P. Normal uulmonary arterial pressures as obtained by Dexter, et al. is from 20 to 32 mm. of Hg. systolic and from 8 to 12 mm. of Hg. diastolic[30]. This compares very favorably to the values obtained by Hamilton[49] in the dog by direct puncture of the pulmonary artery through a London canula which had been previously inserted and allowed to heal in place beneath the skin. Pulmonary arterial pressures are reduced to 8 mm. of of Hg. systolic and 4 mm. Hg diastolic in pulmonic stenosis and are lower than ventricular systolic pressures even in the absence of a septal defect. The P A P may approach the systolic levels in the systemic arteries in pulmonary hypertension (see Sec. V).

a. In a complicated study on the dynamics of the pulmonary circulation, Cournand presented evidence that rapid changes in P A P were the result of variations in blood flow and active engorgement of the pulmonary vascular bed[50]. There was no evidence that neurogenic control of the pulmonary resistance caused variations in P A P. A careful beat to beat analysis of pressure curves of the pulmonary artery, right ventricle, and femoral artery, taken during pressure breathing revealed that the general level of pressure in the right heart and pulmonary artery was a passive propagation of changes in intrathoracic pressure. Thus, during forced inspiration the stroke volume of the right ventricle was increased and the P A P and pulmonary resistance rose slightly, without change in the femoral pulse pressure. During forced expiration, the stroke volume of the right ventricle and P A P returned to basal levels and there was a slight increase in femoral pulse pressure. These changes point to an unequal discharge of the two ventricles during deep breathing with increase in the pulmonary blood volume on deep inspiration and a decrease on full expiration. Many ramifications of the problem are insoluble at present since the gradient of pressure in the lesser circulation cannot be determined directly due to the inaccessibility of the pulmonary veins and left auricule in the intact man. Studies on the effect of breathing 10% oxygen showed that with short periods of anoxia the P A P increased from an average normal of 21/6 mm. of Hg. to 35/13 mm. Hg., a remarkable rise in the pulmonary diastolic pressure[51]. However, the cardiac output was slightly decreased, so that an increased blood flow did not cause the pulmonary hypertension of anoxia. The pulmonary

resistance was increased almost 100 per cent. The exact mechanism of this response of the lesser circulation to anoxia is obscure.

(IV) Studies on Shock

1. The application of the technique of right atrial catheterization to the circulatory dynamics of shock has proved that the mechanism of shock as proposed by Cannon at the end of the first war is valid. Numerous studies on battle casualties in the recent war have demonstrated that there is an average loss of 40% of the normal blood volume in severe traumatic or "secondary" shock[47, 52, 53]. Extreme cases with hemorrhage may show a loss of 60% of normal blood volume[54]. This deficit in circulating volume is the crucial factor in the pathologic physiology of oligemic shock and applies to skeletal injury without external bleeding as well as to hemorrhage[55]. Hemodilution occurs in both skeletal injury and hemorrhage and the view that increased capillary permeability permitted fluid loss in trauma has given way to the concept that loss of whole blood at the site of injury accounts for the reduction in blood volume.

2. 2. The result of a lowered blood volume is a decreased venous return to the heart, a lowered filling pressure, and a proportionate decrease in cardiac output[55]. The effective filling pressure of the heart in the intact animal is the relationship between the auricular pressure (R A P) and the intrathoracic pressure[55]. However, the R A P is an adequate measure of the relative changes in the effective filling pressures of the right heart. Cournand[47] found an average R A P of -8 mm. of water in well established cases of shock as compared to a normal R A P of +33 mm. of water. One value as low as -31 mm. was recorded. The cardiac output was less than two-thirds normal. There is general agreement on the linear relationship between the lowered cardiac output and the filling pressure as measured by the R A P when the latter is lowered to certain levels by loss of blood volume. Both the output and R A P increase in parallel with replacement therapy. According to Stead such a mechanism does not operate to change the cardiac output normally (Section 111, Paragraph 3). The decrease in cardiac output is proportionate to the unsaturation of the mixed venous blood which reflects the degree of tissue anoxia. The A-V difference in shock is elevated to 8.4 or 10 volumes per cent as compared to the normal of 4.5 volumes per cent[47].

3. In general, the fall of the systolic blood pressure in shock is well correlated with the lowered cardiac output designating the importance of a r[illegible]ced pulse pressure. The total peripheral resistance is increased in most cases although there are instances in which it is reduced. Peripheral venous pressure is also increased, thereby, the gradient of pressure from the capillary bed to auricle is increased, indicating active venoconstriction[55].

The total peripheral resistance (TPR is based upon Poiseuille's law, and is expressed by the formula:

$$TPR = \frac{P \text{ (mean blood pressure) mm.hg.x } 1332}{\text{Cardiac Output in Ml. per second}}$$

The result is in dynes sec. cm. -5, usually expressed in absolute units. The average normal value for TPR is 1290. For derivation of the formula see Aperia[56]. TPR defines the resistance of the vascular bed in terms of the loss of pressure from the root of the aorta to the right auricle and the blood flow through the system. Since the auricular pressure in mm. Hg. is nearly zero, it is omitted. Thus the TPR gives the overall resistance but does not reflect the state of the vascular bed in any particular area.

4. An example of the regional vasoconstriction that occurs in shock is found in the kidney. The renal blood flow is measured by diodrast or para-amino-hippurate clearance[57]. The renal fraction,

$\frac{\text{renal blood flow}}{\text{cardiac output}}$ which is normally 19% is reduced to 10 or 3% in shock. When cardiac output is half normal the renal blood flow may be reduced to 1/10th or 1/20th normal, or to complete shutdown with anuria[55]. The reduction in renal blood flow is out of proportion to the lowering of arterial pressure[57]. The renal resistance as calculated by the TPR formula is greatly increased indicating active vasoconstriction in the kidney and specific shunting of blood away from the kidney to organs more sensitive to anoxia such as the brain. After therapy the renal blood flow returns to normal more slowly than the blood pressure or cardiac output. Uncompensated acidosis as manifested by lowered blood CO_2, and rising blood lactate, though originating in anoxic tissue is greatly enhanced by diminished renal function and is another potent indication for early and adequate therapy.

5. Shock from burns, abdominal wounds, and penetrating chest wounds presents different features not only clinically and therapeutically but physiologically. Hemoconcentration occurs in burns and abdominal wounds due to loss of plasma into the burned area or peritoneal cavity[55]. The TPR is about twice normal in shock from burns, averaging 2500 in absolute units. This extreme increase is due in part to increased blood viscosity from the hemoconcentration.

A useful concept in the study of shock is the total oxygen transport[38] a function first described as the oxyhemoglobin flow by Murray and Morgan[58].

The total oxygen transport = arterial oxygen content x cardiac output.

Hence the oxygen utilized by the tissues is expressed by the ratio:

$$\frac{\text{oxygen consumed}}{\text{total oxygen transport}}$$

In normal subjects this ratio is 25%. In traumatic shock the ratio is 60%; in burns the ratio is 35%[55]. The tissue anoxia in burns, due to the hemoconcentration and greater oxygen transport, is not as severe as it is in traumatic shock.

Stab wounds of the chest cause shock that is indistinguishable clinically from shock due to trauma and blood loss[59]. Despite the lowered cardiac output and fall in arterial pressure, the blood volume is not markedly lowered and the R A P is normal. The TPR is decreased. This indicates peripheral vasodilatation and is comparable to the arteriolar reflex dilatation seen in blood donors who have vaso-vagal syncope following the removal of 500 cc of blood (see Section III, Paragraph 3).

Shock in penetrating abdominal wounds has a higher mortality than shock from skeletal trauma[53]. Our own observations on dogs with shock secondary to peritonitis would indicate that plasma volume, R A P, and cardiac output can be maintained at normal control levels if plasma is given early and in adequate amounts. The stage of irreversible shock is characterized by a falling arterial blood pressure and a greatly lowered peripheral resistance (TPR), indicative of vasodilatation presumably due to a toxic effect on the vascular bed. The whole problem of the vasomotor control of the peripheral vascular bed in various circulatory states, and the possible beneficial use of such vasoconstrictor drugs as paredrinol need much further study[60].

6. From the above paragraphs, it is clear that therapy in traumatic shock requires replacement of blood volume with whole blood in adequate amounts, usually from 1000 cc. to 2000 cc., and that except in the case of burns, plasma is no substitute for whole blood if oxygen transport is to be restored. Studies on the use of concentrated human serum albumin in the treatment of shock have shown that albumin effectively replaces the circulating blood volume and causes a rise in R A P, cardiac output, and arterial pressure[61,62]. However, the increase in cardiac output is out of proportion to the improvement in other functions and may be above normal. The same hyperkinetic state of the cir-

culation is observed in anemia[63,64].

A modified Trendelenberg position has no effect on the R A P, cardiac output, or blood pressure in normal subjects or patients in shock with large blood loss. The blood pressure is raised in patients with moderate blood loss by the foot-up position even though there is no significant change in the cardiac output or venous return[55]. The mechanism of this phenomenon of better vasomotor control is not understood but apparently involves a shift of blood from the lower extremities to the upper parts of the body.

(V) Studies on Congestive Heart Failure

The mechanism of congestive heart failure is a subject of which our knowledge is as yet scanty. This is especially true regarding certain pertinent points in its pathogenesis, namely, the relationship between the heart rate and the cardiac output-diastolic filling pressure curves of the diseased heart[69], the exact role of decreased renal blood flow in the retention of sodium and the formation of edema[67], and the quantitative changes in the lesser circulation (particularly in the pulmonary veins) leading to pulmonary congestion[50]. Because of the limitations of the methods available the studies to be described deal chiefly with the dynamics of the right heart.

A. High and Low Output Failure

An advance in our concepts concerning the blood flow in congestive failure was made by Sharpey-Schafer[65]. Using the methods already described for accurately determining cardiac output, he was able to divide cases of congestive failure into two types; those with a high cardiac output and those with a low cardiac output. A list of the etiological classification of heart diseases under each type of congestive failure follows:

'I' Low output failure
1. Hypertensive heart disease
2. Arteriosclerotic heart disease
3. Valvular heart disease

'II' High output failure
1. Cor pulmonale
2. Thyrotoxic heart disease
3. Beriberi heart disease[66]
4. Congestive failure in severe anemia (as in pernicious anemia)[63]
5. Congestive failure associated with large arterio-venous fistulae[45]

Such a distinction establishes the fact that the clinical syndrome of congestive failure occurs with the heart pumping a normal or greater than normal amount of blood. It suggests further that although the output may be greater than normal it is still not sufficient to meet the metabolic demands of the body. Not every instance of failure due to hypertensive, arteriosclerotic, or valvular heart disease will have a low cardiac output. Such patients at rest may have a normal output but their failure is greatly augmented by physical activity, suggesting that the output, though normal by accepted standards, is insufficient when the demand is increased The effect of exercise on the dynamics of the normal and diseased heart is virtually an unexplored field of investigation.

1. The determining factor in the Fick equation in low output failure is the degree of unsaturation of the mixed venous blood, giving a high figure for the A-V oxygen difference in the denominator and a low value for the output. In high output failure, such as thyrotoxic heart disease, the oxygen consumption in the numerator is greatly increased with little change in the A-V difference. In cor pulmonale the denominator is decreased due chiefly to the lowered oxygen content of the arterial blood. Although the oxygen saturation is normal in anemia, the arterial oxygen content is low because of a reduced hemoglobin, Stead and his group[64] have established the critical level of hemoglobin at 7 grams per cent before an increase in cardiac output occurs.

A circulatory improvement logically follows the treatment of thyrotoxicosis with thiouracil[65], with a reduction in cardiac output and diuresis if failure is present. Sharpey-Schafer has reported on the treatment of other types of heart failure with thiouracil. Patients with cor pulmonale treated with thiouracil show a reduction in cardiac output and a fall in R A P. They are clinically improved even though the arterial blood is no better oxygenated. The mixed venous blood, however, is better oxygenated. Patients with low output failure treated with thiouracil are clinically improved even though the low cardiac output is not significantly changed. The R A P falls and the mixed venous blood is better oxygenated. Certain patients with intractable decompensation had a diuresis and were able to achieve some degree of ambulation. In each instance the metabolic demand of the tissues for oxygen was decreased, theoretically decreasing the work of the heart. Levine, Blumgart, and Berlin first recommended thyroidectomy for intractable failure in 1933.

2. Of basic interest is the reduced renal blood flow in low output failure. The normal renal blood flow is 1.2 liters per 1.73 sq. meters of body surface or 20% of the cardiac output; hence a reduction in cardiac output may lead to a considerable lowering of renal blood flow. Merrill[67] found a high positive correlation between the reduction in cardiac output and the reduction in renal blood flow and no correlation between the elevated venous pressure and the reduced renal blood flow. In patients with edema and without evidence of hypertension of renal disease, the renal blood flow was reduced to one-fifth normal when the cardiac output was approximately half normal, pointing to a diversion of blood away from the kidney (so called "forward failure"). Glomerular filtration and sodium excretion by the kidney was also diminished. Among factors precipitating congestive failure, exertion and infection are two of the most common; the possibility that they may produce this effect by diverting blood away from the kidney, thus interfering with renal excretion of salt and water, has not been established as a fact. Values for cardiac output in 18 cases studied with manifest failure ranged from 1.3 to 2.9 liters per minute per square meter of body surface as compared to an average normal cardiac index in that laboratory of 3.3.

3. In the normal human subject, intravenous digoxin causes a decrease in cardiac output and a fall in auricular pressure[68]. The same situation obtains in cor pulmonale with clinical improvement accompanying the decrease in the previous high output. In low output failure, the output is increased by digoxin as the right auricular pressure falls. Mechanical lowering of the auricular pressure by sphygmomanometer cuffs on the extremities[68] and venesection of 1000 cc of blood[69] qualitatively produces the same changes as digoxin. This is in contradistinction to the effects of bleeding in normals (see Section III, paragraph 3) and supports the theory that Starling's curve for the overloaded heart applies to the cardiac dynamics of patients in chronic low output failure with an increased blood volume. The question is again raised as to whether digitalis has a principal or single action on the peripheral vascular bed[68], but the evidence to date would indicate that the increased output after digitalis was due in part at least to increased myocardial efficiency[70].

As decompensation progresses a fairly constant finding is an increase in the total peripheral resistence TPR[70]. Following venesection the TPR is markedly lowered[69]. The cause of this phenomenon awaits further study.

B. Pressures

1. An important feature of right heart failure is the elevation of the central venous pressure R A P. An early observation of Cournand[71,72] showed that the gradient in pressure in the veins from arm to auricle is lost. Normally this gradient is approximately 4 cm. of saline. Recent studies[33] have shown that the auricular pressure may actually be higher than

the peripheral venous pressure. Pressure tracings reveal that only at the time of opening of the A-V valves with rapid filling of the ventricle does the atrial pressure drop, restoring the venous gradient. This clarifies the frequent clinical observation of finding a normal venout pressure in the arm vein in cases of advanced right heart failure with edema.

2. Cor pulmonale offers a unique opportunity to study right ventricular pressure curves of the failing heart since it is dependent for its development upon pulmonary arterial hypertension that is clearly secondary to the increased pulmonary resistance of long standing lung pathology. Considerable degrees of pulmonary fibrosis may exist without change in ventricular pulse pressures. The earliest change is in the ventricular systolic pressure. Clinical failure is not manifest until the ventricular diastolic pressure and right auricular pressure become elevated. Bloomfield, et al.[33] have listed the following characteristic features of the pulse pressure curves:

a. Ventricular systolic hypertension.
b. Abnormal elevation of the general level of the ventricular diastolic pressure.
c. Marked rise of the ventricular diastolic pressure above the minimum which occurs early in diastole, giving rise to a sharp early diastolic "dip" between the arched down-curve of isometric relaxation and the plateau-like remainder of diastole.
d. Elevation of the mean auricular pressure.
e. Accentuation of the drop in auricular pressure at the time of descent of the base resulting from ventricular ejection.
f. Exaggeration of the fall of pressure in the auricle at the time of rapid emptying into the ventricle, corresponding closely in time with the early diastolic dip in the ventricle.
g. Elevation of the peripheral venous pressure.
h. Appearance of retrograde transmission into the peripheral venous system of intra-auricular pressure variations, especially of the negative waves.
i. Diminution of the normal gradient of pressure between the antecubital veins and the right auricle, often to the point where the two differ by but a few mm. of saline.

Systolic ventricular pressures of 80 mm. Hg. have been recorded in cor pulmonale. In this connection it is interesting to note that pressures of 116 mm. of Hg., comparable with systemic arterial pressures, have been recorded in mitral stenosis. This is in keeping with classical conception of the disease and of the pulmonary vascular changes in mitral stenosis that were emphasized by Parker and Weiss. The quantitative changes in the pulmonary circulation leading to pulmonary congestion and responsible for the dyspnea of left myocardial failure remain to be clarified. (see footnote*).

It is interesting to compare the dynamics of shock and low output failure. The reduced cardiac output and increased TPR are the same. The chief difference, of course, is in the blood volume and RAP, both increased in failure, and both decreased in shock. The blood pressure in failure is usually well maintained, except for certain notable instances where the shock syndrome supervenes. A classic example of shock superimposed on congestive failure is seen frequently in acute myocardial infarction. Richards[55] reported two cases presenting this

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*Since this manuscript was written, an excellent review by Richards on the contributions of right heart catheterization to the physiology of heart failure has been published[79]. An interpretation of Starling's law as applied to the cardiac dynamics of the intact man is given. In the light of newer knowledge, the terms "congestive failure" "forward failure", and "backward failure" are discussed. It is pointed out that the constant distrubances in congestive failure are in terms of pressure rather than flow.

picture, where the blood volume was normal, the cardiac output decreased, the arterial pressure low, and the RAP increased. Further study is needed to clarify the complicated dynamics of the shock syndrome in congestive failure.

(VI) Congenital Heart Disease

The chief practical value of catheterization of the heart seems to lie in the field of congenital heart disease. Here the technique is of value in determining the exact congenital defects present and also in defining the physiological effect exerted on the circulation[30].

A. Shunts With Flow of Blood From Left to Right Heart

If samples of blood taken from the inferior vena cava, superior vena cava, right auricule, right ventricle and pulmanary artery are analyzed for their oxygen content the presence of shunts from the right to the left heart or systemic to pulmonary circulation can be determined. In studies of this type the normal variations of oxygen content in various parts of the circulation must be carefully considered. These variations have been extensively studied and are summarized in Section II, Paragraph B2.

In interauricular septal defect the oxygen content of blood from the right auricle exceeds the oxygen content of blood from the superior and inferior vena cava. In 3 cases studied by Stead the average increase was 2.7 volumes per cent[73]. In many cases the catheter can be passed through the defect into the left auricle[74]. The pressure in the left auricle exceeds that in the right auricle[75]. In uncomplicated interventricular septal defect the content of oxygen in blood obtained from the right ventricle and pulmonary artery exceeds that of blood from the right auricle[76]. If blood is taken with the tip of the catheter immediately opposing the defect the oxygen content of the blood obtained may approach that of blood from the femoral artery. Pressures in the right ventricle and pulmonary artery are normal or slightly elevated when the defect is uncomplicated by pulmonic stenosis. In patent ductus arteriosus the oxygen content of blood obtained from the right or left pulmonary artery exceeds that of blood from the right ventricle. There may be elevation of pressure in the pulmonary artery and right ventricle or the pressure may be normal.

Care must be exercised in the diagnosis of small shunts by differences in oxygen content of blood taken proximal and distal to the shunt. Dexter has estimated that cases of patent ductus arteriosus with flows through the ductus of less than 0.5 liters per minute cannot be diagnosed in this manner[30]. This is even more true in patent interauricular septal defect where the normal variation between the oxygen content of blood from the superior or inferior vena cava and right auricle may be large. An accurate diagnosis of small defects can be made if the catheter can be passed through the defect or if the tip of the catheter is placed immediately opposing the defect.

The amount of blood passing through the shunt can be calculated from the oxygen consumption and the oxygen content of blood above and below the shunt together with the oxygen content of arterial blood[30]. The cardiac output is calculated in two ways using the Fick equation described in Section II, Paragraph B1. In one calculation the oxygen content of blood obtained above the shunt is used for venous blood and in the other calculation the oxygen content of blood below the shunt is substituted in the equation. The difference between these two values for cardiac output is equal to the amount of blood passing through the shunt per minute. This is of some interest in determining the strain placed on the heart by the shunt and should be of aid in evaluating the need for operation in patent ductus arteriosus.

B. Pulmonic Stenosis

In pulmonic stenosis there is a marked difference between the systolic pressures in the pulmonary artery and

the right ventricle. Often it is difficult to pass the catheter through the stenotic area and at times this may be impossible[30].

C. Tetralogy of Fallot

Considerable information can be obtained in tetralogy from cardiac catheterization although it cannot be relied on as the sole diagnostic measure[30]. The catheter can be passed without difficulty into the right ventricle. From there it may be possible to pass it through the stenotic pulmonary valve into the pulmonary artery, but often this impossible. On occasion the catheter may pass into the aorta or left ventricle. The systolic pressure in the right ventricle is approximately the same as in the femoral artery in most cases. The systolic pressure and pulse pressure in the pulmonary artery is markedly reduced as a result of the pulmonic stenosis. The O_2 content of blood from the right ventricle is higher than that in the right auricle in most cases indicating that there is a shunt from left to right as well as from right to left.

The pathologic physiology of Tetralogy of Fallot is of interest because of the recent advent of surgical methods of treating this congenital abnormality. Bing has studied exhaustively a group of patients with this disease[77]. He calculated systemic blood flow from the formula:

$$\frac{O_2 \text{ consumption}}{O_2 \text{ content arterial blood} - O_2 \text{ content right auricular blood}}$$

and pulmonary artery blood flow from the formula:

$$\frac{O_2 \text{ consumption}}{O_2 \text{ content pulmonary vein} - O_2 \text{ content pulmonary artery}}$$

He assumed the O2 saturation of pulmonary vein blood to be 95 per cent. Often blood could not be obtained from the pulmonary artery as the catheter could not be passed through the area of pulmonary stenosis. In these cases the sample was taken from the right ventricle near the pulmonary valve which introduces some error because of the interventricular septal defect. The systemic blood flow varied considerably being above the normal value in some instances and below in others. The pulmonary artery blood flow was less than the systemic blood flow, in many cases the decrease being more than 50 per cent. The difference between systemic and pulmonary artery blood flow is assumed to be due to blood passing through the interventricular septal defect and the overriding aorta directly into the systemic circulation. Bing points out that in many cases of tetralogy there is an extensive collateral circulation to the lung by means of bronchial and other arteries. He attempted to define this collateral circulation quantitatively by measuring the pulmonary capillary flow and comparing this with pulmonary artery blood flow. By pulmonary capillary flow he meant the blood flow through the pulmonary capillaries supplied by both the pulmonary and bronchial arteries. He measured this by means of an indirect method involving the determination of the O_2 content of blood reaching the capillaries by equilibration with a carbon dioxide-oxygen mixture inhaled from a bag. The CO_2 content of alveolar air and the CO_2 output of the individual were determined, and the Fick principle was applied in the usual manner. In younger patients the pulmonary capillary flow tended to agree with pulmonary arterial flow but in the older age group there appeared to be evidence of a considerable collateral circulation, the pulmonary capillary flow often being a liter or more greater than the pulmonary arterial flow. The author freely admits there are a number of objections to the accuracy of his methods, but the results are of interest in clarifying the physiology of the disease.

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III. GOSSIP

Dr. Clarence E. de la Chappelle, Professor of Clinical Medicine, New York University, made an excellent contribution to our teaching program November 3 and 4. He spoke at Hennepin County Medical Society on evening of November 3 and gave lectures and clinics on campus both days. His field of interest is cardiology and he has a wealth of information which he dispenses in a highly satisfactory manner. We were fortunate in securing him as guest faculty member as he had been on vacation in Sun Valley prior to the annual meeting of the American Association of Medical Colleges, and stopped off on his way back to New York City. He saw the Veterans' Hospital and remarked that he had never seen a more energetic, well informed group of young men.. The continued success of these hospitals will be the direct result of the ability of those in charge to attract up-and-coming young men. Now non-veterans can be appointed for residency training if suitably qualified veterans are not available....Staff meeting last week by Surgery was well received. Dr. Wangensteen's remarks at the end summed up the situation very well when he explained the purposes of his program. There are few (if any) departments of surgery where better teamwork has been developed between those who work in the experimental laboratories and those who function on the wards and operating rooms....Because of a kind word I had to say for lepers, the editors of the Star, official publication of the patients of the U.S. Marine Hospital, National Leprosarium, Carville, La., is sent to me. Each copy contains the following statement -"This paper and all outgoing mail is sterilized before leaving the hospital. This is done only as a gesture of respect to the unconvinced and not because there is any scientific necessity for it. The Leprosarium at Carville was founded 52 years ago. To date not a single member of the medical or surgical staff has contracted the disease." The paper is also crusading to drop the name leprosy and call it Hansen's Disease. At one time Minnesota was known as a leper state. This distinction was hardly justified as other states also had a good many lepers. Hansen came to Minnesota in early days to observe effect on the course and development of leprosy from the change of climate from the Scandinavian countries to Minnesota,..Cecil J. Watson has just returned from Boston where he was visiting staff member at Peter Bent Brigham Hospital. He will leave shortly for South America on a special mission and will return the middle of December. Our department of Medicine is rapidly becoming known throughout the country as one of the best in the field.. ..Believe it or not, Urologist C. Donald Creevy has returned to his first love. A shortage of urologists in Minneapolis developed during the war largely as a result of the untimely death of Ernie Moland. Don Creevy changed to a part time basis here and opened up an office downtown. When the war was on he brought two outstanding young urologists into his office (Drs. Webb and Smith). As soon as everything was going smoothly he decided to move back. This may be difficult for some people to understand but anyone who has enjoyed the success which Don has in his University connection, will understand....The mail bag yields this one. "My stomach is such good company I scarcely need a radio. How it sings at its work! It gurgles and squeals, chatters and croons, has more fun than a kid with bubble-gum."....Speaking of honors, Leo C. Rigler has been invited to give the Pancoast Lecture in Philadelphia this week. His subject will be "The Limitations of Roentgen Diagnosis....Friday, Nov. 14, the Minnesota Public Health Conference will meet at Radisson Hotel. At general session in afternoon a Panel Discussion on Public Health Problems of an Aging Population will be presented. At banquet in evening Dr. A. J. Chesley will be honored. Our state health officer is one of the best liked men in the business. He has always been a tower of strength in the health movement and many will come to honor him. Four luncheon sessions are for health officers, public health nurses, sanitarians and health educators....It is predicted enrolments will soon begin to drop. Anticipated veteran enrolment did not develop this fall and many G.I.'s are planning to drop out at end of this quarter. Going to school isn't as simple as it seems. The other day a newspaper cartoon showed a couple getting their children off to school while they were doing the same thing. The wife remarks that unless the children hurry, mama and papa will be late too.....