THE APPRAISAL OF CARDIOVASCULAR STATUS IN INFANCY BY PHYSICAL EXAMINATION

F. MASON SONES, JR., M.D. and R. E. SCHNECKLOTH, M.D.
Department of Cardiorespiratory Disease

ADVANCES in cardiovascular surgery have stimulated interest in the prompt appraisal of the functional significance of congenital cardiovascular defects. The demand for precise anatomic diagnosis has led to the utilization of complex diagnostic methods, such as cardiac catheterization, angiocardiography, fluoroscopy, respiratory gas analysis, and simultaneous precordial lead electrocardiography. Initially, however, recognition of the presence of an anomaly, estimation of its functional importance, and an attempt at accurate localization depends on the patient's history and physical examination. This can be accomplished with some degree of exactness in the case of the more common defects encountered in childhood and adult life. In infancy evaluation of the findings on physical examination has been less well defined, and a correct anatomic diagnosis rarely is established on this basis alone.

The highest mortality rate in congenital heart disease occurs in infancy. One reason for this is that certain defects are so severe as to be incompatible with extra-uterine life. Nevertheless a significant number of deaths occur in infants having anomalies which are either relatively benign, when encountered in older age groups, or are amenable to surgery. In a series of 110 postmortem examinations on children with congenital heart disease who died before the third year of life, Marquis[1] noted that 25 per cent of the total group had ventricular septal defects. Deaths due to severe tetralogy of Fallot or to uncomplicated patent ductus arteriosus in the first year of life are not uncommon.

If the mortality and morbidity of congenital heart disease in infants is to be reduced significantly, an early estimate of the functional status of the cardiovascular system must be made. This can be determined on the basis of symptoms and physical findings by (1) recognition of any existing cardiovascular anomaly at the earliest possible age, and (2) prompt functional classification of the nature and severity of the lesion by evaluation of changes evident in the heart and circulation. This approach will provide a reasonable basis for the prompt institution of medical management, intelligent selection of further diagnostic studies, and timely utilization of available surgical technics.

Symptoms which suggest the presence of a cardiovascular malformation are: tachypnea, persistent cough, frequent recurrence of pulmonary infections, cyanosis, syncopal attacks, stridor, growth failure, vomiting, and edema.

The possibility of such a malformation should be considered in any infant whose resting or sleeping respiratory rate persistently exceeds 60 per minute. Tachypnea appears whenever there is a fixed reduction in the volume of pulmonary blood flow due to the presence of an organic obstruction at the origin

of the pulmonary artery. It is also the earliest manifestation of pulmonary engorgement. The latter indicates a relative increase in the total blood volume in the pulmonary circulation. This condition occurs when there is myocardial failure, a mechanical obstruction to pulmonary venous return, or a large shunt of oxygenated blood into the right heart or pulmonary artery with a consequent significant increase in the volume of pulmonary blood flow.

Persistent cough is often a symptom of pulmonary engorgement which may be erroneously attributed to infection. Superimposed attacks of bronchitis and pneumonitis are common, and are recognized by the appearance of leukocytosis and fever. These infections respond to antibiotic therapy, but cough and tachypnea persist because of the abnormal circulation.

The presence of cyanosis is the best physical evidence of a shunt of systemic venous blood into the systemic arterial circulation. In severe myocardial insufficiency cyanosis may be present without a venous-arterial shunt. When anemia exists it is often impossible to exclude defects which are producing mild, or even moderately severe, systemic arterial oxygen unsaturation. Cyanosis is not visible unless there are 5 Gm. or more of reduced hemoglobin per 100 cc. of circulating arterial blood. If the total hemoglobin amounts to only 10 Gm., there must be more than 50 per cent arterial oxygen unsaturation before even minimal cyanosis appears. Cyanosis is caused by a great variety of defects which cannot be anatomically differentiated on the basis of physical findings. These may be classified however, into two groups on the basis of the presence or absence of pulmonary engorgement. Those presenting evidence of pulmonary engorgement seldom can be improved by operation, but those showing deficient pulmonary circulation usually are operable.

Obstruction to pulmonary blood flow, as seen in pulmonary stenosis, frequently causes syncope and at times convulsions. Less commonly these anoxic attacks occur in association with defects producing extreme pulmonary hypertension. Cyanosis increases during attacks, unless there is anemia, in which case a grayish pallor will be evident. These episodes are so characteristic of a severe obstruction to pulmonary flow that their occurrence justifies the clinical diagnosis of pulmonary stenosis, necessitating prompt definitive adjunctive studies.

Infantile stridor occasionally is caused by anomalies of the aortic arch system producing constriction upon the trachea and esophagus. When no cause can be found, a complete cardiovascular examination is indicated since early surgical correction is necessary in order to avoid prolonged pulmonary complications.

Failure to gain weight is apparent sometimes in cyanotic infants, but is encountered much more frequently in the noncyanotic group, when a large shunt of blood from the left to the right side of the heart results in a decreased systemic blood flow. Vomiting may occur because of partial esophageal obstruction or passive congestion of the viscera. The appearance of edema in infants, when due to congestive failure, usually is made evident by periorbital swelling.

The cardiovascular examination of infants should be an orderly and systematic procedure. Unfortunately, examination of the newborn infant is too frequently conducted in or near the bedlam of the nursery, where precise auscultation is often impossible. In our experience most pathologic heart murmers have not been recognized until the baby is 4 to 6 weeks of age, when the examination usually is conducted under more favorable circumstances. The responsibility of assuring the normalcy of a newborn infant should justify the preparation of a satisfactory environment for examination. The only requirements are quiet, warmth, cleanliness, and adequate daylight. If the examination is done during the first hour after feeding, the problem of persistent crying is minimized. Chilling frequently causes a mottling of the skin, sometimes confused with cyanosis.

While the baby is resting or sleeping the rate and character of respirations and the presence of stridor should be noted along with the state of hydration and nutrition. Cyanosis and any evidence of clubbing of the fingers should be observed. A precordial bulge may be recognized on inspection. Gentle palpation over the precordium will assist in an estimation of heart size by localization of the apical impulse. If a thrill is present, its point of maximum intensity should be determined. A warm stethoscope should be used for auscultation, and sudden changes in its position or heavy pressure upon the chest avoided. Loud breath sounds may prevent precise identification of faint murmurs and abnormal heart sounds. Newborn infants, when at rest, have periods of apnea, which are advantageous if the examiner will wait for and utilize them. The heart rate is counted by auscultation. Careful palpation will reveal the existence of hepatomegaly or intrinsic pulsations in the liver. Abdominal aortic and femoral arterial pulsations should be compared with those of the radial arteries. Auscultation and percussion of the posterior chest is then performed with the infant in the prone position. If cyanosis has not been noted previously, crying should be stimulated, and any color changes in the skin evaluated. When performed in the manner described a satisfactory examination may be accomplished with minimal loss of time.

A systolic heart murmur is the most frequent reason for suspecting cardiovascular disease in infants. During the first 3 or 4 days of life one occasionally hears a grade 2 to 3 systolic murmur in an otherwise normal baby. Taussig[2] has attributed these murmurs to functional patency of the ductus arteriosus. The murmur disappears after a few days with functional closure of the ductus, which precedes complete anatomic obliteration by as long as 3 months. From 5 to 7 days after birth until the age of 6 or 8 months a grade 2 or louder systolic murmur almost invariably signifies a congenital cardiovascular malformation. One is never justified in assuming these murmurs to be physiologic in origin. Actually, because of the difficulties encountered in precise auscultation at this age a natural process of screening takes place and faint murmurs of transient occurrence, which would be classified as physiologic, rarely are recognized. During a study of 630 infants, Cox[3] found only 10 with systolic murmurs at any examination up to the age of 6 months. Of these, murmurs were incon-

stant in 6, suggesting an incidence of physiologic murmurs lower than 1 per cent in the group under 6 months of age. From 6 to 8 months physiologic murmurs appear more frequently and after 2 years are encountered in the majority of preschool children.

In infancy the auscultatory characteristics of a murmur are of little value in locating the defect producing it, or in appraising its functional importance. Almost all murmurs present before the age of 6 months are systolic in time. Because of the small heart size the point of maximum intensity of the murmur is difficult to locate, and the thin chest wall results in nonspecific transmission of the murmur throughout the chest. Systolic murmurs caused by anomalies resulting in cyanosis are indistinguishable from those produced by uncomplicated septal defects of patency of the ductus arteriosus. A septal defect without symptoms or cardiac enlargement may produce a murmur similar to that found in another infant with an identical, but larger defect which has caused functional disability.

Diastolic murmurs are seldom heard during infancy. When audible, however, they are less intense and more readily localized than are systolic murmurs. Of particular importance is a short, late, rough diastolic murmur, most audible medial to the cardiac apex; this is detected in noncyanotic infants with cardiac enlargement and pulmonary engorgement. Its presence has often been associated with the development of myocardial insufficiency in this group of patients. In older infants a blowing, high pitched, early diastolic murmur, loudest in the second or third left interspace, may be due to pulmonary insufficiency in the presence of severe pulmonary hypertension. In a few instances a loud systolic murmur continuing into mid-diastole is encountered in the same area. This murmur is caused by a compensatory shunt of blood to the lungs when there is obstruction to flow at the pulmonary outflow tract, such as a naturally occurring patent ductus arteriosus in the presence of a tetralogy of Fallot. In infants a continuous or "machinery" murmur is never due to an uncomplicated patent ductus arteriosus, since the diastolic pressure difference between the systemic and pulmonary circulation is small. A few severe malformations occur without murmurs, and in these cases extreme cyanosis will direct attention to the cardiovascular system. A truncus arteriosus arising from a single ventricle may be responsible for this clinical picture.

Careful study of the heart sounds provides valuable information. If splitting of the second sound occurs, one may strongly infer that both aortic and pulmonic valves are present and capable of closure. This excludes those cyanotic defects in which one valve is absent or incapable of closure. When both valves are present and functioning, their closure may be synchronous, but careful repeated auscultation usually will reveal at least slight reduplication of the second sound. A third heart sound occurring in early or mid-diastole is heard in normal infants and often is accentuated in the presence of septal defects of sufficient magnitude to produce cardiac enlargement. This is more often the case in auricular than in ventricular septal defects.

In infancy, a bulging sternal deformity usually is evidence of enlargement of the right ventricular outflow tract. When this is perceptible in a cyanotic defect there is usually an associated increase in pulmonary blood flow. Conversely, those anomalies causing cyanosis and decreased pulmonary flow, such as tetralogy of Fallot, ordinarily do not show this specific type of enlargement, and sternal deformity is absent. The exception to this rule is valvular pulmonary stenosis with an intact ventricular septum. In this instance, the pulmonary outflow tract proximal to the pulmonary valve becomes enlarged, and despite a decreased pulmonary blood flow, precordial bulging occurs. Noncyanotic defects accompanied by large shunts of arterial blood into the pulmonary circulation also lead to enlargement of the pulmonary outflow tract, which condition is not caused by identical anatomic defects of smaller size. Thus, in noncyanotic lesions such as auricular and ventricular septal defects, and patency of the ductus arteriosus, one may roughly estimate the functional severity of the lesion by the presence or absence of precordial bulging. Generalized cardiac enlargement without eccentric hypertrophy of the pulmonary outflow tract may be recognized by displacement of the cardiac apex from its normal position in the fourth or fifth interspace at the left midclavicular line. This is estimated more accurately by palpation of the apical impulse than by percussion of cardiac dullness. Dextrocardia is easily recognized by this method.

Auscultation over the hilar areas and posterior lung fields may reveal persistent, fine, crackling inspiratory rales due to pulmonary engorgement. When this occurs in noncyanotic lesions, with an intracardiac left to right shunt, it implies major functional disability. In cyanotic lesions rales are absent if there is a reduced pulmonary blood flow. The significance of such rales can be evaluated only when pulmonary infection or atelectasis are excluded. Pulmonary edema is not observed in infancy except as a terminal complication.

Hepatomegaly and venous distention are early manifestations of congestive failure, and may exist for months before the appearance of dependent edema, ascites, or pleural effusion. Intrinsic or expansile pulsations in the liver may be caused by tricuspid atresia, or pulmonary stenosis with an intact ventricular septum, when there is only a small auricular septal defect allowing escape of venous blood from the right auricle. Anasarca, when due to congestive failure, is usually a terminal event.

The absence of pulsations in the abdominal aorta and femoral arteries makes possible the diagnosis of coarctation of the aorta in early infancy. When this anomaly is uncomplicated, its presence should always be recognized at the time of physical examination. In rare instances, when a patent ductus arteriosus empties its blood from the pulmonary artery into the aorta beyond the occluded segment, femoral pulsations may be felt. In such instances cyanosis usually is present in the lower extremities and absent in the head and arms.

Summary

The early recognition of cardiovascular malformations offers the best opportunity for lowering the mortality and morbidity caused by these defects

in infancy. This is primarily the responsibility of the physician entrusted with the routine examination and care of newborn infants. In this age group it is possible to recognize the presence of a cardiovascular anomaly and to make an estimate of its functional severity on the basis of symptoms and physical findings. The usefulness and limitations of the signs and symptoms most frequently encountered have been presented. Their recognition and evaluation provide the best foundation for medical management, intelligent use of more complex diagnostic methods, and the timely utilization of appropriate surgical technics.

References

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