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posed to the last remaining whooping cranes by continued Air Force bombing practice near nesting sites on Matagorda Island. Since that time additional information has become available that indicates the seriousness of the problem.

Matagorda Island is used for bombing practice by the Air Force. It went through a period of little use during the 1960's at the time of our involvement in Vietnam. With the return of our aircraft, however, bombing activities have been stepped up.

The Air Force uses Matagorda for both high- and low-altitude bombing practice with a variety of aircraft including the giant B–52's, FB–111's, Huey Cobra helicopters, F–4's, and A–7's. Ordnance ranges from 250-pound practice bombs to 1,000-pound live drops, plus 20-millimeter cannon and grenade launcher static firing. About 450 Army helicopter sorties are made a month in addition to the thousands of fixed-wing bombing runs. There is laser bombing practice, aerial gunnery, dive bombing, and rocket practice.

Some of these missions have been supersonic as indicated by the operational orders for using the range. Thus, there are literally thousands of airspace intrusions through and over the island yearly, some with accompanying sonic booms. Even given the suspect Air Force figures for bombing accuracy of 98 percent, the possibility for misses outside the range increases with the number of missions flown. It can be expected that there will be numerous bombing errors every year which could result in live ordnance being dropped on or near nesting whooping cranes. The cranes have been sighted in the restricted bombing zone. It can be concluded that continued Air Force bombing will present a great danger to these rare birds.

AIR FORCE WANTS TO RETAIN EXCLUSIVE HUNTING AND FISHING RESORT

Perhaps the real reason why the Air Force wants to retain Matagorda Island lies in its special status as a hunting and fishing resort for military personnel. The Air Force has admitted that 68 generals, 30 high-ranking civilians, and hundreds of other military personnel have used the island as an exclusive hunting and fishing preserve for years. There are 1,245 quail shelters and 38 turkey roosts built by the Air Force for the hunting pleasure of its guests. A number of Air Force personnel are available as guides and aides to the visiting dignitaries that stay at a distinguished visitors' quarters with de luxe rooms, a lounge, dining area, and a chef. They also have the use of some 50 vehicles for transportation to and from their selected recreational entertainment.

So that the day ends right, there is a facility for cleaning and dressing the game. All in all it is a pretty nice operation at taxpayers' expense. Of course the taxpayers cannot use it. It is for generals and such.

Mr. President, I have written to the Air Force asking for the names of all the generals/admirals and high-ranking civilians using the island's recreational benefits over the past 5 years. I have also asked for a full justification for the hunting and fishing facilities on the island and a breakdown of how many Air Force men have been used for these purposes. Should it become necessary, I will ask the General Accounting Office to document the expense and manpower allotted to this project. First, however, I would like to review the Air Force reply.

In the meantime, I hope that the Secretary of Defense will make every effort to place a large portion of the island off limits to recreational use by the military, restrict flights over or near nesting areas, and turn control of the island over to the Department of the Interior.

SUDDEN INFANT DEATH SYNDROME

Mr. MONDALE. Mr. President, on December 11, the Senate passed S. 1745, the Sudden Infant Death Syndrome Act of 1973.

For nearly 2 years I have had a deep interest in the tragedy of crib death or sudden infant death syndrome. One of the most frustrating aspects of this disease is our inability to predict it or to prevent it from striking thousands of babies who give every appearance of being healthy.

It is very gratifying to note the growth in both public and professional interest in SIDS. I am pleased that the Senate has passed and the House is considering the legislation needed to counsel families who lose children and to mount a major medical research effort.

There are dedicated researchers who have been working for years, on limited funds, on clues to the cause of SIDS. The November 29 issue of the New England Journal of Medicine includes several articles describing the status of some of this research. I ask unanimous consent that these articles be printed in the RECORD.

There being no objection, the articles were ordered to be printed in the RECORD, as follows:

SUDDEN, UNEXPECTED AND UNEXPLAINED DEATH IN INFANCY—A STATUS REPORT—1973

In 1953 the subject of sudden death in infancy lay dormant. Little related research was under way, and pertinent scientific publications were few. Twenty years later, on the contrary, there are numerous relevant articles and myriads of applications for the support of research in this field. Multiple factors participated in this change, not the least of which was the strength of organized parent groups. Physicians and governmental representatives have become aware and are taking appropriate action. The National Institute of Child Health and Human Development has responded with a comprehensive program for the stimulation and support of research and the education of physicians and of the laity.

Sudden, unexpected death in infancy (crib death) is currently defined as "the sudden death of an infant or young child, unexpected by history, in which a thorough postmortem examination fails to demonstrate an adequate cause for death." It is essential that this definition be strictly adhered to in any studies dealing with this subject.

Approximately 10,000 crib deaths occur annually in the United States, the greatest incidence being in infants one to four months of age. This is the leading cause of death among infants between one week and one year of age. It is more apt to occur during the late winter and early spring, may be preceded by a mild respiratory infection, and is more common in socio-economically deprived areas than in middle-class and upper-class communities. Perhaps as a corollary to the latter, there is a higher rate of occurrence among infants of low birth weight, and among those born to mothers less than 20 years old, to mothers who have had little or no prenatal care and to unwed or addicted mothers. In this country the lowest rate of occurrence is among Orientals, followed by whites, Mexican-Americans, blacks and finally, American Indians; the explanation for these apparent racial differences is not known.

An infant who suddenly, silently and without struggle, stops breathing, and becomes cyanotic and limp, and who is subsequently resuscitated has been called a "near-miss." Each such infant who is brought to an emergency room presents a real problem, since among the few who are discharged, a certain proportion are brought back dead within a week or so. In the infants admitted to the hospital, studies are almost always noncontributory. Should all such infants be admitted and monitored, or should their families monitor them? To date, there are no ready answers to these questions.

Most of these infants die at night during sleep. Perhaps the most important observation in recent years is that of Steinschneider, who has found that normal infants two to three months old tend to have apneic periods during sleep, and that these episodes are more prolonged during upper respiratory infections. He has, moreover, documented recurrent periods of spontaneous prolonged apnea in infants who subsequently died suddenly and unexpectedly and in whom autopsy failed to reveal a cause of death.

Naeye, elsewhere in this issue of the *Journal*, has demonstrated significant thickening of the walls of small pulmonary arteries in these infants as compared to controls. These changes are similar to those present in normal infants and children living at high altitude and suggest to him the effects of chronic hypoxia. Perhaps the changes are related to the aforementioned prolonged periods of apnea.

The observation that cessation of respiration can be induced in very young monkeys by a variety of stimuli may be related to the above findings, and perhaps this phenomenon is simply a reversion to the fetal state.

Hypotheses other than those related to spontaneous apneic episodes during sleep have recently been suggested but remain unproved. Certain histologic features of the myocardial conduction system, observed in infants dying suddenly and unexpectedly as well as in controls have been implicated as the cause of these deaths. Our study has failed to validate such a conclusion.

An assortment of common viruses has been recovered at autopsy from various anatomic sites in 23 to 42 per cent of these infants. Except for the upper airway, however, histologic sections give no morphologic evidence of infection. There is no evidence of overwhelming viremia. If viruses participate in causing these deaths the exact mechanisms by means of which they do so is unkown.

A number of immunologic mechanisms have been suggested as being responsible for these deaths. Anaphylaxis seems unlikely since serum IgE is apparently normal, and the third component of complement is not depressed. IgM, variously reported as elevated or normal at autopsy, has been found retrospectively to be normal in umbilical-cord blood. Specific antibody titers to common viruses are within normal limits. Failure to demonstrate antinuclear factor or anti-antibodies suggests that immunologic mechanisms play no part in these deaths, but further studies are necessary.

It has been suggested that these infants die because they are obligatory nose-

breathers; occlusion of the nasal passages, as by a respiratory infection, might then be responsible for suffocation. Against this theory, however, are the facts that obligatory nose-breathing characterizes the neonate rather than the infant of three months, and that post-mortem radiologic studies have demonstrated clear passageways.

There is as yet no substantial proof that laryngospasm is responsible for these deaths, nor lack of vitamin E or selenium; compressive hemorrhage about the cervical spinal cord has been disproved. Neither a genetic nor ametabolic defect has been substantiated, and hypogammaglobulinemia, parathyroid and adrenal insufficiency, hypocalcemia and lack of potassium or calcium in the myocardium have been excluded; magnesium deprivation probably is not responsible.

For the practicing physician, currently the single most important aspect of the matter is the care of the stricken family. Unfortunately, in many areas that care is far less than optimal. Infanticide is often suspected, and the parents are treated as though guilty until proved otherwise. This, of course, simply enhances their normal feelings of guilt. It is incumbent upon all of us to do whatever we can to correct this unfortunate state of affairs.

St. Christopher's Hospital for Children, Philadelphia, Pa. 19133.

MARIE A. VALDES-DAPENA, M.D.

THE MYOCARDIAL CONDUCTION SYSTEM IN SUDDEN DEATH IN INFANCY

(By Marie A. Valdes-Dapena, M.D., Marguerite Greene, M.D., Nirmala Basavanand, M.D., Robert Catheran, M.D., and Raymond C. Truex, Ph.D.)

The ultimate mechanism responsible for sudden unexpected death in infancy remains unknown. Current hypotheses tend toward the concept of an instantaneous interruption in some basic physiologic function such as the control of respiration or cardiac action.

In 1968 James proposed the hypothesis that these deaths are due to lethal disturbances in the conduction system triggered by focal "histopathologic changes" in the atrioventricular bundle and the atrioventricular node. The histologic features that he described are focal resorptive degeneration of conduction tissue cells, cell death, removal of dead cells by macrophages and replacement with collagen produced by young fibroblasts. These abnormalities were observed not only in the hearts of 40 infants whose deaths were unexplained after autopsy but also in the hearts of 16 whose deaths were otherwise explained. Thus, James concluded that these microscopical changes constituted part of a "molding" process to be expected in the conduction system in all infants during the first year of life.

Although he conceived of the process as a kind of physiologic focal death and resorption he reasoned that the active lesion might in certain cases instigate sudden malfunction of the system and thus be responsible for sudden, unexpected death.

The observations and conclusions of James were later reaffirmed by Anderson et al. Still later Ferris suggested that the presence of "cartilage" in the anulus fibrosus, which he had seen in two instances, could also initiate fatal conduction disturbances.

The purpose of this investigation was the re-examination of the histologic characteristics of the atrioventricular node and bundle in infants who had died suddenly and unexpectedly and in age-matched controls whose deaths were explained after autopsy.

From the departments of Pediatrics, Pathology and Anatomy, St. Christopher's Hospital for Children, Temple University School of Medicine, and the Division of the Medical Examiner, Department of Public Health (address reprint requests to Dr. Valdes-Dapena at the Department of Pathology, St. Christopher's Hospital for Children, 2600 N. Lawrence St,, Philadelphia, Pa. 19133).

Supported by a grant (1 ROI ND05063-01) from the United States Department of Health, Education, and Welfare, Public Health Service, National Institute of Child Health and Human Development.

MATERIAL

The conduction system was removed in block from the hearts of 47 infants who had died during the first year of life. Autopsies were conducted at Temple University School of Medicine, St. Christophers Hospital for Children and the Office of the Medical Examiner of the City of Philadelphia. Thirty-one of the infants died suddenly and unexpectedly, and their deaths remained unexplained after the performance of a complete autopsy including the examination of numerous microscopical sections. The 16 infants who served as controls died of the following conditions: bronchopneumonia (four); sepsis (three); pulmonary hyaline-membrane disease (two); and one each of coalitis secondary to aganglionosis, peritonitis secondary to atresia of bowel, diarrhea and dehydration, renal-vein thrombosis, methemoglobinemia, angiomatosis and prematurity (birth weight of 735 g). Of the 31 sudden deaths, 18 were in male and 13 in female infants; 23 infants were black, seven white, and one oriental. As for the 16 controls, there were nine females and seven males, 12 blacks, three whites and one Puerto Rican. In both groups the majority of infants were between one and four months of age, the youngest being a premature newborn (weighing 735 g) and the oldest 10 months of age.

At autopsy the entire heart was fixed in 10 per cent formalin after the removal of a few peripheral sections. Thereafter, a single block was resected and trimmed to include the atrioventricular node, the bundle of His and the upper portions of the left and right bundle branches. All four faces of the block were diagrammed and photographed for purposes of orientation during examination of the sections.

Each block was embedded in paraffin and sectioned serially in the coronal plane, the sections being 10 μ in thickness. The average number of sections cut was 1500. All sections were mounted individually on glass slides, and every fifth section was stained with hematoxylin, phloxine, and saffranin. Intervening sections were employed, as indicated, for special stains, including Masson's trichrome, Weigert's methenamine silver, periodic acid-Schiff and Van Gieson.

Sections were studied by two examiners without knowledge of the nature of the case. The histologic architecture of the conduction system was diagrammed in detail in each case.

OBSERVATIONS

In all infants the fibers of the conduction system were supported by a "skeleton" of more or less loosely woven connective tissue. Special stains revealed most of the fibrils in these more lightly stained areas to be collagen fibers with only a few randomly scattered elastic fibers. In these loosely woven areas no dead or degenerating cells could be identified. There was no infiltration by macrophages, nor was there any active "replacement" by young fibroblasts.

Petechiae were encountered within the conduction system in 11 hearts, eight of the 31 crib deaths (26 per cent) and three of the 16 controls (20 per cent). The difference is not statistically significant. The more frequent occurrence in the former group is paralleled by the number of minute hemorrhages in nearby myocardium (50 per cent of the sudden deaths and 37 per cent of the controls).

The tissue of the annulus fibrosus in many exhibited a cartilagenoid character: no true cartilage, however, was observed in any.

DISCUSSION

Clearly the discrepancy between this study and that of James stems from differences in interpretation of histologic observations. He saw focal, loosely woven, palestaining areas as part of an active process capable of stimulating the conduction tissue to dysrhythmia; we saw, in the same areas, no dead cells, no phagocytosis, no replacement fibrosis, nor any evidence of an active process that could be considered part of rapid remodeling. Loosely woven, pale-staining connective tissue is one of the characteristics of supporting structures in the infant in many sites; on the other hand, it is unusual in adults. Its appearance in the growing conduction system of the infant heart is thus not surprising; we question the validity of attributing malfunction of this or any other anatomic system to features of its normal developmental histology.

We are indebted to Mrs. Dorothy Johnson for the technical preparation of our histologic sections.

PULMONARY ARTERIAL ABNORMALITIES IN THE SUDDEN-INFANT-DEATH SYNDROME

(By Richard L. Naeye, M.D.)

ABSTRACT

The sudden-infant-death syndrome is the greatest single cause of death between one week and one year of age in the United States. In the current study, 40 such babies had 1.6 times as much muscle in their small pulmonary arteries as controls. Three quarters of this added muscle was due to hypertrophy, and the rest to hyperplasia of smooth-muscle fibers. Increased muscle at these sites is a characteristic consequence of chronic alveolar hypoxia. Age-matched infants living at high altitude at the time of death had 2.3 times as much muscle in their small pulmonary arteries as the low-altitude controls. These findings are consistent with the recent observation that some victims of the syndrome have had periods of apnea before death. (N Engl J Med 289:1167-1170, 1973)

After the neonatal period, the sudden-infant-death syndrome is the greatest single cause of death during the first year of life, accounting for an estimated 10,000 deaths in the United States each year. The typical patient is a generally healthy infant, two to five months old, who dies silently during sleep. Many hypotheses have been developed to explain the disorder. Some have been disproved, but others have not and are difficult to test. Perhaps the greatest mystery relates to the final mechanism (or mechanisms) responsible for the unexpected deaths. Recently, prolonged apneic and cyanotic episodes have been reported in a number of disorders during sleep, and in some cases such episodes have preceded sudden death. Such cases raise the possibility that apnea occurring during sleep is part of the final pathway in the syndrome. The current study supports this hypothesis by demonstrating pulmonary arterial abnormalities of a type commonly associated with chronic alveolar hypoxia.

PATIENTS

Dr. Russell S. Fisher, chief medical examiner of the state of Maryland, provided most of the cases used in the study. All infants one month to one year of age autopsied by Dr. Fisher and his staff between January 1, 1972, and March 1, 1973, were reviewed, and 40 cases placed in the category of sudden-infant-death syndrome when the death was unexplained by any clinical or post-mortem findings. An additional 22 infants were placed in the same category with pulmonary inflammation when they had bronchopneumonia, tracheobronchitis or interstitial pneumonia of too mild a degree to explain death. None of those with bronchopneumonia had any gross or microscopical consolidation, and the infants with tracheobronchitis had only

a mild or moderate peribronchial lymphoid infiltration with a small neutrophilic component. Interstitial pneumonia, when present, consisted of a few widely scattered small foci of moderate infiltrate.

Ten children who died in Leadville, Colorado (altitude of 3100 m, and mean barometric pressure of 525 mm Hg), served as hypoxic controls. This low barometric pressure leads to chronic alveolar hypoxia. Eight of the 10 children died as a consequence of acute infectious processes. Two others, one and three months old, had apneic spells and a poorly explained cause of death. In one of the infants, the apneic spells were reported during feeding and other types of handling. No records are available that describe their respiratory patterns during sleep. Thirty-nine nonhypoxic control infants were also used in the study. They were victims of accidents, homicides or acute bacterial meningitis. None of the cases included in any part of the study involved congenital cardiovascular anomalies or any other disorder known to affect the pulmonary vessels.

METHODS

In each case lungs were dissected, and blocks of tissue fixed in formalin within 20 hours of death. Multiple blocks of tissue from lungs and kidneys in each infant were sectioned at 6 μ and stained with hematoxylin and eosin. In some cases trichrome stains were used to assure that neither adventitia nor intima was being included in measurements of arterial media. To be measured, an artery had to be between 30 and 100 μ in diameter and cut in cross-section. By means of the point-counting technic of Chalkley under a constant magnification of 1000 times, relative cross-sectional areas of intimal nuclei, media and medical nuclei were determined for 20 arteries in both lungs and in kidneys. The numbers of intimal and medical nuclei were also counted in each artery. The mean area of individual pulmonary and renal arterial intimal nuclei did not vary greatly between different groups of cases so that the total area of these nuclei in an individual vessel was used as an internal standard or base line to which other measurements could be referred. The ratio $\frac{\text{area of arterial media}}{\text{total area of intimal nuclei}}$ was adopted as a measure of the relative area of medial smooth muscle present in individual arteries. A mean ratio was determined for arteries in each organ in each case.

The medial cystoplasmic area of each artery was calculated by subtraction of the combined area of medial nuclei from the total medial area. The relative cytoplasmic area of individual muscle cells in an artery was used as a measure of hypertrophy. It was determined by division of the medial cytoplasmic area by the number of medial nuclei present. An index reflecting the number of medial nuclei present in an artery, which was used as a measure of hyperplasia, was determined by division of the number of medial nuclei in an artery by the number of intimal nuclei. None of these three indexes were influenced by moderate dilatation of small arteries. An analysis of 20 arteries takes about 30 minutes. To avoid bias, all histologic analyses were undertaken without prior knowledge of an infant's designation as disease or control.

RESULTS

Blacks outnumbered whites, and males outnumbered females in the two categories of sudden infant death and in the nonhypoxic controls. All the high-altitude controls were white. Relative medial muscle mass in the small pulmonary arteries was significantly greater in the two categories of sudden infant death than in the nonhypoxic controls but less than in the high-altitude hypoxic controls. This muscle mass was somewhat greater in cases of sudden death without than in those with pulmonary inflammation. In both the cases of sudden infant death and the high-altitude cases, the increased pulmonary arterial medial mass was due to the combined effects of smooth-muscle hyperplasia and hypertrophy.

The small pulmonary arteries were usually dilated in cases of sudden infant death by comparison with the nonhypoxic controls. This arterial dilatation makes it very difficult to recognize the increased arterial muscle mass in sudden-infant-death syndrome by simple microscopical inspection. The dilatation also prevents recognition of the increased pulmonary arterial muscle mass in such infants by representing pulmonary arterial-wall thickness in per cent of the vessel's diameter (this widely used technic is simple to perform, but its numerical values are reduced by arterial dilatation). There were no abnormalities in the small renal arteries of the patients by comparison with the control. No intimal proliferation or thromboembolic lesions were detected in any arteries of the patients or the controls.

DISCUSSION

The current study demonstrates that smooth-muscle fibers in small pulmonary arteries are commonly both hyperplastic and hypertrophied in the sudden-infant-death syndrome. This abnormality seems confined to the lesser circulation since it was not found in the small renal arteries of the systemic circuit. This pulmonary vascular abnormality is most probably related to chronic alveolar hypoxia, since there was no evidence of congenital cardiovascular anomalies or other disorders that might increase pulmonary arterial pressure or blood flow in these infants. Such chronic alveolar hypoxia is a common cause of increased pulmonary vascular resistance in a variety of disorders. The list includes disorders that damage central mechanisms of respiratory control, interfere with the bellows mechanism of the chest and obstruct airways. Most of these disorders cause alveolar hypoxia by reducing alveolar ventilation. The pulmonary circulation has a characteristic response. Arterial segments exposed to hypoxic air from adjacent alveoli develop a coat of hyperplastic and hypertrophied smooth-muscle fibers, a presumed response to increased vasomotor activity in the involved vessels. Such arteries also are usually dilated, a possible reflection of an increased pulmonary blood volume. Arterial intimal proliferative lesions do not develop.

Individual differences appear to influence the pulmonary vascular response to alveolar hypoxia. When the newborn lives at high altitude, alveolar hypoxia appears to arrest the normal neonatal decrease of pulmonary arterial-muscle mass in some children, but not in others. This correlates wtih the finding that some children at high altitude have pulmonary arterial pressures as low as those found at sea level whereas others have pressures as high as those normally present at birth, both presumably having a similar degree of alveolar hypoxia. Such individual differences in the pressor response to alveolar hypoxia may partially explain the differing mass of pulmonary arterial muscle from one patient with the syndrome to another, so that even a normal muscle mass in small pulmonary arteries might not exclude antecedent alveolar hypoventilation.

Immaturity or malfunction of central mechanisms of respiratory control appears the most likely explanation for the pulmonary arterial abnormalities in the syndrome of sudden infant death. Most victims die silently during sleep, and some have been observed to have episodes of prolonged apnea and cyanosis, usually during sleep, before death. Both cardiac and respiratory abnormalities may contribute to these episodes, but the evidence is stronger for a respiratory mechanism. Both periods of apnea and sudden infant death are far more common in premature than in full-term infants. Probable mild respiratory-tract infections were identified in 22 of the 62 babies with sudden death in the current study, and the association has often been reported in the past. There is some evidence that such infections may prolong episodes of apnea and cyanosis in the early months of life and hence may pose a special hazard to those predisposed to the syndrome.

The Pickwickian syndrome has certain features in common with that of sudden infant death. Alveolar hypoventilation, increasing during sleep, is a complication of the disorder, and several involved patients have reportedly had a sudden, unexpected respiratory death during sleep. If a central mechanism of respiratory control is responsible for the sudden infant deaths, the abormality may be in the lower brainstem. The syndrome of sleep apnea, Ondine's curse, has been reported after high bilateral cervical cordotomy and lower-brainstem damage.

Finally, there is a small possibility that the increased muscle in small pulmonary arteries of babies with sudden death is a primary rather than a secondary phenomenon. Pulmonary arterial medial hypertrophy is the predominant abnormality in a small number of children with primary pulmonary arterial hypertension.

We are indebted to Dr. Elliot D. Weitzman, Montefiore Hospital, New York City, for advice and to Dr. Geno Saccomanno, St. Mary's Hospital, Grand Junction, Colorado, who kindly supplied the cases from Leadville, Colorado.

From the Department of Pathology, M. S. Hershey Medical Center, Pennsylvania State University, College of Medicine, Hershey, Pa. 17033, where reprint requests should be addressed to Dr. Naeye.

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REMARKS OF GOVERNOR VANDERHOOF BEFORE THE COLORADO RIVER WATER USERS ASSOCIATION

Mr. DOMINICK. Mr. President, on November 26, 1973, Honorable John D. Vanderhoof, Governor of Colorado, addressed the 30th Annual Session of the Colorado Water Users Association at Las Vegas, Nev. This organization represents the seven Colorado River Basin States.

The importance of the Colorado River cannot be over emphasized. The Governor's speech sounds a clear warning that the potential impact on available water supplies of the Colorado River could become enormous as a result of increased demands for energy production. Certainly, we are all becoming more and more aware of the necessity for increased energy production, as we acutely recognize the severity of this crisis touching on each one of us.

Mr. President, the remarks of Governor Vanderhoof deserve the attention of any person interested in the Colorado River. I ask unanimous consent that his speech be printed in the RECORD.

There being no objection, the address was ordered to be printed in the RECORD, as follows:

REMARKS OF JOHN D. VANDERHOOF

The Colorado River Water Users Association is an organization of the seven Colorado