

UCLA
Nutrition Bytes

Title

Nutritional Management of Children with Congenital Heart Disease

Permalink

<https://escholarship.org/uc/item/6jh767ps>

Journal

Nutrition Bytes, 8(2)

ISSN

1548-4327

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Publication Date

2002

Peer reviewed

Introduction

Congenital heart disease (CHD) refers to any anatomic defect in the heart or major blood vessels that is present in children at birth. CHD occurs in approximately 1% of live births in developed countries. Infants and children with CHD exhibit a range of delays in weight gain and growth. In some instances the delay can be relatively mild, whereas in other cases, the failure to thrive can result in permanent physical or developmental impairment (1,2). While the cause of abnormal growth and development is multifactorial, reduced energy consumption and increased energy expenditure, or both, may be the most important players (1,2,3). Aggressive feeding strategies must be employed early with these children in order to prevent permanent growth disturbances. This paper will discuss the recent research into the mechanisms by which congenital cardiac abnormalities cause growth retardation as well as strategies to help these children achieve normal weight.

Causes of Growth Disturbances

Decreased Energy Intake:

Inadequate caloric intake has been shown to be the most important cause of growth disturbances in children with CHD (4). Hansen and Dorup (5) showed that caloric intake in CHD patients was 76% that of normal age matched controls. When calories consumed are viewed in relation to actual body weight, intake was 88% of that recommended by the FAO/WHO/UNU. However, this last statistic may be inflated since the children are likely to be underweight to begin with, therefore a diet based on actual weight may underestimate caloric needs. Rather the diet should be based on the normal intake for children of the same age.

Several possibilities have been proposed to explain the decreased intake in these patients including fatigue upon feeding and malabsorption (3,4). Chronic hypoxia leads to both dyspnea and tachypnea during feeding causing the child to tire easily and thus reduce the quantity of food consumed. Furthermore, since anorexia is a recognized symptom of cardiac failure in adults, it is reasonable to think that this may also be a mechanism of reduced intake for children with CHD (6). Malabsorption is sometimes seen in children with CHD due to intestinal dysfunction from reduced blood flow to and from the splanchnic circulation. This suggests that even children with CHD whose caloric intake is normal for their age may not be receiving enough calories to achieve normal weight. While there is still considerable controversy regarding the role of malabsorption in growth disturbances (if any), this mechanism should be considered until further research is presented (3,4,6).

One recent study has shown that glucose may be the most important component of the diet with respect to calories because disorders of carbohydrate metabolism are commonly seen in patients with CHD (9). These patients were found to have lower fasting glucose levels ($p=0.025$) and elevated insulin secretion rates. Although the reason for this is not clear, it could be related to higher levels of circulating catecholamines or a switch from fatty acid β -oxidation to glycolytic metabolism, which is inefficient and uses more of the

available glucose (6). This raises the possibility that CHD patients are chronically hypoglycemic which could contribute to the fatigue they experience while feeding.

Increased Energy Expenditure:

Infants with failure to thrive due to ventricular septal defects (VSD) have been shown to have a 40% elevation in total energy expenditure (TEE). Surprisingly, resting energy expenditure (REE) was found to be the same between control and VSD children. The difference between REE and TEE was 2.5 times greater in the VSD group than control indicating that their energy during activity is much greater (7). In patients with a VSD there is significant mixing of venous and arterial blood reducing the arterial oxygen saturation. While at rest, this is not a problem and therefore the REE does not increase. However when active, they are not able to deliver enough oxygen to their tissues and must switch to anaerobic metabolism which is inefficient and causes increased energy expenditure.

Children with CHD in which there is congestive heart failure or an increase in afterload (coarctation of the aorta or pulmonary hypertension) often present with increased REE (3,6,8). This is because the heart must work much harder in order to pump an adequate amount of blood against a greater opposing force. In contrast to VSD, this type of lesion leads to a more inefficient use of energy at all times including rest and therefore the REE is elevated.

Another reason for the increased metabolic rate seen in children with CHD is due to their body composition. Due to decreased caloric intake and greater energy expenditure, they have less energy available for fat deposition. As a result, they have an elevated percentage of lean body mass which tends to increase their basal metabolic rate (8). In this case, an increased metabolic rate is causing a further increase in metabolic rate, which if left untreated can dramatically worsen the child's overall health status.

Treatment of Growth Disturbances

Structured Feeding Programs:

Due to the fact that inadequate caloric intake is the leading cause of growth disturbance in this population, it seems logical that increasing the number of calories consumed would have a positive effect on growth and development. Unger, et al., showed that dietary intervention including nutritional analysis and counseling increased mean intake from 90% to 104% of the RDA for calories and increased weight from 83.1% to 88.3% of ideal body weight (10). The fact that counseling alone was enough to increase the caloric intake and weight in these children suggests that there is a definite role for a parental education from a dietitian to optimize feeding.

While some children may be able to benefit from nutritional counseling alone, other children are incapable of consuming larger quantities of food due to the high energy costs of eating. In order to overcome this, Jackson and Poskitt have proposed supplementing

formula or breast milk with glucose polymers (11). The authors increased mean energy intake by 31.7% resulting in a weight gain improvement from 1.3 g/kg/day in controls to 5.8 g/kg/day with high energy feeding. However, there are problems with this method. Another study found that feeding malnourished children high-energy formula may stimulate greater diet induced thermogenesis and increase metabolic inefficiency canceling some of the positive effect (8). Furthermore, these children may not be able to tolerate the concentrated glucose needed to allow for weight catch up and should be monitored closely when on such a feeding program.

Despite the most aggressive feeding programs, some children are still unable to ingest enough calories in order to achieve or maintain a normal body weight. In these patients, it may be advisable to provide enteral (direct gastric) nutritional support through a continuous food pump. One study has shown that percutaneous endoscopic gastrostomy (PEG) is a safe method to deliver calories to children who are severely malnourished due to CHD (12). This method of feeding has the advantage that the child does not need to expend any energy to feed and therefore more is available for growth. In another study it was shown that nutritional treatment with PEG could be used to prevent growth disturbances in newborns with CHD (13). In this study of 13 patients, 12 had normal growth rates for their age. This is far above the normal growth rate for children with CHD on traditional feeding programs suggesting that prophylactic enteral nutrition may be indicated in patients with CHD even before growth disturbances become apparent (12,13).

Breast vs. Bottle Feeding:

The decision of whether to breast or bottle feed an infant is very important for both the child and parents. Most people are aware that when possible, breast feeding is best because it helps foster the bond between mother and child as well as providing the best source of nutrients. However, many parents feel that breast feeding may be too difficult for children with CHD and instead, opt to bottle feed either with breast milk or formula. Furthermore, many parents believe that by bottle feeding they can control the flow rate and therefore deliver more volume with less effort. Surprisingly however, Marino, et al., have shown that oxygen saturations in infants with CHD were lower in the group that was bottle feeding than the group that was breast feeding ($p < 0.0001$) indicating that it is less stressful for the infant to breast feed than bottle feed (14). It is important to note, however, that both the CHD patients with breast and bottle feeding had a lower oxygen saturation than either group of control infants suggesting that the main cause of food refusal is cardiac. Nevertheless, it is important to consider a potential contribution of non-cardiac factors such as parent-child interaction during feeding. Clemente, et al., showed that mothers of infants with CHD experienced more anxiety during feeding than mothers of control infants which could negatively affect feeding (15). For this reason, it is very important that mothers of children with CHD receive additional support and education in breast feeding techniques to reduce stress and increase success.

One potential problem with breast feeding exclusively in children with CHD is that it contradicts other studies that show increased weight gain in children on high energy

supplemented breast milk or formula (11). The answer to this question of breast or bottle feeding is not simple and depends heavily on the type of lesion the child suffers from. In infants with cyanotic lesions, it is most important to keep their oxygen saturation as high as possible and therefore it may be necessary to breast feed at the expense of energy needs. Conversely, in children with acyanotic lesions it may be perfectly acceptable to bottle feed as this will have little adverse effect on oxygen saturation. In summary, the strategy used to feed infants with CHD is case dependent and therefore requires the cooperation of parents, pediatricians and other health care professionals to develop the best approach.

Surgical Correction:

Most children with CHD eventually require surgical correction of their defect. In most cases, surgical correction is performed as soon as possible after the child reaches ideal weight. When infants fail to thrive despite an aggressive nutritional program, it may be advisable to operate despite the increased mortality and morbidity associated with cardiac surgery in underweight infants (6). Two recent studies have shown that surgical correction of the defect lowered energy expenditures in the children, one of the main causes of failure to thrive. Mitchell, et al., (16), showed that one week after surgical correction there was a significant ($p < .001$) reduction in energy expenditure compared to preoperative values as well as values for control children not undergoing surgery. This shows that it may be advisable to operate on children that are not at ideal weight after all other measures have been exhausted. In another study (17), the authors showed that five years after surgery, energy expenditure (REE and TEE) is normal compared to a control group. This indicates that surgical correction is a cure for failure to thrive secondary to CHD and should be done as soon as possible in order to prevent permanent damage from malnutrition.

Conclusion

Failure to thrive is well recognized as a serious problem in children with CHD. In caring for these children, it is important to develop a nutritional strategy that takes into account all of the factors at play, both physical and psychological. Due to the differences between congenital cardiac lesions and the mechanisms by which they retard growth, no single strategy will be adequate to treat all cases. Therefore, there must be a concerted effort between parents, physicians, nurses and other healthcare professionals to develop a plan that will be appropriate on an individual basis. However, in order for this to be accomplished, further research is needed to better identify specific patterns of growth failure as well as to identify the most effective interventions to treat different causes of growth failure.

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