Family Practice Grand Rounds

Hypertension in Children

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DR. JUNE TUNSTALL *(Third year family practice resident):* The orientation of today's Grand Rounds will be pediatric. I would like to introduce Dr. Peter Viles, who is associate professor of pediatrics and cardiovascular medicine at the University of Massachusetts Medical School, and Dr. Louis Fazen, who is pediatric coordinator at Worcester Hahnemann Hospital. I will present the case and Dr. Viles will give us a differential diagnosis. After his presentation, we would like some discussion about the diagnosis, following which Dr. Fazen will talk about screening for hypertension and current views on the problem of hypertension in children.

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Patrick is a two-month-old infant born at the Memorial Hospital in Worcester, Massachusetts, on December 26, 1976, after a 36-week gestation. Early delivery was prompted by premature rupture of membranes and start of active labor. Delivery was by repeat cesarean section. Apgar scores at birth were given as 7 at 1 minute and 9 at 5 minutes. Birthweight was 2,669 gm. Physical examination at birth was within normal limits except for some nasal flaring.

Within hours after birth, the infant began grunting and required nasal oxygen because of respiratory distress. After a sepsis workup, which included umbilical catheterization, he was started on ampicillin and kanamycin. He responded quite well to this regimen, and within 48 hours was transferred out of the intensive care nursery. The initial sepsis workup was negative.

When the infant was returned to the nursery after his first visit with his mother, he suddenly became mottled with decreased urinary output and

increased urine specific gravity. Arterial blood gases revealed metabolic acidosis with a decreased pH, ranging between 7.12 and 7.20. One pH was as low as 7.01. This was initially thought to be secondary to dehydration and hypovolemia; the infant had lost 250 gm in weight since the previous day. Despite several pushes of Plasmanate and sodium bicarbonate his pH never went above 7.20. A chest x-ray revealed an enlarged heart but clear lung fields. A loud systolic murmur was heard for the first time, but peripheral pulses were good. Blood pressure was 130/100 mmHg. A repeat sepsis workup was done. Complete blood count and platelets were normal, and cerebrospinal fluid was negative. The patient was digitalized and given furosemide 2 to 5 mg intravenously. Antibiotics were changed to oxacillin and gentamicin. The diagnoses offered by a cardiology consultant were a large ventricular septal defect (VSD) or hypoplastic left heart syndrome. It was decided that the patient should be transferred to Massachusetts General Hospital for further evaluation. In the meantime, he was intubated and placed on a respirator, and another umbilical artery catheter was inserted.

During the prenatal period, the mother, a 28-year-old gravida-4, para-2, ab-1, was hospitalized twice because of urinary tract problems. The first admission was at 16 weeks gestation; urine cultures were positive for over 100,000 colonies of Escherichia coli. She was treated with sulfisoxazole and did well until her seventh month, when she developed another urinary tract infection requiring hospitalization. An intravenous pyelogram revealed obstruction of her right kidney, thought to be secondary to pressure on the ureter. She again was placed on sulfa with good results.

DR. PETER VILES *(Associate Professor, Pediatric Cardiologist, Department of Pediatrics):* While I have some idea of the diagnosis, I was not the cardiologist consulted in this case and have not seen the child. One of the pertinent points in the history is that although the child had some distress at first, within hours he did quite well. It wasn't until the second or third day of life that again he had trouble.

It would appear that this second difficulty was characterized by metabolic acidosis, the appearance of a heart murmur, and some cardiomegaly. He was found to have a blood pressure of 130/100 mmHg. If we took blood pressures on newborns more frequently, we would find that elevations in blood pressure are more common than we now believe. As you know, most of the time when newborn blood pressures are taken, the concern is hypotension. Hypertension is a much less common problem.

I would like to set out some guidelines on what constitutes hypertension in the newborn. First, birth weight at postnatal age influences blood pressure. The number of feedings and the total fluid intake have been suggested as factors, but it is not clear that these are independent factors separate from age. Though there has been no statistical difference found between the blood pressure of white newborns and black newborns, there is an epidemiological association with the mother's diastolic pressure. This association is not great enough to predict that an individual infant will be hypertensive. However, if one ranks the maternal cohorts by their diastolic pressure, their infants rank along with them. This familial aggregation of blood pressure is now well known in children, and can be detectable in the neonatal period. One of the most important influences on blood pressure in newborns is the sleep state or the state of activity. The difference between an awake infant and an infant in quiet sleep is around 10 to 15 mm of mercury. If we define hypertension as a blood pressure above two standard deviations from the mean, or above the 95th percentile, then for a sleeping infant to be classified as hypertensive, blood pressure would have to be at least 90/60 mmHg and for an awake infant, at least 105/70 mmHg. So regardless of our patient's activity state, he is indeed hypertensive and rather strikingly so, compared to the normal values.

The possible etiologies may fall into three areas: cardiovascular, renal, and endocrine. Three prominent endocrine factors are hypercalcemia, adrenal-genital syndrome, and adrenal hyperfunction. Some hypercalcemic infants have been described as hypertensive; however, it is unclear whether this could occur in the first week of life. These infants are often characterized by vomiting, poor feeding, and irritability. This does not seem to be Patrick's problem. There is one variety of the adrenal-genital syndrome due to defective 11-hydroxylase, which is associated with hypertension. However, the hypertension usually occurs in midchildhood, not in infancy or the first years of

life. Adrenal hyperplasia, presumably on a central basis, must be very unusual.

Coarctation of the aorta is the only cardiovascular etiology worthy of consideration. If there is coarctation in this case, it would presumably be simple coarctation, unassociated with intercardiac defects. In most children with coarctation and ventricular septal defect, the size of the VSD is enough to allow free communication between the two ventricles. With an acute increase in afterload—that is, increase the aortic pressure or resistance—the only consequence is that more blood will shunt across the VSD. Therefore, hypertension in the upper extremity with possible coarctation implies either that a ventricular septal defect is small or, more likely, that the ventricular septum is intact. Feeling the femoral pulses is important. Because many newborns can keep their ductus open and increase their pulmonary vascular resistance, many with coarctation have reasonably good femoral pulses because essentially the right ventricle is pumping to the lower extremities. However, with carefully taken blood pressures, a difference usually is noted. If this child had a coarctation, he would have had a degree of hypotension in the lower extremities. Palpable femoral pulses do not exclude coarctation, and absent femoral pulses raise the possibility of coarctation. As far as this patient's murmur and cardiomegaly are concerned, the murmur at the apex suggests mitral insufficiency. Anything that would put an acute load on the left ventricle with some degree of left ventricular failure could cause that. Papillary muscle dysfunction has been described in children. Indeed, in one series of children with coarctation, of those who came to autopsy, 30 percent had evidence of myocardial fibrosis and myocardial infarction.[1]

It is obvious, then, that one can compromise the myocardium quite acutely in the newborn with an acute rise in afterload. Given a degree of left ventricular dilatation and mitral insufficiency the heart is going to enlarge. Therefore, the murmur and cardiomegaly could all be secondary to the hypertension and would not necessarily imply primary cardiovascular disease. Occasionally, one sees a child in the early stages of heart failure with vasoconstriction and a mild rise in blood pressure. Usually at that point the pulse has become somewhat weak and thready, although when measured with a doppler it may be elevated. Patrick's pulses are described as good. So this does not seem to be a cardiovascular lesion that just happens to be associated with some mild hypertension. A leg blood pressure would help to resolve that question.

Finally, renal etiologies should be considered. The mother had some renal problem, although there is doubt that this is related. It is possible that the child had a renal arterial embolus or thrombus or, worse, an aortic thrombus secondary to the catheterization done at birth.[2] Of course, there could be renal vascular anomalies on a congenital basis or hypoplastic kidneys with renal vascular hypertension.

Renal causes do seem more likely than cardiovascular or endocrine etiologies. Dr. Tunstall, over to you!

DR. TUNSTALL: Are there any comments from the audience? Any other differential diagnoses anyone would like to make?

RESIDENT: You mentioned a murmur of mitral insufficiency. Could the baby be hypertensive just on the basis of congestive heart failure?

DR. VILES: In early and mild-to-moderate congestive heart failure there is some secondary attempt at maintaining flow and some vasoconstriction and elevation of blood pressure. Usually it is not to the degree found in this child. When looking at an infant with possible congestive heart failure, feeling the pulses and getting a qualitative assessment is of utmost importance. Are they brisk, bounding pulses, or weak pulses? With many infants in failure, the only palpable pulse is in the right arm, usually the axillary, and as one proceeds around the arch and down the lower extremities the pulses become weaker. I am always hesitant to make the diagnosis of coarctation on the basis of reduced femoral pulses in an infant in profound congestive heart failure. Many times when cardiac output improves, the femoral pulses improve. At the lowest level of cardiac status, the only decent pulse may be the one in the right arm.

DR. TUNSTALL: The patient was transferred to Massachusetts General Hospital, and the metabolic acidosis remained. A series of tests, including electrocardiogram and chest x-rays, confirmed what was done at Memorial. An intravenous pyelogram showed poor uptake of dye in both kidneys. Arteriograms revealed dissection of the renal arteries to both kidneys, which was thought to be secondary to the umbilical catheterization. Dr. Viles was right on target here. A nephrectomy

then was done and the patient managed at Massachusetts General Hospital for one month. On discharge, he had gained 2 1/2 pounds and was doing very well. When seen last week, his blood pressure was 90/60 mmHg. He's on hydralazine and hydrochlorothiazide for management, and will be seen at Massachusetts General on a monthly basis. Are there any comments?

DR. JAMES CHAILLET *(Second year family practice resident):* Will he require long-term antihypertensive therapy? And if so, what is the reason for the hypertension?

DR. TUNSTALL: Dr. Viles will be able to answer that. His dosage of hydralazine and hydrochlorothiazide has already been cut,and he is still maintaining a 90/60 mmHg blood pressure.

DR. VILES: This depends on how well the blood flow in the remaining right kidney is improved and maintained. It could represent an extraluminal catheterization entirely from the umbilical artery or it could represent the fact that the tip of the catheter in the aorta got underneath the intima and dissected. Many factors contribute to vascular complications from an umbilical artery catheter. The more the catheter is manipulated, including putting things through it, the higher the incidence of complications. Using it for drawing blood and pressure monitoring is associated with the lowest incidence of complications. It is not known what was given through this infant's catheter.

DR. CHAILLET: How great is the incidence of renal artery thrombosis secondary to catheterization?

DR. VILES: Between five and ten percent of the catheterizations are associated with some clinical manifestation of vascular problem. The incidency in autopsy series ranges as high as 30 percent.[3] It depends on the population discussed. A careful follow-up study is needed for residual problems in infants who have umbilical arterial catheterizations.

DR. TUNSTALL: At Massachusetts General this is supposedly only the third reported case of dissection secondary to catheterization.

DR. LOUIS FAZEN *(Assistant Professor, Department of Family and Community Medicine, University of Massachusetts Medical School; Pediatric Coordinator, Worcester Hahnemann Hospital):* Dr. Tunstall asked me to expand the topic of hypertension to include a discussion of the epidemiology of hypertension in children. Hypertension in children may be of particular interest to physicians who are treating both adults and children. Although the prevalence of essential hypertension is estimated to be as high as 10 to 20 precent of the adult population of this country, the natural history of adult type of essential hypertension beginning in childhood is not well understood. Our present efforts to diagnose and treat hypertension in childhood are severely limited because of lack of knowledge of the natural history of childhood hypertension as it relates to hypertensive adults.

In order to begin to discuss the epidemiology of childhood hypertension, it is necessary to describe the normal limits of blood pressure in children. Two recent publications have considerably improved the description of normal blood pressure values for children. The first report was published in 1973 by the National Health Survey.[4] It covers pressure levels of children from 6 to 11 years of age with particular concern for age, sex, race, and socioeconomic status. More recently, the National Heart, Lung, and Blood Institute's Task Force on Blood Pressure Control in Children reported their findings.[5] Both studies involve standardized cross-sectional blood pressure recordings on large groups of children. The first study examined 7,000 children, and the second one over 11,000. The systolic measurement was considered to be the onset of Korotkoff sounds. The diastolic measurement was the point of muffling of sounds, or the fourth Korotkoff sound. The technique of blood pressure examination is important. The patient should be in a relaxed state, and the blood pressure cuff should cover at least two thirds of the upper arm. It is interesting to note that the auscultation of blood pressure by a stethoscope results in slightly lower values than either the intraarterial or the ultrasonic measurement of blood pressure. Blood pressure measured by palpation was even lower, and blood pressure measured by the flush technique was significantly below the auscultation level.[6]

Prior to the above-mentioned studies, most of the information regarding normal blood pressures for children had been compiled by a pediatrician, as a result of data accumulated in his private practice. In 1966, Sol Londe published normal blood pressure levels for about 1,500 boys and girls from 4 to 15 years of age.[7] Although the sample of patients was limited to his personal practice, the

technique was standardized. His data provided an important step in understanding the variation of blood pressure with age in children. As an aside, I want to emphasize the need for continued office epidemiology as a valuable tool for accumulating important health data. In 1939, William Pickles wrote a small but stimulating book in which he appealed to clinicians to collect simple medical observations in the process of their practice. Pickles believed this would augment their understanding of medical epidemiology and the natural history of disease.[8]

During the remainder of this discussion, I will first speak briefly about the prevalence of hypertension and the etiology of hypertension in children. I will then discuss the common risk factors associated with essential hypertension in both children and adults.

Hypertension in children is usually defined as at least three blood pressure recordings greater than the 95th percentile for age and sex. The prevalence of hypertension in children varies widely in the literature because of differences in the populations selected for study. From numerous reports, approximately one to two percent of children have been found to have elevated blood pressure recordings.[9] The prevalence of hypertension in children is considerably less than the estimated 10 to 20 percent of adults who have persistent hypertension.

The etiology of hypertension in children is subject to considerable debate. Estimates of the proportion of hypertensive children with essential, or unknown etiology, hypertension have been from as low as 6 percent to as high as 93 percent of all children with hypertension.[10] After conducting an extensive medical and radiologic workup in a large medical center, Loggie found that 60 to 80 percent of children do have a specific etiology accounting for their hypertension.[9] In contrast, only 10 to 20 percent of adult hypertensives have a specific etiologic disease causing their hypertension.

What then are the epidemiologic risk factors of hypertension in children compared to adults?

1. Age. Blood pressure definitely increases with age. The greatest increase in blood pressure with age occurs in the systolic recording. The median systolic blood pressure from age 2 to age 18 increases by 25 mm of mercury. The diastolic pressure, however, over the same time period, increases by only about 15 mm of mercury. In adulthood, the systolic blood pressure continues to increase with age. The diastolic also increases with age but, as in childhood, relatively slowly. It should also be noted that there are exceptions to the general rule. For example, certain isolated island populations have been found which do not exhibit increased blood pressure with increased age.

2. Sex. Blood pressure does differ with sex. However, the relationship is not consistent in each age group. The National Health Survey data for 1973[4] revealed that after the age of six, girls have slightly higher systolic pressures than boys. The diastolic pressure was also higher for girls, but to a lesser degree. In general, adult men have higher blood pressure readings than women until they reach 50 to 60 years of age. After that age, women generally have higher blood pressure recordings.

3. Race. Blood pressure definitely differs with race. From the 1973 survey, black children had consistently higher diastolic levels than white children of the same age.[4] Systolic pressures were approximately the same for both races. Data accumulated for adults shows the continuation of the same trend, with black adults exhibiting higher blood pressures than whites of the same age.

4. Genetics. Familial aggregation of elevated blood pressures has been reported both for adults and for children. It has been noted that both siblings and parents of hypertensives are more likely to have hypertension. The influence of genetics has also been observed in twin studies with children with hypertension. However, it is important to point out that the familial relationship of hypertension may be influenced by the common environmental factors experienced by family members, as well as by purely genetic factors.[11,12]

5. Stress and socioeconomic factors. Both these factors are covered extensively in a text by Stamler dealing with the epidemiology of hypertension.[13] At present, there is only circumstantial evidence implicating stress and socioeconomic

factors as important etiologic agents for essential hypertension in either adults or children.

6. Obesity and Diet. Obesity is implicated in almost all studies conducted on children with hypertension. More elaborate studies on adults with essential hypertension repeatedly demonstrate the correlation between hypertension and excessive weight gain. Obesity appears to be a solid risk factor for increased blood pressure in both children and adults. It has been shown that the association between blood pressure and body weight is a real association and is independent of the arm circumference.[14] However, the relationship of diet, and in particular salt consumption, to hypertension is not as clearly understood. A recent summary of work in this area by the American Academy of Pediatrics Committee on Nutrition concluded that salt intake is probably only one of the many factors responsible for essential hypertension.[15] Although the committee recognizes salt as a relatively minor factor in hypertension, they recommended decreasing the amount of salt added to baby food products since many children and most adults greatly exceed the minimum salt intake required for normal growth and development. Additional information on the epidemiology of hypertension in adults and a review of the literature on hypertension has been presented by Kessner and Kalk in a study for the Institute of Medicine.[16]

Finally, the crucial question remains: Are children with elevated blood pressures above the 95th percentile for age destined to become hypertensive adults? Unfortunately, there has not been a sufficient number of published long-term follow-up studies of children with essential hypertension. However, Stamler studied 61 young adults and followed their hypertensive disease for 20 years. He found that the risk of these young adults was twice as high as that of the normal population.[13] In a separate study, Heyden followed 30 untreated young adult hypertensives for seven years.[17] Forty percent of the hypertensive patients became normotensives at the end of the study, as compared to 90 percent of the matched controls. Almost one third of the untreated patients experienced significant hypertensive disease, as compared to none of the controls.

Given the incomplete data for the epidemiology of hypertension in children, and combining that with the known facts for hypertension in adults, how can the primary care physician develop a rational plan for taking blood pressures in children? Until more information becomes available, the following suggestions seem applicable.

1. Blood pressures should be recorded regularly on children with the known risk factors of hypertension. This means blood pressures should be taken regularly on children with hypertensive siblings or parents, and on all obese children. If sustained elevated blood pressures above the 95th percentile are recorded in a child with risk factors for hypertensive disease, the patient should be watched very closely. One may seriously consider beginning long-term antihypertensive treatment.

2. Blood pressures should be conducted routinely on all children admitted to a hospital and on all children who are seen in the office because of acute or chronic illess. Blood pressures should be included as part of the vital signs on any child evaluated for an illess. In this context, the blood pressure measurement may be of diagnostic value in the medical assessment of a sick child. Loggie has described numerous causes of curable and incurable hypertension in children.[18]

3. The Task Force on Blood Pressure Control in Children recommends that you take blood pressures on all children entering your office. However, without understanding the natural history of hypertension in asymptomatic children, it is difficult to understand and interpret the results of routine blood pressures on normal children. It is particularly difficult to decide how to approach the child with borderline or slightly elevated blood pressure recordings who is otherwise healthy and does not have risk factors of hypertension. In my opinion, recording blood pressures on healthy children without risk factors may actually result in overdiagnosis and in excessive workups for hypertension, with subsequent inappropriate labeling and possible overtreatment of hypertension in childhood. In a recent editorial, North accentuated that routinely measuring blood pressure in healthy children would result in a heavy financial

cost to society and would offer no known benefit.[19]

4. The implementation of blood pressure recordings as a screening test in childhood is not indicated. First, it is more likely that curable forms of hypertension would be detected as part of the clinical manifestation of the underlying disease process than through a screening program. Secondly, screening healthy children for the essential type of hypertension is also unwarranted because we do not understand the natural history of essential hypertension well enough. We do not know which children will ultimately become hypertensive adults and thereby benefit from early diagnosis and treatment.

5. Further research efforts should be conducted to delineate the natural history of essential hypertension in childhood. Following the approach of Dr. Pickles, I think there is a need for clinicians to gather information about hypertension in their office practices. In particular, those of you in family practice could begin to gather data by utilizing families as the basic unit of blood pressure and following the family blood pressure curve through time. Part of the lack of information concerning the natural history of hypertension in childhood is that the disease has been studied either in adults or in children. Very few studies have looked at the continuum of the disease from childhood to adulthood. You in family practice have the opportunity to study the family unit of blood pressure. This may be particularly important in illustrating the natural history of essential hypertension.

Dr. Viles will now discuss the medical workup of the child who does have hypertension.

DR. VILES: My comments are based mainly on the literature, not my personal experience. Loggie's data would indicate that around 55 percent of her population base has primary hypertension.[18,20] Londe puts the percentage at 95. The confusion seems to come from the populations that have been studied. Certainly as more screening and looking at healthy populations occurs, we are going to identify the asymptomatic base of children with a primary elevation of blood pressure. Londe has a fine article in the American Journal of Cardiology on his approach.[21] He merely makes note of the blood pressures between the 90th and 95th percentiles. He calls this a suspect level and does not mention it to the family. If there is definite elevation of blood pressure, above the 95th percentile for age and sex, he repeats the measurement three or four times on a monthly basis. In his experience, one half of the children will have one normal blood pressure in that period of time. A procedural question arises: how many times should you take the blood pressure? Using Londe's guidelines, if the first measurement is normal, there is nothing to be gained by repeating it. If the systolic value is elevated, repeat it three times. Again, often one of those readings will be lower, and would be considered a labile pressure. It is not known whether that patient is at greater risk of developing hypertension in 10 or 20 years. Diastolic elevations rarely fall with repeated measurements, which points out the consistency of diastolic blood pressure. To summarize, if the systolic pressure is elevated, repeat the measurement; if the diastolic pressure is elevated, it probably is a true value. If all the readings are elevated over three or four months, the patient presumably is hypertensive.

What sort of workup is indicated? In addition to physician examination, family history is important. Remember that in Londe's data, 50 percent of the parents of his hypertensives are, themselves, hypertensive, and five percent of the siblings are hypertensive. The family physician is in a particularly good situation because he has access to that data. A urinalysis is necessary, and urine culture and weight measurement advisable. Fifty-five percent of Londe's children are "obese," although he does not define that term. A chest x-ray and an electrocardiogram are also indicated. The electrocardiogram is helpful in assessing the past duration of the patient's hypertension and the usual level of pressure. However, it is difficult to diagnose a left ventricular hypertrophy in children from an electrocardiogram. Yet we have certainly seen a number of children who have mild or moderate elevation of blood pressure and in whom the electrocardiogram shows high voltage and flatter-than-normal T waves over the left precordium. Also indicated is some simple test of the renal function, whether it be a blood urea nitrogen or a more formal creatinine clearance. More extensive workups probably are not indi-

cated unless there is a specific pointer to a specific organ system. Loggie indicates that in her experience children under ten years of age and white teenage girls have a higher incidence of secondary causes of hypertension; she selects those patients out for more extensive investigation. If there is marked elevation in diastolic pressure—above 110 mm of mercury—there usually is some renal etiology.

Data are just beginning to be collected on treatment: its necessity, effectiveness, and side effects. Weight reduction and some limitation of sodium intake is desirable. The dosage schedule of drugs, if used, should be kept simple. Drugs that need to be given frequently—that is, three or four times a day—are often difficult for children to take during school. So, drugs should be selected with this in mind rather than based on the ideal drug in another setting. The long-term side effects of drug treatment are not known. Loggie has presented data on a very small group of children receiving hypertensive medication who were followed for several years, and their weight and height growth has been normal. The long-term effects vs the many possible benefits of hypertensive medication are not known. Medications used by children are no different than those used by an adult.

Any questions or comments?

DR. FAZEN: What is being done now at your health center about blood pressures in children? When you see families, are you checking for blood pressure in children whose parents have elevated blood pressure?

DR. TUNSTALL: No, at our health center, it is not done routinely. Pediatric cuffs are not always available.

DR. VILES: I think the very small cuffs are not very good. It is like putting a rubber band on the child's arm, and I don't think you get very accurate levels. At what age should you start taking children's blood pressures? Londe has data down to age three. More of his cases come between the fifth and sixth years than practically any other time. I think if you are going to start consistently measuring blood pressure in children you really need to start in the preschool years. For that age you can get rather simple blood pressure cuffs that are of adequate size. Under age two or three, I am not sure that screening blood pressure in a healthy child is going to be terribly productive. Other routine screenings, such as urinalysis, will have a high chance of picking up those with renal problems.

DR. TUNSTALL: Our time is up. Thank you Dr. Viles and Dr. Fazen for your presentations.

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