

Prevalence of anemia in First Nations children of northwestern Ontario

Elaine A. Whalen, RN Laura E. Caulfield, PHD Stewart B. Harris, MD, MPH, CCFP

ABSTRACT

OBJECTIVE To estimate the prevalence of anemia among First Nations children of northwestern Ontario.

DESIGN Retrospective review of all hemoglobin determinations between 1990 and 1992 in the Sioux Lookout Zone.

SETTING The Sioux Lookout Zone Hospital, a secondary care referral hospital for 28 remote First Nations communities in northwestern Ontario, affiliated with the University of Toronto's Sioux Lookout Program.

PARTICIPANTS All First Nations children age 3 to 60 months who had produced venipuncture or fingerprick blood samples between 1990 and 1992 (614 children had a total of 1223 hemoglobin determinations).

MAIN OUTCOME MEASURES Prevalence of anemia by age, sex, geographical location, and diagnosis. Anemia was defined as a hemoglobin value less than 110 g/L.

RESULTS Prevalence of anemia peaked in the age range of 6 to 24 months with prevalence rates of 51.7% to 79.3%. Conditions most commonly associated with anemia were respiratory tract infections. Children living in communities in the western part of the Sioux Lookout Zone were 1.64 times more likely to have anemia (95% confidence interval 1.15, 2.35) than children in the other communities.

CONCLUSIONS Anemia appears to be a serious public health problem among preschool children in the Sioux Lookout Zone.

RÉSUMÉ

OBJECTIF Estimer la prévalence de l'anémie chez les enfants des Premières nations du nord-ouest de l'Ontario.

CONCEPTION Revue rétrospective de tous les dosages d'hémoglobine effectués entre 1990 et 1992 chez les patients de la zone Sioux Lookout.

CONTEXTE Le Sioux Lookout Zone Hospital, centre hospitalier de référence pour soins de niveau secondaire desservant 28 communautés des Premières nations vivant en régions éloignées du Nord-ouest de l'Ontario, et rattaché au programme Sioux Lookout de l'Université de Toronto.

PARTICIPANTS Tous les enfants des Premières nations âgés de 3 à 60 mois chez qui on avait prélevé entre 1990 et 1992 des échantillons de sang par ponction veineuse ou piqûre du doigt (614 enfants ont fourni 1223 dosages d'hémoglobine).

PRINCIPALES MESURES DES RÉSULTATS Prévalence de l'anémie par groupes d'âge, de sexe, de localisation géographique et de diagnostic. On posait le diagnostic d'anémie lorsque le taux d'hémoglobine était inférieur à 110 g/L.

RÉSULTATS Le pic de prévalence de l'anémie s'est retrouvé entre les âges de 6 et 24 mois avec des taux de prévalence variant de 51,7 % à 79,3 %. Les infections respiratoires furent les conditions les plus fréquemment associées à l'anémie. Les enfants qui vivaient dans les communautés les plus occidentales de la zone Sioux Lookout étaient 1,64 fois plus à risque d'être anémiques (intervalle de confiance à 95 %, de 1,15 à 2,35) comparativement aux enfants vivant dans les autres communautés.

CONCLUSIONS L'anémie semble constituer un grave problème de santé publique chez les enfants d'âge préscolaire de la zone Sioux Lookout.

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RESEARCH

Prevalence of anemia in First Nations children of northwestern Ontario

Iron deficiency has been identified as the most prevalent nutritional problem in the world.¹ One third of iron-deficient children have the more severe form of the disorder, anemia. Both in developing countries and in Canada, moderate iron-deficiency anemia (hemoglobin levels below 100 g/L) has been shown to depress mental and motor functioning in affected infants.^{2,3} Although there is controversy regarding the effectiveness of treatment with iron to reverse these effects, there is universal consensus that iron-deficiency anemia is associated with developmental cognitive and motor deficits and that prevention is essential for every infant.^{4,5}

Among the First Nations children of northwestern Ontario, the prevalence of iron-deficiency anemia is considered to be high, but a systematic study of anemia has never been undertaken. To evaluate its importance as a public health concern in this area, we estimated the prevalence of anemia among children younger than 5 years of age in the Sioux Lookout Zone (SLZ) by reviewing hemoglobin determinations conducted from 1990 to 1992.

The study population lives in an area of Ontario defined by Health Canada's Medical Services Branch. Fourteen thousand registered Algonquin-speaking Cree and Ojibway First Nations people live in 28 scattered remote communities in northwest Ontario. Living conditions for these people are hard; adequate housing, central heating, safe water, and adequate disposal systems for sanitary waste are not universally available. Overcrowding and chronic unemployment are serious problems.

In the past, First Nations people had a nomadic lifestyle consisting primarily of hunting, fishing, and trapping as they followed the migratory supply of wildlife. Their diet was high in protein from wild meats and seasonally supplemented with berries and roots. With the development of the reservation system, they were forced to settle in one area. The primary source of food changed from wildlife to the Northern Store (a dry goods and grocery store chain).

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Ms Whalen was a Research Assistant for the University of Toronto Sioux Lookout Program when this article was written. **Dr Caulfield** is an Assistant Professor at the Centre for Human Nutrition at The Johns Hopkins School of Hygiene and Public Health in Baltimore. **Dr Harris**, formerly the Medical Director for the Sioux Lookout Program, is an Assistant Professor in the departments of Family Medicine and Epidemiology at the University of Western Ontario in London, Ont.

Health care in the town of Sioux Lookout is directed from a 42-bed hospital operated by the Medical Services Branch in conjunction with the local First Nations Health Authority. Community health nurses and physicians provide front-line care.

METHODS

The Sioux Lookout Zone Hospital (SLZH) provides all laboratory services. Blood is drawn by venipuncture (at SLZH) or fingerprick (at nursing stations) when a child presents with illness, for a preoperative check, or for a general physical checkup. Specimens are shipped to the hospital laboratory daily. According to laboratory policy, carbon copies of the laboratory slips are kept on file for 3 years; therefore, it was possible to obtain and review the results of all hemoglobin determinations for the study period. Information contained on each laboratory slip included community of residence, date of birth, date specimen was drawn, reason for blood draw, and hemoglobin value. Confidentiality of data records was maintained at all times.

Between 1990 and 1992, 1223 hemoglobin determinations were made. From these we excluded six determinations with implausible values for the child's age and 311 determinations taken from infants younger than 3 months of age. Young infants were eliminated because of the inherent difficulties in interpreting their hemoglobin values. This left 906 determinations consisting of one to 15 determinations for 614 children. One determination for each child was randomly chosen to remain in the sample, leaving a final sample size for analyses of 614.

Anemia was defined as a hemoglobin value less than 110 g/L.⁶ For analysis, data were broken down into 12 categories according to months of age, and the reasons for the blood draws were grouped into seven categories: respiratory infections, other infections, preparatory for dental work, preoperative check, anemia and other nutritional conditions, unknown (unspecified), and other. The community of residence of the child was categorized according to its geographic location in the SLZ as north, south, east, or west.

The distributions of hemoglobin and the prevalences of anemia were compared across categories of each variable. Multiple logistic regression analyses were used to determine whether observed differences in the prevalences of anemia across variables remained after adjusting for age.

RESULTS

Table 1 presents the means and standard deviations of the hemoglobin values and the estimated prevalences of anemia across categories of selected variables. The prevalences of anemia vary across the age range from 79.2% for infants 9 to 12 months of age to 12.9% for children 48 to 60 months of age.

The prevalences of anemia vary by region within the SLZ; the highest prevalence is found in the west. The prevalences of anemia in Sandy Lake and Pikangikum (the principal communities in the western region) are 61.3% and 68.4%, respectively, compared with a prevalence of 52.5% for Sioux Lookout, which is located in the south.

The prevalences of anemia by reason for the hemoglobin determination are also listed in **Table 1**. The prevalences of anemia vary by diagnosis; children diagnosed with respiratory infections have the highest prevalence of anemia (65.6%), whereas those presenting for dental work or a preoperative check have the lowest prevalences of anemia (21.2% and 25.6%, respectively). Only 8.5% of the determinations were made because of anemia or other nutrition-related conditions, and the prevalence of anemia for this group was 61.5%.

The reasons for blood draw differ depending on the age of the child. For example, 42.5% of children younger than 1 year were diagnosed with respiratory infections, compared with only 3.2% of children younger than 3 years. No children younger than 1 year were tested because of future dental work as compared to 10.2% of children younger than 3 years. Because the observed differences in prevalences of anemia by diagnosis could be due to differences in the ages of children presenting with particular conditions, we compared the changes in prevalence of anemia with age for each diagnosis category. The same age-associated declines in anemia prevalence are observed irrespective of the reason for the blood draw, with the exception of children tested because of anemia or other nutritional conditions (**Figure 1**). For those children, no decline in anemia with age is observed.

Results of the multiple logistic regression analyses revealed that, after controlling for age, children living in the west were 1.64 times (95% confidence interval [CI]: 1.15, 2.35) more likely to have anemia than children from other regions, whereas those having bloodwork done for dental work or as a preoperative check were 0.48 times (95% CI: 0.29, 0.82) less likely to have anemia than children presenting with all other conditions.

Table 1. Hemoglobin levels and prevalence of anemia (hemoglobin <110 g/L) by selected characteristics

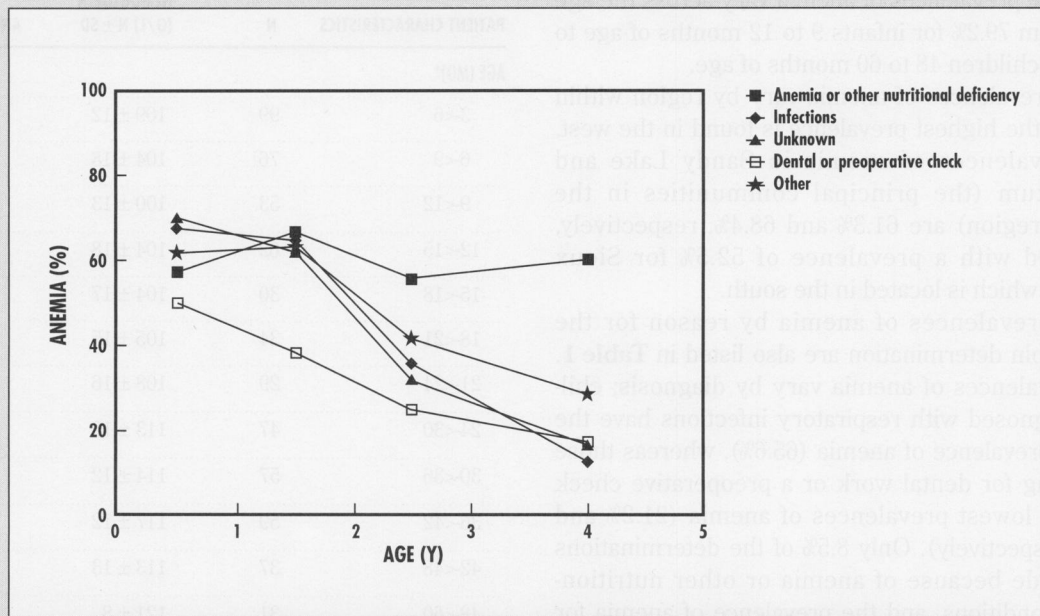
PATIENT CHARACTERISTICS	N	HEMOGLOBIN (G/L) N ± SD	ANEMIA (%)
AGE (MO)*			
3-6	99	109 ± 12	55.6
6-9	76	104 ± 18	71.1
9-12	53	100 ± 13	79.3
12-15	62	104 ± 18	62.9
15-18	30	104 ± 17	70.0
18-21	34	105 ± 15	61.8
21-24	29	108 ± 16	51.7
24-30	47	113 ± 13	38.3
30-36	57	114 ± 12	28.1
36-42	59	117 ± 12	18.6
42-48	37	113 ± 13	27.0
48-60	31	121 ± 8	12.9
SITE OF BLOOD DRAW			
Nursing station	61	104 ± 20	55.7
Sioux Lookout Zone Hospital	553	109 ± 15	49.2
REASON FOR HEMOGLOBIN DETERMINATION*			
Respiratory infection	151	105 ± 15	65.6
Other infection	88	108 ± 15	52.1
Dental work	104	116 ± 11	21.2
Preoperative check	43	115 ± 12	25.6
Anemia or other nutritional deficiency	52	101 ± 18	61.5
Unknown	105	108 ± 14	55.2
Other	71	110 ± 17	52.1
RESIDENCE IN SLZ*			
North	113	112 ± 13	39.8
West	244	106 ± 17	59.0
East	158	109 ± 14	44.9
South	99	110 ± 15	46.5

*Significant difference across categories, $P < 0.05$.

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Figure 1. Age-associated changes in the prevalence of anemia (hemoglobin < 110 g/L) by diagnosis: Values are plotted at the midpoint of each age interval.



DISCUSSION

These analyses clearly substantiate the clinical suspicion of childhood anemia as a serious public health problem among preschool children in the SLZ. Overall, anemia was identified in 46.6% of the children tested. More than half the infants 3 to 6 months of age were identified as anemic. Children 6 to 24 months of age were at greatest risk of anemia with prevalences ranging from 52% to 80%. Beyond 24 months prevalences declined substantially, but were still between 10% and 40%.

Some caution should be taken in interpreting the results due to the limitations of the methodology. This was not a representative survey of children younger than 5 years living in SLZ, but rather a review of all hemoglobin determinations performed on SLZ children 3 to 60 months of age over a 3-year period. Reasons for the determinations varied greatly, but most were performed as a routine matter on children with acute illness. Thus, the prevalences of anemia are likely to be overestimated to the extent to which children who are sick are also more likely to have anemia.

Only a small proportion of determinations were done because anemia was suspected. The age-associated changes in prevalence of anemia were consistently observed regardless of the reason for blood draw (except when anemia was suspected). Thus, any selection bias is likely to be constant across the age range of children studied here. The prevalences of anemia will also be somewhat biased because hemoglobin concentrations fall by as much as 10%, even with mild infections.⁷ Despite these limitations, the analyses presented here represent a reasonable first step in defining the public health importance of iron-deficiency anemia in this population, using data commonly available in many settings.

Because SLZ is a finite population, we can use information on population size to further refine our prevalence estimates. Hemoglobin determinations were performed on 614 of the estimated 2053 children younger than 5 years living in SLZ at that time. If we make the crude assumption that all anemic children were in fact identified, the overall prevalence of anemia would be re-estimated at 14%. Further, if we assume a constant birth rate of 410 births yearly and that all children with anemia were identified, new

age-specific prevalence estimates calculated for children 3 to 12 months, 12 to 24 months, and 24 to 60 months would be 49.1%, 23.4%, and 4.5%, respectively.

The assumption that all children who did not have their hemoglobin measured were nonanemic is improbable, but serves to provide a baseline to the prevalence estimates reported here. Although these new estimates are much lower than those based on children having bloodwork done, they do not refute the conclusion that anemia is a serious public health problem in SLZ, particularly for children younger than 2 years.

Our results are higher than but consistent with those reported in other studies involving First Nations populations. In 1983, Plaxton et al⁸ performed a similar review of the health care provided for preschool children in the SLZ. Of the preschooler's charts they reviewed, 55% recorded a hemoglobin value, and 36% had hemoglobin levels less than 120 g/L, indicating the presence of anemia in this same study population. Moffat⁹ demonstrated that 42.5% of children 6 to 24 months living in Norway House, Man, had hemoglobin levels below 110g/L. He also reported an incidence of iron-deficiency anemia of more than 50% in urban First Nations children attending a Winnipeg clinic.⁹ The prevalence of anemia among low-income First Nations people in the United States is also high. Yip et al¹⁰ reported the prevalence of anemia among First Nations children younger than 2 years participating in the Pediatric Nutrition Surveillance System to be 15% to 20%.

Based on hemoglobin values alone, we cannot determine the predominant cause of anemia in this population. Specific hemoglobinopathies resulting in lower hemoglobin concentrations are not known to exist in this population. Research indicates that, when the prevalence of anemia is high (15% to 20%), nearly all cases of anemia can be attributed to iron deficiency.¹¹ The peak period of risk as well as the age-associated declines in the prevalence of anemia observed here are also consistent with patterns of anemia associated with depletion of iron stores.¹² The Nutrition Canada survey¹³ in the early 1970s identified 40% of children aged 1 to 4 years with abnormal serum ferritin values. This most likely represents an underestimate due to the exclusion of infants younger than 1 year. We conclude that iron deficiency is the most likely cause of anemia in this population.

Change from traditional eating and breastfeeding patterns could be important factors in the development of anemia in this population.¹⁴ Unlike other First Nations populations, the prevalence of

breastfeeding at hospital discharge within SLZ is high (85% to 90%)¹⁵ and has remained consistent over the past decade according to annual reports from the SLZH maternity ward.¹⁶ Nurses in the community, however, report that breastfeeding is nearly always supplemented with other items, such as tea with sugar, condensed evaporated milk diluted with water, or juice, and that non-iron-fortified infant cereals are introduced as early as 3 weeks of age. Thus, despite a high rate of initiation of breastfeeding, many foods are being introduced during early infancy that are known to deplete iron stores and lead to the development of anemia.^{17,18}

Given our results, determining the cause or causes of anemia in this First Nations pediatric population is imperative. The first 2 years of life are filled with numerous growth and developmental milestones, both mental and physical. Children experiencing anemia in this population often exhibit developmental delays that will be irreversible. Further longitudinal research is needed to document the development of iron-deficiency anemia in this population and to identify risk factors for developing iron-deficiency anemia during the first 2 years of life, with the ultimate goal of developing an effective strategy for prevention. ♣

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Correspondence to: Dr Stewart B. Harris, Assistant Professor, Department of Family Medicine, The University of Western Ontario, Thames Valley Family Practice Research Unit, 100 Collip Circle, Suite 245, UWO Research Park, London, ON N6G 4X8; telephone (519) 858-5028, fax (519) 858-5029, e-mail sharris1@julian.uwo.ca

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DDAVP* NASAL SOLUTION

Desmopressin Acetate Intranasal Solution

THERAPEUTIC CLASSIFICATION

Antidiuretic

INDICATIONS

Diabetes Insipidus

DDAVP (desmopressin acetate) is indicated for the management of vasopressin sensitive central diabetes insipidus and for the control of temporary polyuria and polydipsia following head trauma, hypophysectomy or surgery in the pituitary region.

Nocturnal Enuresis

DDAVP (desmopressin acetate) is indicated in the short-term management of nocturnal enuresis in patients 5 years of age and older who have normal ability to concentrate urine. DDAVP should be used in conjunction with non-medical therapy such as motivational counselling and bladder exercises.

CONTRAINDICATIONS

Hypersensitivity to desmopressin acetate or to any of the constituents. Because of the risk of platelet aggregation and thrombocytopenia, DDAVP should not be used in patients with type IIB or platelet-type (pseudo) von Willebrand's disease.

WARNINGS

For intranasal use only. DDAVP (desmopressin acetate) is not effective in controlling polyuria caused by renal disease, nephrogenic diabetes insipidus, psychogenic diabetes insipidus, hypokalemia or hypercalcemia.

Fluid intake should be adjusted in order to reduce the possibility of water retention and hyponatremia especially in very young and elderly patients (see Dosage and Administration). Particular attention should be paid to the risk of an extreme decrease in plasma osmolality and resulting seizures in young children.

Changes in the nasal mucosa resulting from rhinitis, scarring, edema or other disease may cause erratic, unreliable absorption in which case intranasal DDAVP should not be used. In the case of temporary rhinitis, consideration should be given to using an injectable form of desmopressin, until the nasal mucosa returns to normal.

PRECAUTIONS

General

DDAVP (desmopressin acetate) at high dosage (40 mcg or more) has very occasionally produced a slight elevation of blood pressure, which disappeared with a reduction in dosage. The drug should be used with caution in patients with coronary artery insufficiency and/or hypertensive cardiovascular disease because of possible tachycardia and changes in blood pressure.

In the control of diabetes insipidus, the lowest effective dose should be used and the effective dosage, as determined by urine volume and osmolality and, in some cases, plasma osmolality, should be assessed periodically.

DDAVP should not be administered to dehydrated patients until water balance has been adequately restored.

Desmopressin should be used with caution in patients with cystic fibrosis because these patients are prone to hyponatremia.

Children and geriatric patients should be closely observed for possible water retention due to over ingestion of fluids. When fluid intake is not excessive, there is little danger of water intoxication and hyponatremia with the usual intranasal doses of desmopressin used to control diabetes insipidus. Fluid intake should be carefully adjusted to prevent overhydration.

There are reports of changes in response over time, usually when the drug has been administered for periods longer than 6 months. Some patients may show decreased responsiveness, others a shortened duration of effect. There is no evidence that this effect is due to the development of binding antibodies, but may be due to local inactivation of the peptide.

For control of nocturnal enuresis a restricted fluid intake is recommended a few hours before administration.

Drug Interactions

Clofibrate, chlorpropamide and carbamazepine may potentiate the antidiuretic activity of desmopressin while demeclocycline, lithium and norepinephrine may decrease its activity.

Although the pressor activity of DDAVP is very low compared with the antidiuretic activity, use of large doses of DDAVP with other pressor agents should be done only with careful patient monitoring.

Pregnancy

Reproductive studies performed in rats and rabbits have revealed no evidence of harm to the fetus by desmopressin. The use of DDAVP in pregnant women with no harm to the fetus has been reported. However, no controlled studies in pregnant women have been carried out. Unlike preparations containing the natural hormone, DDAVP in antidiuretic doses has no uterotonic action, but the physician should weigh possible therapeutic advantages against potential risks in each case.

Nursing Mothers

There have been no controlled studies in nursing mothers. A single study on a post-partum woman demonstrated a marked change in maternal plasma DDAVP level following an intranasal dose of 10 mcg, but little DDAVP was detectable in breast milk.

Paediatric Use

DDAVP (desmopressin acetate) has been used in children with diabetes insipidus. The dose must be individually adjusted to the patient with attention in the very young to the danger of an extreme decrease of plasma osmolality with resulting convulsions. Dosage in infants younger than 3 months has not been established. Dose should start at 5 mcg or less. Use of DDAVP in infants

and children will require careful fluid intake restriction to prevent possible hyponatremia and water intoxication.

LABORATORY TESTS

Diagnosis of Central Diabetes Insipidus

Central diabetes insipidus may be demonstrated by the inability to produce urine of osmolality above 175 mOsm/kg with dehydration severe enough to cause a loss of greater than 2% of body weight.

Patients are selected for therapy by establishing a diagnosis by means of a water deprivation test, the hypertonic saline infusion test, and/or response to 5 units arginine vasopressin given s.c. after dehydration. Continued response to DDAVP can be monitored by urine volume and osmolality. In cases of severe dehydration, plasma osmolality determination may be required.

ADVERSE REACTIONS

Infrequently, high doses of desmopressin have produced transient headache and nausea. Nasal congestion, rhinitis, flushing, and mild abdominal cramps have been reported. These symptoms disappeared with reduction in dosage. Side effects reported from controlled clinical trials involving 638 subjects included headache (2%), rhinitis (1%), nasal discomfort (1%), epistaxis (1%) and abdominal pain (1%). Other effects, reported at a frequency of less than 1%, included dizziness, chills, wheezing, rash, edema of face and hands, nausea, constipation, anorexia, increased appetite, conjunctivitis and aftertaste in the mouth. These symptoms disappeared with reduction of dosage or withdrawal of drug. Adverse effects rarely necessitate discontinuation of the drug.

OVERDOSAGE

Symptoms and treatment

Overdose symptoms include headaches, abdominal cramps, nausea, and facial flushing. There is no known antidote. Dosage and frequency of administration should be reduced, or the drug withdrawn according to the severity of the condition.

Water retention can be controlled by decreasing the dosage of desmopressin; severe water retention caused by overdosage may be treated with a diuretic such as furosemide.

DOSAGE AND ADMINISTRATION

Dosage

Diabetes Insipidus

Central diabetes insipidus may be demonstrated by the inability to produce urine of osmolality above 175 mOsm/kg with dehydration severe enough to cause a loss of greater than 2% of body weight (see Laboratory Tests). Dosage in children up to 3 months of age has not been established. Dosage must be individualized but clinical experience has shown that the average daily dose for adults is 10 mcg to 40 mcg DDAVP (desmopressin acetate) and for children 3 months to 12 years of age, 5 mcg to 30 mcg. This may be given as a single dose or divided into two or three doses. About one third of patients can be treated with a single daily dose. Geriatric patients may be more sensitive to the antidiuretic effect of the usual adult dose of desmopressin acetate.

In those children who require less than 10 mcg, the rhinoly presentation or the 2.5 mcg spray should be used. In some patients, better control of polyuria is attained with smaller doses given at 6 to 8 hour intervals.

Most adults require 20 mcg daily, administered in two divided doses (in the morning and the evening). Initially, therapy should be directed to control nocturia with a single evening dose. Response to therapy can be measured by the volume and frequency of urination and duration of uninterrupted sleep. The dosage of desmopressin should be adjusted according to the diurnal pattern of response, with the morning and evening doses being adjusted separately. Patients being switched from parenteral to intranasal administration generally require 10 times their maintenance intravenous dose intranasally.

To institute therapy with DDAVP, patients should be withdrawn from previous medication and allowed to establish a baseline polyuria to permit determination of the magnitude and duration of the response to medication. In less severe cases, prior water loading may be desirable to establish a vigorous flow of urine. When the urine osmolality reaches a plateau at low level (in most cases, less than 100 mOsm/kg), the first oral dose of DDAVP (10 mcg) is administered intranasally. A urine sample is obtained after two hours and hourly thereafter following DDAVP administration. Urine volume and osmolality is measured. When the patient has reached the previous baseline urine osmolality and urine flow, the drug effect has ceased and the next dose of DDAVP is administered. The cycle is then repeated until the patient has reached a stable condition.

Nocturnal Enuresis

Dosage must be individualized by the physician. The clinically effective intranasal dose varies between patients and ranges between 10 mcg and 40 mcg desmopressin acetate daily. A suitable starting dose for adults and children is 20 mcg given one hour before sleep. A restricted fluid intake is recommended a few hours before administration.

How Supplied

Metered dose spray pump (2.5 mL) provides 25 doses of 10 µg desmopressin acetate. Also available, 5.0 mL which provides 50 doses of 10 µg desmopressin acetate.

Product monograph available upon request

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