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# Heart Catheterization in Congenital Heart Disease

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## INDEX



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# I. THE VALUE AND LIMITATIONS OF HEART CATHETERIZATION IN CONGENITAL HEART DISEASE

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## (I) History

Recent advances in the surgical treatment of congenital disorders of the heart, pioneered chiefly by the work of Gross[1], and Blalock[2], have increased the importance of accurate diagnosis. The physical and x-ray signs, as well as the other well established methods for cardiac study, are often unsatisfactory in understanding the dynamics of the circulation in patients with congenital heart lesions. Many of these time tested methods, plus the older methods of measuring cardiac output, are applied with difficulty, if at all, in such patients, and certain aspects of the pulmonary circulation have heretofore been almost immune to measurement.

The introduction by Forssman[3], of the cardiac catheter in 1929, and its practical improvement by Cournand and Ranges[4], have offered a new opportunity for the study of congenital heart disease. By these techniques, pressures may be measured in the great veins, the right auricle and ventricle, and the pulmonary artery. Analysis of blood samples obtained from these areas and from the femoral artery, permits the calculation of the cardiac output by the Fick principle, and the calculation of the volume of any existing shunts, may be estimated by this same method.

## (II) Methodology

### A. Technique of catheterization:

The actual technique of catheterization has been well described by Cournand and Ranges[4], and indeed, at a staff meeting bulletin here by Borden and Ebert[5]. Briefly, under strict surgical asepsis, and using local novocaine anesthesia, the median baselic vein is exposed, usually in the left arm, and a size #8F or 9F, 100 cm. cathe ter introduced into the vein through a small fish-mouth incision. The catheter is then directed under fluoroscopic control into the right auricle, and by rotating the tip, it is made to pass into the right ventricle and thence into the pulmonary artery. Beginning at this point, blood samples are withdrawn through the catheter under oil as described by Cournand[6]. Blood samples and pressures are obtained first from the pulmonary artery, next the right ventricle, and finally from the right auricle and the vena cava. The oxygen content is determined routinely on all samples, plus a sample from the femoral artery, by the method of Van Slyke and Neill[7]. In addition hemoglobin concentrations are determined on all samples by photocolorimetry, and the percentage oxygen saturation calculated. Pressures are recorded optically through the use of strain gauges (a modified wheatstone bridge), that are connected with a string galvanometer.

As near as possible to the time of actual catheterization, calculation of the patient's oxygen consumption is carried out, using a standard basal metabolism machine, with the oxygen consumption figured in terms of cubic centimeters of oxygen per minute.

### B. Calculations of blood flow:

The most accurate method at present for the estimation of cardiac output in man is based on the principle outlined by Fick[8], in 1870. Fick derived an equation which may be restated as follows:

$$\text{Cardiac output (Liters per minute)} = \frac{\text{Oxygen intake (c.c. per minute)}}{\text{Arteriovenous oxygen difference (c.c. per Liter)}}$$

In normal individuals without shunts, the calculation is based on the deter-

mination of oxygen consumption, oxygen content of arterial blood, and oxygen content of mixed venous blood, this latter sample best secured from the pulmonary artery where samples show minimal variation and represent acceptable values for mixed venous blood[9]. To estimate the flow through a shunt, the peripheral blood flow and the pulmonary blood flow must be calculated separately. The peripheral blood flow may be calculated in the usual fashion except that the sample of mixed venous blood must be obtained proximal to the shunt. For example, in a patent ductus arteriosus, it must be obtained from the right ventricle, in cases of ventricular septal defect from the right auricle, and in cases of auricular septal defect, from the vena cava. Since true mixing becomes progressively poorer in this sequence, errors in the application of the Fick principle become greater. The difference then, between the peripheral and pulmonary flows will give us the volume of flow through single, unidirectional shunts or will permit calculation of the sum total of the volume of the shunt when two unidirectional shunts are present. If the shunt is in both directions a more complicated method may be used, but this will not be discussed in this paper.

(III) General Considerations of the Need for Cardiac Catheterization in Congenital Heart Disease.

A. Present methods available for diagnosis:

1. History and physical
2. X-ray
3. Electrocardiography
4. Circulation times
5. Heart catheterization
6. Angiocardiography
7. Electrokymography
8. Oximeter studies

B. As has been indicated by Shapiro[10] and others, the correct diagnosis of congenital heart disease depends on a fundamental knowledge of cardiac physiology. It is worth emphasizing that in a large percentage of cases, a precise diagnosis can be made by the usual methods, using only the first four in the table above. However, in many cases a precise diagnosis is not possible, or may be questionable, and it is only when the last four techniques are used that more precise diagnoses are possible. With the giant strides that cardiac surgery has taken in recent years, it behooves us to examine closely every patient with congenital heart disease, so that we will not deny surgery to every deserving patient.

It is our purpose, then, to discuss the value of cardiac catheterization in those patients in whom accurate diagnosis has either been impossible by the routine methods, or in cases where the clinician has found reasonable doubt as to the precise diagnosis.

With this armamentarium at his disposal, the clinician finds himself in a better position to make accurate diagnoses, and his patient will benefit from the knowledge that no stone has been left unturned in an effort to assess his lesion, from the standpoint of possible surgical relief.

C. In this paper, we will limit ourselves to the discussion of the value of cardiac catheterization alone, and point out the demonstrable values of this technique in a number of patients seen at the University Hospitals, and also point out the limitations of this technique.

(IV) Normal Values

A. The following table will illustrate the relationship between the pressures and oxygen saturations in the various chambers of the heart. These values represent a mean value for a group of cases studied in our laboratory, and do not represent actual values in any one case. However, they do represent typical situations in patients with congenital heart disease of the types listed.

NORMAL AND PATHOLOGICAL VARIATIONS
IN CONGENITAL HEART DISEASE

| LESION | INTRACARDIAC PRESSURE | | PERCENTAGE OXYGEN SATURATION | | | | | |
|---|---|---|---|---|---|---|---|---|
| | RT. VENT | PULM. ART. | VENA CAVA | RT. AUR. | RT. VENT. | PULM. ART. | FEM. ART. | PULM. VEIN |
| NORMAL | $\frac{25\pm5}{0}$ | $\frac{25\pm5}{8\pm4}$ | 65-80% | Same | Same | Same | $96\pm3\%$ | Same |
| INTER-ATRIAL DEFECT | Normal or Elevated | Same | 60% | 85% | 85% | 85% | " " | " " |
| INTER-VENT. DEFECT | Normal or Elevated | Same | 80% | 80% | 90% | 90% | 93% | ---- |
| PATENT DUCTUS | Normal or Elevated | Same | 70% | 74% | 75% | 84% | 98% | ---- |
| ISOLATED PULMONIC STENOSIS | High | Low | 70% | Same | Same | Same | 98% | ---- |
| TETRALOGY OF FALLOT | High | Low | 60% | 60% | 62% | 59% | 80% | ---- |
| EISENMENGER COMPLEX | High | High | 68% | 59% | 74% | 74% | 78% | ---- |
| ANOMALOUS PULM. VEIN | Normal or Elevated | Same | 70% | 80% | 80% | 80% | 98% | 98% |

It must be mentioned again, that the variation in oxygen content in the different chambers of the right heart is important in the diagnosis of congenital defects in which a shunt exists. In this regard, multiple samples must be obtained from each chamber. Warren, Stead, and Brannon[11] found a maximum normal variation of oxygen content in the auricle of 2.3 volumes per cent, and in the ventricle of 1.8 volumes per cent. Dexter[9] has shown that the maximum normal variation in oxygen content of pulmonary artery samples was 0.4 volumes per cent. The greatest normal variation in oxygen content from vena cava to right auricle is 2.0 volumes per cent, from the right auricle to the right ventricle, 0.9 volumes per cent, and from the right ventricle to the pulmonary artery, 0.5 volumes per cent.

B. In our studies it has been the policy to obtain blood samples from both inferior and superior vena cava; two samples from the right auricle; two samples from the right ventricle, (one low in the ventricle, and the other just below the pulmonary valve); and two from the pulmonary artery, (one in the main pulmonary artery, and the other in either the right or left main branch). In addition, a sample of blood is obtained from the femoral artery.

(V) Congenital Heart Disease - Types amenable to diagnosis by heart catheterization

A. Acyanotic Group

1. Patent Ductus Arteriosus - the ductus arteriosus is a vessel of considerable size and importance in utero, acting as a shunt to permit the blood from the pulmonary artery to reach the aorta. Normally within a few moments after birth this ductus closes, functionally at least, to subsequently become a fibrous structure. Should the ductus persist, however, the result is a congenital shunt that produces a varying degree of symptomatology and alteration in cardiac physiology. The symptoms of a patent ductus arteriosus will depend largely on the size of the ductus, and therefore on the amount of blood shunted from aorta to pulmonary artery. With small shunts there will be few or no symptoms, but in larger shunts symptoms of left heart failure will eventually appear. The diagnosis of patent ductus arteriosus is usually easily made in the typical case, by the recognition of the following features:

a. Machinery murmur- this is a characteristic murmur heard best over the pulmonic area, and often accompanied by a palpable thrill. The murmur itself is heart throughout systole and diastole, and within the murmur one can usually hear an accentuated pulmonic second sound.

b. The heart may or may not be enlarged, and if enlarged it is the left ventricle that accounts for the enlargement.

c. The pulmonary artery and its branches are prominent, with prominent pulsations evident on fluoroscopy.

d. The aorta is prominent on roentgen examination.

e. With any significant shunt there is an increased pulse pressure, usually accompanied by other peripheral signs such as capillary pulse, positive Duroziez's sign, etc.

f. The electrocardiogram is usually normal or may show a left axis deviation.

g. The lesion is more common in females.

Case Reports:

The following case illustrates the classical findings in an 18 year old male. This boy was seen with a history of exertional dyspnea, palpitation, and occasional dizziness. On examination he was found to have a large heart, left ventricular in type, a loud machinery murmur over the pulmonic area, and enlargement of the pulmonary artery on x-ray examination. The diagnosis of patent ductus arteriosus was made as a result of these findings and confirmed by subsequent cardiac catheterization and surgery. The findings on catheterization in this patient revealed an oxygen content of 12.0 volumes per cent in the right auricle, 11.7 volumes per cent in the right ventricle, and an oxygen content of 15.2 volumes per cent in the pulmonary artery, proof of a large shunt from aorta to pulmonary artery. Calculated on the Fick equation this represented a shunt of nearly 12 liters per minute. The cardiac catheterization, was, of course, unnecessary from a diagnostic standpoint but was done to obtain good control values. A second case, however, demonstrates the value of catheterization. This girl, B.B., age 13, was seen in the Out-patient Department where most of the findings of patent ductus arteriosus were demonstrated. However, the murmur heard in this patient was systolic only in time, and was also shown to be systolic in time by phonocardiogram. Because of this finding the need to rule out an interventricular or interauricular septal defect became important if this girl was to be given the benefit of surgery. Accordingly, cardiac catheterization was carried out, and evidence of an aortic pulmonary shunt was demonstrated, and the girl operated upon. At the time of surgery a large ductus was found and divided. The values for oxygen content in the various chambers of the heart revealed an increase of 1.0 volumes per cent from right ventricle to pulmonary artery, again, compatible with the diagnosis of a patent ductus arteriosus.

This case then demonstrates the need for catheterization, for had this proved to be a septal defect and surgery done without a prior cardiac catheterization, the patient would have been exposed to an unnecessary surgical risk.

2. Interauricular Septal Defect.-

The interauricular septal defect is the result of failure of fusion of the interauricular septum, and here again, the size of the defect is the determining factor in the production of symptoms and findings. The diagnosis of this lesion is dependent on the following:

a. Patients are usually stunted in their development.

b. There is usually a systolic murmur heard over the pulmonic area, but this is often absent. The murmur is harsh and often accompanied by a thrill.

c. Right ventricular enlargement is characteristically found, and the heart is usually globular in shape.

d. The pulmonary artery is greatly enlarged, and on fluoroscopy the branches pulsate vigorously.

e. The left ventricle and aorta are underdeveloped as a result of the shunt from left auricle to right auricle.

f. The pulse pressure is normal.

g. The electrocardiogram is either normal or may show right axis deviation.

Case Reports

The following case illustrates a fairly typical interauricular septal defect. , a 12 year old boy, had a history of fatigue, palpitation, and a heart murmur known since age 3. On examination there was a systolic murmur heard over the entire precordium, accompanied by a thrill. The blood pressure and pulse were normal. On electrocardiogram there was

right axis deviation. On x-ray examination the heart was globular in shape and it was noted that the aorta was small, the right ventricle enlarged, and the pulmonary artery very prominent with large, pulsating pulmonary artery branches. A clinical diagnosis of inter-auricular septal defect was proved on catheterization, by virtue of the following findings. The oxygen content of the superior vena cava was 9.0 volumes per cent, the right auricle 13.5 volumes per cent, and the right ventricle and pulmonary artery 13.5 and 14.0 volumes per cent.

A second case, however, was somewhat more difficult of diagnosis, and here cardiac catheterization helped in arriving at the diagnosis. ., a 7 year old girl, showed most of the classical findings of an interauricular septal defect, but in view of a moderately elevated pulse pressure, and the finding of a tendency to left axis deviation on the electrocardiogram, some doubt existed as to whether she might not have an atypical patent ductus arteriosus. Accordingly, heart catheterization was done, and the clinical impression of an interauricular septal defect proved by the passage of the catheter through the auricular septal defect into the left auricle and from there into a pulmonary vein where the oxygen content was 14.0 volumes per cent, (or 100 per cent saturated as calculated on the basis of the hemoglobin concentration of that sample). This case, while fairly typical, by virtue of catheterization ruled out any room for doubt as to the diagnosis.

3. Interventricular septal defect.

As in interauricular defects, this lesion results from failure of fusion of the various portions of the interventricular septum. Again, the symptoms and findings depend on the size of the defect, and accordingly, the amount of blood shunted from the left ventricle to the right ventricle. The diagnosis of this lesion in the typical case is dependent on the following:

a. A loud systolic murmur is usually heard over the lower end of the sternum, often accompanied by a palpable thrill.

b. On x-ray examination the heart is usually normal in size.

c. The pulmonary artery and its branches are enlarged and will pulsate vigorously as a rule.

d. The blood pressure and pulse pressure are within normal limits.

e. The electrocardiogram will be normal or occasionally will show a heart block due to interference in conduction over the bundle of His.

The chief error in diagnosis is in the differentiation between this lesion and an interauricular septal defect.

Case reports:

The following case, , a male, age 5, is indicative of this difficulty. On examination there was a harsh systolic murmur and thrill, best heard at the 4th interspace to the left of the sternum. The heart was moderately enlarged with a small aorta, and prominent pulmonary artery and vascular markings. The blood pressure was normal, and the electrocardiogram was normal, with normal axis. A pre-catheterization diagnosis of interauricular septal defect was made, both clinically and radiologically, but on catheterization a shunt between the ventricles was well demonstrated. In this case the oxygen content in the vena cava was 11.0 volumes per cent, in the right auricle 10.9 volumes per cent, and the right ventricle and pulmonary artery, 12.5 and 12.8 volumes per cent. This indicated a shunt from left ventricle to right ventricle.

A second case, , a 6 year old female, was similar to the first in that a diagnosis of inter-auricular septal defect

had been made. In this case the systolic murmur was heard over the left 2nd interspace, the x-ray findings demonstrated a small aorta, and enlarged pulmonary artery, compatible with an interauricular septal defect. On catheterization, however, the findings of an interventricular septal defect were demonstrated by the finding of an oxygen content of 11.3 volumes per cent in the superior vena cava, a content of 11.1 volumes per cent in the right auricle, and an oxygen content of 12.4 volumes per cent in the right ventricle and pulmonary artery.

These cases, then, are evidence of the value of heart catheterization, and while the differentiation, in these two cases, between the lesion of an interauricular and an interventricular septal defect, may be academic at the present, it is well to keep in mind the fact that one day one or both of these lesions may well be amenable to surgical attack.

4. Isolated pulmonic stenosis -

While this is a rare lesion, it is met often enough to be considered in our discussion. In this lesion, the sole defect is a stenosis of either the pulmonary valve, or a stenosis of the infundibular portion of the right ventricle. The findings in such cases are usually as follows:

a. Absence of cyanosis - although this problem is one of wide discussion, the cases we have examined here have been free of any cyanosis, and review of recent literature on the subject would indicate that cyanosis is not present in pure pulmonary stenosis. In those cases with accompanying patency of the foramen ovale it has been postulated that the increased pressure in the right ventricle will raise the pressure in the right auricle and thence shunt venous blood into the systemic circuit to cause cyanosis. However, our studies indicate that the pressure change in the right auricle is insignificant, in spite of extremely high right ventricular pressures.

b. Systolic murmur over the pulmonic area. This is a harsh murmur, with an accompanying faint or absent pulmonic second sound.

c. The heart is enlarged with prominent of the right ventricle, and may be globular in type.

d. The pulmonary artery is small and the lung pulsations much decreased.

e. The electrocardiogram will show a right axis deviation, and frequently a right heart strain pattern.

Case report:

The following case was of interest in this regard. , female, age 18, was referred to us as a possible patent ductus arteriosus or interauricular septal defect. On examination here she demonstrated an enlarged heart with right ventricular prominence, and a large pulmonary trunk shadow. The lung pulsations, however, were diminished. A harsh systolic murmur was heard over the pulmonic area and the pulmonic second sound was decreased. While the findings then, indicated reduced flow to the lungs, it was not evident why she had such a large pulmonary trunk shadow. On catheterization, however, she proved to have a markedly elevated pressure in the right ventricle, and an abnormally low pressure in the pulmonary artery. The pressure in the pulmonary artery was 12/8 mm. of mercury, and the right ventricular pressure was 120/0 mm. of mercury.

Catheterization then, in this patient demonstrated that the pulmonary artery enlargement was in the nature of a paststenotic dilatation.

5. Anomalous pulmonary vein. - This rarity has been encountered

on two occasions in our studies. Ordinarily, this lesion may take on several forms. In the most serious form all of the pulmonary veins empty into the right auricle, a lesion incompatible with life. Less serious are those cases with one or two veins entering the right atrium. In the latter case the appearance of such patients is not suggestive of an interauricular septal defect, and usually presents itself with the classical features of such a defect as described above. However, by its nature it is significantly different in that it lends itself to surgical approach.

Case report:

Of greatest interest to us was the case of , a female, age 16. This girl was referred to us with a history of the discovery of "congenital heart disease" at age 6. She had had 2 episodes of acute rheumatic fever after age seven. The striking feature of the case, however, was the fact that in spite of a typical mitral diastolic murmur, she was practically asymptomatic, and showed no sign of left auricular enlargement. There was a rough systolic murmur over the pulmonic area, and a very short, high-pitched diastolic murmur accompanying this. The x-ray findings were considered in keeping with a diagnosis of interauricular septal defect, and with the findings of a mitral stenosis the clinical impression was "Lutembacher's" syndrome. On catheterization of this patient's heart an anomalous vein that entered the right atrium was demonstrated in that the catheter passed directly from the right atrium into the lung field, and blood samples from this area revealed arterial blood with a saturation of 98 per cent. This then, was invaluable information, as it explained the lack of symptoms from the mitral stenosis. In essence, then, this patient had provided herself with a vascular shunt that reduced the left auricular load, a mechanism that has recently been used by Bland and Sweet[12] in the surgical treatment of mitral stenosis.

(V)

B. Cyanotic congenital heart disease

1. Tetralogy of Fallot -

This lesion is responsible for about 75% of all patients with cyanotic heart disease, and as evidenced by its name, has four characteristics, pulmonary stenosis, high interventricular septal defect, overriding aorta, and right ventricular hypertrophy.

The most important feature of this lesion, however, is the inadequate flow of blood into the lungs by virtue of the pulmonic stenosis. The classical features of this entity, and the diagnostic features are as follows:

a. Increasing cyanosis from birth. The cyanosis is due to the admixture of venous and arterial blood. This cyanosis is accompanied by severe clubbing of the fingers.

b. Systolic murmur. This is heard best over the pulmonic area, and is usually accompanied by a thrill. The pulmonary second sound is usually absent.

c. The heart is usually normal in size but on x-ray examination there is relative right ventricular enlargement, with absence of a normal pulmonary artery shadow. This latter feature gives the picture of a "couer en sabot".

d. The pulmonary artery branches are diminished in size, with unusually clear lung fields, the so-called "anemic" lung.

e. The electrocardiogram will show a pronounced right ventricular predominance.

2. Eisenmenger's complex.

This complex is presented here before discussion of any case reports because of its close associa-

tion to the Tetralogy. In this lesion the defects are exactly the same as in the Tetralogy, except that there is no pulmonary blood flow and pulmonary blood pressure are not reduced. This one feature is the determining factor in the differentiation of these two types of cyanotic congenital heart disease. For this reason the clinician must be able to determine the presence or absence of an elevated blood pressure in the pulmonary artery before recommending surgery for any patient with cyanotic heart disease. The classical features then, of Eisenmenger complex are as follows:

a. Late and less severe cyanosis.

b. Systolic murmur over pulmonary area.

c. Moderate, globular enlargement of the heart.

d. Enlarged pulmonary artery and branches with normal pulmonic 2nd sound.

e. Electrocardiogram shows right axis deviation.

Case Reports

The first case is that of ., a 13 year old female. The history revealed the development of cyanosis at age 5. There was marked clubbing of fingers and toes. A systolic murmur was heard over the pulmonic area and the heart did not appear enlarged to percussion. On x-ray examination there was right ventricular enlargement with decreased pulmonary markings. However, the main pulmonary artery appeared prominent, and this coupled with the latter development of cyanosis gave some doubt as to the diagnosis of Tetralogy of Fallot.

Accordingly this patient was studied by heart catheterization, and the pressure in the pulmonary artery was found to be 8/4 mm. of mercury. When the catheter was drawn back into the right ventricle the pressure was found to be 50/0 mm. of mercury, thus proving the presence of pulmonic stenosis.

A second case is of even more interest. This patient, , a 6 year old male, had a history of moderate cyanosis since age 2. With this there was only moderate clubbing of the fingers and toes, and a coarse systolic murmur and thrill were evident over the pulmonic area. X-ray examination, however, showed a diminished pulmonary artery shadow. The electrocardiogram showed a tendency to left axis deviation. With these findings the clinical impression was one of either Tricuspid stenosis, Tetralogy of Fallot with trilocular heart, or possible Eisenmenger complex.

On catheterization it was found that the pressure in the pulmonary artery was 96/60 mm. of mercury and the pressure in the right ventricle 110/0 mm.Hg. Therefore, it was clearly demonstrated that there was no pulmonic stenosis, and consequently any surgical procedure to increase pulmonic blood flow would be unnecessary.

(VI) Limitations of cardiac catheterization in congenital heart disease.

A. Lesions of the left ventricle and aorta.

1. Coarctation of the aorta. This lesion is unapproachable by means of right heart catheterization, and attempts to catheterize the aorta through the brachial artery are of value only if dyes can be injected near the area of the coarctation. This procedure, however, is not without some danger.

2. Congenital sub-aortic stenosis. For the same reasons as noted above this lesion is beyond the scope of heart catheterization.

3. Rheumatic valvular disease of mitral and aortic valves does not permit specific diagnosis by this method, but the presence of pulmonary hypertension and determination of cardiac output

are easily made in these situations.

B. In a certain number of cases of cyanotic heart disease where the presence or absence of pulmonic stenosis must be clearly established it has been impossible to pass the catheter into the pulmonary artery. While this in itself is presumptive evidence of high grade pulmonic stenosis it can not be considered diagnostic. In these cases, however, through the use of other procedures such as electrokymography, oximeter studies, and angiocardiography, the diagnosis may be established. These procedures, however, will be discussed in a future paper.

C. In other and more varied situations where heart disease of congenital origin is suspected, either cyanotic or acyanotic, it has been impossible to make an accurate diagnosis. These cases include pulmonary arteriovenous fistula, anomalies of the aortic arches, etc.

Case Reports

In the first case, ., a 6 year old male, cyanosis was present since age 4. The clinical findings were consistent with a tetralogy of Fallot, and the X-ray findings suggested decreased pulmonary markings. However, because of the moderate and late development of cyanosis he was subjected to heart catheterization. In our attempts we were unable to pass the catheter into the pulmonary artery, but observed a pressure of 100/0 mm. of mercury in the right ventricle. While this was presumptive evidence of pulmonic stenosis, it nevertheless was an unsatisfactory study and left us with no answer as to the presence or absence of pulmonic stenosis. Further studies with angiocardiography did demonstrate evidence of pulmonary stenosis, however, and gave us further support for the diagnosis of tetralogy of Fallot.

A second case, that of ., an 18 year old male, was seen with the history of onset of cyanosis at age 14. The case was further complicated by the presence of a severe cirrhosis of the liver. Marked clubbing was present, the heart was not enlarged appreciably and appeared normal in contour. With the late onset of cyanosis, the normal appearing pulmonary artery and branches, plus a good pulmonic 2nd sound, a clinical diagnosis of Eisenmenger's complex or possible pulmonary arteriovenous fistula was made. On catheterization, however, an entirely normal pressure was found in both right ventricle and pulmonary artery, and there was no evidence to suggest any shunt.

Further studies with angiocardiography and electrokymography also proved valueless and the cause of his cyanosis is still unexplained.

(VII) Complications of right heart catheterization.

A. That venous catheterization is a benign procedure even in fairly sick patients has been affirmed repeatedly[13,14,15], and our experience with over 100 patients has confirmed this. However, certain minor difficulties have been encountered by us, i.e.,

1. Venous spasm. This has usually been associated with bodily discomfort and apprehension. By the use of novocaine injected every 15 to 20 minutes into the site of incision, and the use of an air mattress and pillows, this complication is usually avoided.

2. Ventricular extrasystoles may be a source of discomfort to the patient. These are produced by contact of the catheter on the ventricular wall. However, if the catheter tip is maintained well within the lumen of the ventricle this is usually avoided.

3. Post catheterization chill and

fever has been noted on rare occasion, and is probably no more common than the incidence of chills and fever following any intravenous fluid administration. At no time have any organisms been cultured from these few patients exhibiting such febrile reactions. Nevertheless, to obviate any possible contamination, we routinely give penicillin pre- and post-operatively, and often include it in our intravenous catheter drip.

(VIII) Summary

A. We believe that cardiac catheterization in congenital heart disease has proved itself to be a valuabl tool in the diagnosis of congenital heart disease. It must be emphasized, however, that the correct diagnosis of congenital heart disease depends on a fundamental knowledge of cardiac physiology, and that it is only in those cases that cannot be successfully diagnosed by the routine methods, that cardiac catheterization need be employed.

A group of cases has been presented in this paper that indicate the real contribution that catheterization can make, for without these studies many of these patients would have been denied surgical procedures, or more unfortunately, would have undergone the risk of unnecessary cardiac surgery.

It is also evident that cardiac catheterization is not without its limitations. As indicated, technical difficulties have often prevented us from advancing the catheter into areas such as the pulmonary artery, and also, lesions of the left side of the heart and of the aorta are beyond the reach of the cardiac catheter as far as accurate diagnosis is concerned.

Certain complications of heart catheterization have been discussed, but it is evident that this procedure is extremely innocuous, and the complications that do occasionally arise, are of a minor and transitory nature.

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Cardiac output in man by Direct Fick Method.
Brit.Heart J., 6:33, '44.

15. Brannon, E. S., Weens, H. S., and Warren, J. V.
Atrial septal defect. Study of hemodynamics by technique of right heart catheterization.
Am.J.M.Sc., 210:480, '45.

## II. MEDICAL SCHOOL NEWS

### Coming Events

January 26 - 28 - Continuation course in Pediatrics for General Physicians.

January 30 - February 11 - Continuation course in Neurology for Internists, Psychiatrists, and Pediatricians.

January 31 - J. B. Johnston Lecture - "Cortical Localization" - Fred A. Mettler, Columbia University; 8:00 p.m., Natural History Museum Auditorium.

February 16 - 18 - Continuation course in Cancer for General Physicians.

February 16 - E. Starr Judd Lecture - "Growth in the Field of Anesthesia" - Henry K. Beecher, Harvard University Medical School; Museum of Natural Science Auditorium, 8:15 p.m.

March 6 - 8 - Continuation course in Gastro-Intestinal Diseases for General Physicians.

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### Continuation Course in Pediatrics

Dr. Franklin Top of the Herman Kiefer Hospital, Detroit, Michigan, will participate in the continuation course in Pediatrics to be presented at the Center for Continuation Study, January 26, 27, and 28.

The course, which is intended for general physicians, will consider dermatological conditions in pediatrics, contagious diseases, and human genetics as related to pediatrics.

Dr. Top will discuss "Immunilogical programs" and "Poliomyelitis - Early Diagnosis and Differential Diagnosis". Dr. J. A. Böök, Head of Medical Genetics, University of Lund, Sweden, will present, "Medical and Genetical Problems in North Sweden". Clinical and full-time members of the University academic staff will make up the remainder of the faculty for the course.

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### Symposium on Hypertension to Honor Drs. Bell, Clawson, and Fahr

A Symposium on Hypertension will be presented on the campus of the University of Minnesota on September 18, 19, and 20, 1950, in honor of Doctors Elexious T. Bell, Benjamin J. Clawson, and George E. Fahr. Doctors Bell and Clawson retired from the Department of Pathology in June, 1949, and Dr. Fahr will retire in June, 1950, from the Department of Medicine.

A Symposium on Hypertension in honor of these men is appropriate since all three have had a special interest in this disease. The symposium will bring to the University distinguished scientists from the United States and abroad. Support for the symposium has been provided by the Variety Club, the Mayo Foundation, and the University of Minnesota. All interested physicians will be welcome.

III. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
CALENDAR OF EVENTS

January 22 - January 28, 1950

No. 274

Sunday, January 22

9:00 - 10:00 Surgery Grand Rounds; Station 22, U. H.

10:30 - 11:00 Surgical Conference; Sympathectomy in Varicose Ulcers; Thomas Murphy; Pm. M-109, U. H.

Monday, January 23

8:00 - Fracture Rounds; A. A. Zierold and Staff; Ward A, Minneapolis General Hospital.

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; M-109, U. H.

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 11:50 Physical Medicine Seminar; E-101, U. H.

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

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

12:00 - 1:00 Physiology Seminar; Eosinophile Responses as Tools in Steroid Hormone Research; Franz Halberg; 214 M. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - 1:20 Pathology Seminar; The Hargraves Cell in Lupus Eryphemotosis; Donald Gleason; 104 I. A.

12:30 - 1:30 Surgery Problem Case Conference; A. A. Zierold, C. Dennis and Staff; Small Classroom, Minneapolis General Hospital.

1:30 - 2:30 Surgery Grand Rounds; A. A. Zierold, C. Dennis and Staff; Minneapolis General Hospital.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Medical-Surgical Conference; Emphysema; Russell Wilson; Bldg. 1, Main Conference Room, Veterans Hospital.

4:00 - Public Health Seminar; Subject to be announced; 113 Medical Sciences.

4:00 - Pediatric Seminar; Chromatography in Modern Research; Mr. Salmon; 6th Floor West, Child Psychiatry, University Hospitals.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; D. Creevy, O. J. Raggenstoss and Staffs; M-109, U. H.

Tuesday, January 24

8:00 - 9:00 Fracture Conference; Auditorium, Ancker Hospital.

8:15 - 9:00 Roentgenology-Surgical-Pathological Conference; Craig Freeman and L. G. Rigler; M-109, U. H.

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

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

10:30 - 11:50 Surgical Pathological Conference; Lyle Hay and E.T. Bell; Veterans Hospital.

12:30 - Pediatric-Surgery Rounds; Drs. Stoesser, Wyatt, Chisholm, McNelson and Dennis; Sta. I, Minneapolis General Hospital.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

1:30 - 2:30 Pediatric Psychiatry Conference; R. A. Jensen and Staff; 6th Floor, West Wing, U. H.

1:00 - 2:30 X-ray Surgery Conference; Auditorium, Ancker Hospital.

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 - 5:00 Physiology-Surgery Conference; Transfusions with Hemolized Blood; Russell Nelson and Clarence Dennis; Eustis Amphitheater.

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

5:00 - 6:00 X-ray Conference; Presentation of Cases by Ancker Hospital Staff; Drs. Aurelius, Peterson and Marshall; Todd Amphitheater, U. H.

5:00 - 6:00 Porphyrin Seminar; C. J. Watson, Samuel Schwartz, et al; Powell Hall Amphitheater.

Wednesday, January 25

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:30 - 9:30 Clinico-Pathological Conference; Auditorium, Ancker Hospital.

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; Veterans Hospital.

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

12:00 - 1:00 Radio-Isotope Seminar; Preliminary Experiments with Tagged Anti-Bodies; J. Friedman; 113 Medical Sciences.

3:30 - 4:30 Journal Club; Surgery Office, Ancker Hospital.

Wednesday, January 25 (Cont.)

4:00 - 5:00 Infectious Disease Rounds; University Hospitals, Todd Amphitheater.

5:00 - 5:50 Urology-Pathological Conference; C. D. Creevy and Staff; E-101, U. H.

Thursday, January 26

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 Classroom, Minneapolis General Hospital.

12:00 - 1:00 Physiological Chemistry Seminar; Free Radicals in Biochemistry; Richard Von Kerff; 214 M. H.

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

2:00 - 3:00 Errors Conference; A. A. Zierold, C. Dennis and Staff; Large Classroom, Minneapolis General Hospital.

4:15 - 5:00 Bacteriology and Immunology Seminar; Immunological Status of the Agent of Chicken Sarcoma I; Mr. Hower Shear; 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; Osteitis Condensans Ilii; Walter Ude; Case Presentations by University Hospital Staff; Todd Amphitheater, U. H.

7:30 - 9:30 Pediatrics Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

Friday, January 26

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; 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, O. S. Wyatt, A. V. Stoesser, and Staffs; Minneapolis General Hospital.

11:45 - 12:50 University of Minnesota Hospitals General Staff Meeting; Experimental Production of Megaloblastic Anemia in Relation to Megaloblastic Anemia in Infancy. Charles D. May: Powell Hall Amphitheater.

Friday, January 27 (Cont.)

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

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

1:00 - 3:00 Pathology-Surgery Conference; Auditorium, Ancker Hospital.

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

3:00 - 4:00 Neuropathology Conference; F. Tichy; Todd Amphitheater, U. H.

3:00 - 6:00 Demonstrations in Cardiovascular Physiology; M. B. Visscher, et al; 301 M. H.

4:00 - 5:00 Clinical Pathological Conference; A. B. Baker; Todd Amphitheater, U. H.

4:15 - 5:15 Electrocardiographic Conference; Right and Left Ventricular Preponderance; Ernst Simonson; 106 Temp. Bldg., Hospital Court, U. H.

5:00 - 6:00 Otolaryngology Seminar; Review of Current Literature; Dr. Wheeler; Discussor, Dr. Boies; Todd Memorial Room, U. H.

Saturday, January 28

7:45 - 8:50 Orthopedics Conference; Wallace H. Cole and Staff; M-109, U. H.

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

8:30 - 9:30 Surgery Conference; Auditorium, Ancker Hospital.

9:00 - 11:30 Neurology Conference; Atrophies and Dystrophies; Powell Hall Amphitheater, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

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

9:00 - 11:30 Surgery-Roentgenology Conference; Todd Amphitheater, U. H.

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 - 12:00 Anatomy Seminar; Hyperchromatism as a Phase of Chromatolysis; Harold Haft; Survey of the Pulvinar; William H. Boyd; 226 I. A.