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Message From the Director

Looking to the Future of the Indian Health Service

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Since I was confirmed and sworn in as Director of the Indian Health Service in April 1994, my work has been rewarding and challenging. It has been challenging because of the pressures on the Agency that require us to change how we conduct business and deliver health care. It has been professionally rewarding because the guiding principle of putting patient care, prevention, and community health care first has been strengthened, and this principle continues to serve as the focus for decision-making in all aspects of the Indian Health Service. I have noticed a dramatic change in discussions with our partners in Indian health that reflects this guiding principle. From finance to facilities, from paperwork to personnel, the effect on primary and preventive health care is considered in decisions we make. The realization that how the telephone is answered can impact patient care as much as spending time with a patient or their family is profoundly sobering. You never know if the caller is from Congress, tribal governments, the Department, another employee, or most importantly, a patient . . . they all can influence the support Indian programs receive and how these programs are perceived. The effect of our words and actions today can benefit or harm us many times over.

Employees of Indian health programs have helped to raise the health status of American Indians and Alaska Natives significantly. Since 1973, infant mortality has decreased 60% and is nearing the rate of the general population; mortality rates have also dropped for tuberculosis (80%), gastrointestinal diseases (76%), and deaths due to unintentional injury (56%). Life expectancy at birth for American Indians and Alaska Natives has increased 12.2 years since 1973 and is now just 2.6 years below that of the general population. These are examples of the achievements made in the last 20 years.

It is the collaboration of Indian Health Service, tribal, and urban Indian health programs that has made these achieve-

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ments possible. Together, we have gone from providing health care services for 455,000 outpatient visits in 1955 to more than 6.3 million visits today; on the other hand, the number of hospital admissions is lower today than it was in 1965. This change can be attributed, in part, to the general shift from inpatient to outpatient care and an increased emphasis on health promotion and disease prevention services. Clean water and sanitation systems are key to improving health; in this regard, the facilities engineering program of the Indian Health Service has made and continues to make important contributions.

Much has been accomplished, but there is much more for us all to do. The health status of American Indians and Alaska Natives continues to remain lower than that of the general United States population. Death rates for specific health status indicators are higher in American Indians and Alaska Natives than in the general population, including alcoholism (5.7 times higher), tuberculosis (5.3 times higher), diabetes (2.7 times), pneumonia and influenza (1.5 times), suicide (1.5 times), homicide (1.4 times), and cervical cancer (age-adjusted rate, 1.6 times higher). While Indian women are less likely to die from breast cancer than women in the rest of the population (the death rate in the U.S. general population is nearly twice as high), they tend to survive a shorter period of time after the cancer is detected. Deaths from unintentional injuries occur at an age-adjusted rate 2.8 times the national average. In three Indian Health Service Areas, deaths from unintentional injuries are more than four times the U.S. rate. These disparities in health status indicators have a severe impact on Indian communities and families. Years of potential life lost for American Indians and Alaska Natives, as a result of a relatively high rate of premature deaths, is 50% greater than for individuals in the general population.

One of the greatest contributions to improved health is access to safe water and a safe sanitation system. Yet today, 12% of American Indian and Alaska Native homes still lack safe drinking water and a safe way to dispose of waste. The cost to make necessary improvements to existing water and sewage treatment systems, to address the deficiencies in existing home systems, and to provide for those homes that have no water or sewer access is more than $1.5 billion.

Addressing these compelling health needs will require new strategies, partnerships, resources, and sources for funding and support, as well as fundamental changes in the way the Indian Health Service conducts business. It is obvious that the Agency must change if it is to exist and be effective in the next century. Change must be managed to be successful. While transition occurs, it must not adversely affect the quality of care that we provide to American Indian and Alaska Native people.

Fourteen years ago I spoke about Indian health care and the Agency. Some of the facts I cited included: In 1956, the Indian Health Service received a $39 million budget; in 1960, $50 million; and in 1981, $638 million. That was the trend: an ever-increasing level of resources for the Indian Health Service. Today, the Indian Health Service has a budget close to $2 billion, but, unfortunately, the trend of an annually increasing budget leveled out four years ago. A level budget means lower purchasing power because of the negative impact of medical inflation and more expensive medical procedures, a service population increasing in number at a rate of more than 2% annually, and Congressionally-mandated expenditures. The budget gains that the Indian Health Service realized several years back have been eroded by the level budget. In fact, the Indian Health Service has less per capita purchasing power today than it did in 1956. In 1956, IHS had $1,260 (in constant 1995 dollars) to spend on each person in the service population; in 1995, that figure is only $1,242.

Even as the budget stopped increasing there continued to be an ever increasing number of beneficiaries for health services and demand for all services; an increasing cost of health care, other goods, and staff; an increasing number of elderly; and increasing mandates for cost containment in all areas. Other external pressures on the Agency included changing patterns of disease among Indian people, leading to more chronic conditions that influence and are influenced by social and economic factors and the quality of life for American Indians and Alaska Natives. Additionally, politics had started to play a larger role in the health care arena.

Today those pressures have not changed, but have been joined by other, more complex external influences: an environment of unparalleled Federal budget reductions, the transfer of many Federal programs and resources to States, decreases in discretionary programs in the Federal budget, the overall erosion of resources, and a strong anti-government sentiment. Another significant additional pressure is the influence by competitive and economic based driven managed health care programs on the public health focus of health care.

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There have been discussions about the Contract with America. I have asked before, and continue to ask, "What about the treaties with American Indians and Alaska Natives? What about the American Indians and Alaska Natives who signed the first treaties; who believed in them; who gave up land; who gave up fishing rights, mineral rights, and forests; and, who lost their lives? Anti-government sentiment must not be allowed to be a reason to ignore responsibilities and treaty obligations to American Indian and Alaska Native people.

A number of leaders in government and health care are talking about changes in programs that affect public health, the poor, and the underprivileged. Many health, social, and welfare programs are being considered for budget cuts or elimination. These reflect the strong anti-government senti-

ment across the nation, in Congress, and with the public. That sentiment affects programs that directly serve American Indians and Alaska Natives on and off the reservations.

During my confirmation hearings, I conveyed to Congress that my focus for the Agency would be for it to become more efficient, effective, and accountable. I said that there needed to be an increased awareness of costs and benefits and an involvement of local communities and individuals in the decision-making process, that new and different ideas needed to be incorporated into operating programs, and that there needed to be effective communication about what the Agency is doing in those areas.

Positive changes have taken place since that time. Collaboration with tribes and Indian leaders has increased. Tribes and Indian organizations are now involved in the development of the Indian Health Service budget and the setting of health priorities. An open style of management is taking hold; selections for key Indian leadership positions within the Agency are made with the direct involvement of tribal and Indian organization representatives within that Federal process; and the redesigning of the Indian Health Service is guided by a team of tribal and Indian leaders with Agency employees. We have made strides toward inclusion and involvement. In addition, tribal governments are identifying ways the Agency can be included in or supportive of their health delivery programs. I continue to believe that the Indian health system, comprising the Indian Health Service, tribal, and urban Indian programs, will improve the health of Indian people.

As I stated to Congress, everyone in the Indian Health Service has an obligation to provide to tribes and the Congress complete information and their best assessment of possible effects of a course of action for mutual discussion and decision. That management goal is taking hold. Major decisions involve all tribes: those that contract with the Indian Health Service to provide their own health services, those that compact to assume administrative and operational control over their health programs, and those tribes that choose to stay within the Federal system of health care delivery. A new Indian Health Service budget structure and a business plan are being developed to reflect the commitment to self-determination and to provide for participation. At present, almost one-third of American Indian and Alaska Native people receive health services through tribal programs that were once administered and operated by the Indian Health Service. The trend of providing services through self-determination contracts and compacts is expected to continue and to increase within five years to where at least half of the American Indian and Alaska Native population,if not more, receive health services through tribal programs. The business plan will assess expenses, cost allocation systems, the budgetary process, how to generate resources within Federal programs, and also how the Agency can assist tribes and Indian organizations in networking for outside resources. The business plan's focus is on how best to benefit patient care and preventive services by generating and efficiently using resources.

It has been two years since I was sworn in as Director and ,with tribal participation and congressional support, we are making the changes necessary to begin a new era in Indian health care. We are making the Indian Health Service program more effective so it can survive future political and economic pressures. From the beginning, the leadership of the Indian Health Service, tribal governments, and Indian organizations shared the concern that if we did not address these pressures and make the necessary changes, then others would make those changes for us. Together, we have avoided having others step in and implement their changes. Self-imposed changes are being made in Indian health, and the Congress has endorsed those changes by increasing the Indian Health Service 1997 budget to slightly above the 1996 level, rather than reducing it as they have for other Indian agencies and programs.

These demonstrations of support for Indian health programs are welcome, but we must not lose sight of the fact that the Indian Health Service is only one part of the Indian health system. The Indian Health Service does not have the resources to solve everything, and that is why we must collaborate to find joint solutions for the health of Indian people. I continue to believe that collaboration accomplishes more than what can be achieved alone. Advocating for Indian health is a shared responsibility throughout the Indian health system. The partners of the Indian health system must invite

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others to participate so that Federal, tribal, and urban Indian health care programs can become more self-sufficient and less dependent on the Federal stream of dollars that is so inconsistent. Private and public foundations, universities, organizations and the medical community can play a role in helping the Indian health system meet the health needs, particularly the unmet needs, of American Indian and Alaska Native people.

Over time, we have recognized that change within the Indian Health Service is not the only factor needed to improve the Indian health system. While I have been Director, the Agency and the Department of Health and Human Services have been involved in a number of initiatives. Serving on the Domestic Policy Council, together with the Department of the Interior, we are examining issues related to the protection of elders and children, telecommunication systems to provide information to remote and rural areas of our country, and the sharing of services for non-medical functions of our agencies across all Federal Departments. In addition to our participation, we are bringing with us representatives of tribes and

Indian organizations so everyone can benefit from their perspectives and priorities, and to reinforce the obligation that Federal agencies have to work directly with American Indian and Alaska Native sovereign governments and the people they represent.

Within the Indian Health Service, I will continue to address priorities and shift resources to where the services are provided. We have transferred or reduced more than 1200 positions from the Headquarters and Area Office levels. The local service units have gained close to 400 staff positions. I acknowledge the perception that many service units do not notice the addition of staff but only a continued reduction, since most of the additional positions have gone to staff new facilities. Vacant positions at the service unit level are a reality, and we will work with tribal and Agency health leaders and communities to address this issue.

I am committed to the goal of the Indian Health Service: to raise the health status of American Indians and Alaska Natives to the highest possible level. When putting together Indian Health Service work teams, I look for an organizational commitment to do what needs to be done with professional integrity and an appreciation of the fact that we are here to serve Indian people and are accountable to them. As an Agency, it is essential that we develop clinical and administrative management leadership programs. We need to look at who are the Indian health leaders of the future, and provide opportunities for these individuals to develop their abilities so they are prepared to take leadership roles in Indian health. Qualifications for positions of leadership within the Indian Health Service include an understanding of tribal issues, the ability to listen, a management style of openness, and experience in managing a complex and diverse program with multiple demands and needs.

I think it is important that all employees understand the goals of the Agency that support the mission. I established health emphasis areas in my Vision Statement and will continue to advocate for elder health care, Indian women's health issues, youth substance abuse prevention programs, and mental health services. As a result of suggestions received during meetings with employees and Indian leaders, I have added specific initiatives that support these health emphasis areas. I am particularly interested in the initiative to incorporate traditional healing as part of our health care program. I believe it is very important to the health of Indian people that traditional healing be respected and made a part of the policy and operations of our programs. I recognize that traditional healing methods are tribal- and culture-specific, therefore, our goal is to allow for maximum flexibility so that local communities can work with the service units to integrate traditional healing practices into Indian Health Service programs. I have just approved an Initiative on Domestic Violence and Child Abuse. Initiatives for the future include one on Disabilities, another on Veterans Health, and one on Men's Wellness.

We have made strides toward inclusion and involvement.

The Indian Health Service has a proud history of success. The combined efforts of tribal, urban Indian, and Indian Health Service health programs will enable us to expand that success and lead us into the new era of Indian health care. Challenges are not barriers. Challenges are guideposts for improving the effectiveness of health programs for Indian people. Our reward is knowing that we have done the very best we possibly can to support and provide health care to American Indian and Alaska Native people nationwide. □

LETTER TO THE EDITOR □

I wanted to congratulate you for printing the article "Perceptions of Caring Behaviors in Health Providers." While the insert identifies the tribe as Pima Maricopa, I think their perceptions are generic and apply to us all. What I really appreciated was the table contrasting [behaviors of] the caring versus the non-caring health professional. This article gave me a feeling of pride in my work, as I found I had been doing some things right. I copied the article and passed it around.

This type of article filled a need in my work. Please continue to print articles about cultural sensitivity.

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Underreporting of Deaths Among American Indian Children in California, 1979-1993

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Abstract

Mortality rates for American Indians in California are believed to have been underreported for many causes of death because of misclassification of Indian race on state death certificates.[1] In a previous study, mortality rates for American Indian infants in California who died between 1984 and 1988 were found to be 2.8 times greater than reported when adjusted for racial misclassification.[2]

This study matches birth records of American Indians born between 1979 and 1993 in California to state death certificates to determine the extent of racial misclassification reported for deaths among children under 15 years of age. Birth files were matched to death certificates through an automated computer linkage system. After adjusting for racial misclassification on death certificates, estimates for the number of American Indian children under age 15 who died in California were three to four times greater than reported in the state mortality data.

Introduction

Many of the objectives in the Indian Health Care Improvement Act (P.L. 102-573) mandated by Congress seek to reduce mortality rates among American Indians due to injury or preventable illness. Misclassification of American Indian populations in state and national health data has lead to underreporting of morbidity and mortality rates for this population.

The problem of race misclassification originates outside of the agencies that publish mortality statistics. The race classification on the state death certificate is usually completed by the funeral director. The person who is responsible for completing the death certificate may not be the same person who performs the funeral service and communicates with the family.

Some reports published by the national office of the Indian Health Service (IHS) routinely exclude or separate mortality data for California American Indians, because of suspected inaccuracies due to racial misclassification. Racial misclassification of American Indians on infant death certificates has been widely acknowledged[3] and reported in studies of infant mortality in California and Montana,[2] Oklahoma,[4] and Washington State.[5] After matching birth files with infant death files, the number of infant deaths for American Indian infants increased from 20% to 150% depending on the study. A recent study in 1993 showed that racial misclassification led to underreporting of injury rates among American Indians in Oregon.[6]

To evaluate trends of health and disease, the California Area Office of the IHS collects and analyzes mortality data among American Indians in California. The objectives of the California Area study of racial misclassification are to: determine the extent of racial misclassification that occurs in mortality data reported for American Indian infants and children in California; provide more accurate data for use in determining the need for resources aimed at preventing morbidity and mortality among American Indians; and ascertain which counties have the highest levels of racial misclassification in order to direct efforts to improve the accuracy of race reporting on California state death certificates.

Methods

Each year in California, approximately 5,000 infants are born whose mother or father are identified as American Indian. The IHS requested the State of California Vital Records Department to provide copies of all birth records for American Indian children born between 1979 and 1993 (the study population). An automated computer mortality linkage system was used to establish the association between study population file records and state mortality file records.[7] The computer program performed a linkage of personal identifiers on the birth certificates of American Indian infants and death certificates for all races in California reported during this same period.

The program utilized a probabilistic evaluation of matches on date of birth, mother's maiden name (available after 1985),

gender, county of residence, and phonetic recognition of first and last names. The record pairs were then classified as either high or low likelihood linkages. If available data failed to meet the minimum requirements for an acceptable match, the record was excluded. Records that were complete but inconclusive (e.g.) a common name with residence in a large county) were labeled as "questionable matches."

The following predictors were tested for association with probability of non-Indian race (misclassification) on death certificates: age at death, date of death, sex, and county of residence at time of death. Fisher's Exact Test was used to obtain *p* values for univariate associations with misclassification and logistic regression was used for multivariate analyses. Statistical analyses were done with and without the "questionable matches"; as there was no significant difference, we included all records in our analyses. Age groups at death were aggregated the same as state reports: under 30 days, one month to one year, 1-4 years old, 5-9 years old, and 10-14 years old. Date of death was dichotomized into two time categories: 1979 to 1986 and 1987 to 1993.

The number of deaths of American Indian children by county was too small for analysis. Instead, residence at time of death was divided into two categories: children whose county of residence was within an IHS-designated Contract Health Service Delivery Area (CHSDA), or those children living in counties outside of the Indian Health Service delivery area. The Indian Health Service provides funds for contract health services to federally recognized land-based tribes living in 38 largely rural counties in California. The 20 non-CHSDA counties encompass most of the urban areas from Sacramento south to Los Angeles.*

In this paper, infants are defined as being less than one year of age; children are defined as being at least one year old and less than 15 years of age. For infant deaths, we determined annual trends in infant mortality from 1979 through 1993 for American Indians compared to all races in California. We were unable to calculate trends in child mortality because of the relatively small number of deaths in this age group. We examined levels of race misclassification for the major leading causes of death for infants and for children. For these analyses, we included all state death certificate records of American Indian children who were born in California after 1978 and who died during our study period. We then merged the additional records of children identified as American Indian by our birth/death file linkage system.

Lastly, in order to target efforts at improving reporting, records were sorted by county of residence at time of death. Although critically ill infants and children may have died in large hospitals outside of their county of residence, race classification on death certificates were usually completed and filed by funeral directors in their county of residence. The number of American Indian deaths reported by each county in the state mortality files were then compared to the number identified after using our computer linkage system.

* California counties excluded from CHSDA services are Alameda, Contra Costa, Los Angeles, Marin, Orange, Sacramento, San Francisco, San Mateo, Santa Clara, Santa Cruz, Kern, Merced, Monterey, Napa, San Benito, San Joaquin, San Luis Obispo, Solano, Stanislaus, and Ventura.

Results

Misclassification error factor. Through computerized linkage, 2240 possible matches were found between American Indian birth and death certificates. The linked files were weighted for the probability of the validity of the linkage on a scale from 1.0 to 10.0 by the computer program. Using only those matches with a high rating of compatibility, we identified 953 American Indian children. Of these, 233 children were coded as American Indian on death certificate records and 720 children had been classified as some race other than American Indian (75.6% of all files, 95% C.I. 72.7% to 78.2%). Based on these linked birth and death record matches, we found that the number of deaths among American Indian children (953) was 4.1 times greater than reported on death certificates alone (233).

During this same 15 year period (1979-1993), 331 deaths were reported among American Indian children under age 15 who were born in this state. The computer linkage system found 233 matches between these death certificates and our American Indian birth file. The remaining 98 records of American Indian deaths may not have been identified by the computerized linkage system if (1) the race of the parents was missing or not identified as American Indian on the birth certificate, (2) an error occurring on the death record may have listed the date of birth before 1979 and would have placed the record outside our study range, or (3) the infant or child's name was incomplete or so different between the birth and death records that a computer match was not detected.

Health care agencies responsible for monitoring the health status of American Indian populations rely on mortality data for measures of health.

By linking birth and death certificates over a 15-year period, we identified four times as many American Indian infants and children who died in California than had been reported on state death certificates. However, our computerized linkage system was unable to find birth and death record matches for all of the identified American Indian children who died in this state during our study period. If we included all of the deaths of American Indian children born in the state, even those not matched to American Indian births, we would come up with a lower estimate of racial misclassification. *Thus, we propose that the actual number of American Indian infants and children who die in California may range between 2.8 and 4.1 times greater than what is reported on state death certificates when adjusted for errors in racial misclassification.*

Race classification on birth and death certificates. Of the 953 matches of American Indian death certificate files to birth records, 233 children (24.4%) were correctly coded as American Indian. Seven hundred and twenty children were incorrectly coded: 676 (70.9%) were coded as White, 39 (4.1%) were coded as Black, and 5 (1%) were coded as other races or unknown on the death record. Hispanic children may be included in any of the racial groups as ethnicity is coded separately from race.

Predictors of misclassification. Misclassification on death certificates was more likely to occur if the child resided outside of the Indian Health Service delivery areas (87.7%) than in CHSDA counties (65.5%). Misclassification was likely to be higher if the child died between 1979 and 1986 (78.8%) than between 1987-1993 (72.6%). Multivariate analyses revealed that the only differences in misclassification by time period were confined to children living in CHSDA counties.

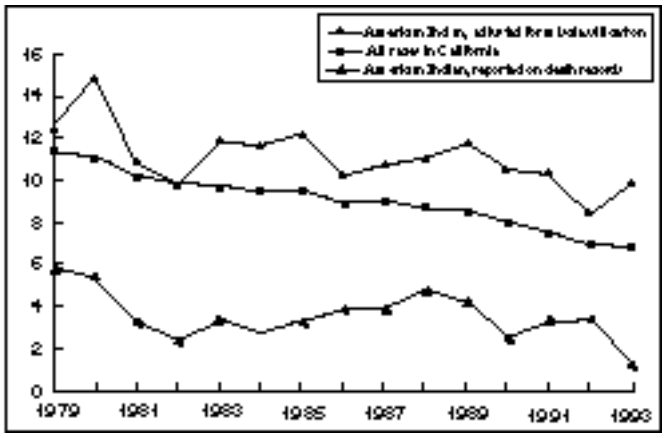
The majority (80%) of the deaths occurred during the first year of life. Nearly half of these deaths (403/834) were to children under one month of age at the time of death. Infants who died under one month of age had a higher likelihood of being misclassified than older children (p=0.072). Fourteen percent of the deaths occurred among children ages one to four, 4% among children ages 5 to 9, and 1% among those 10 to 14 years old. No other variables were identified as predictors of misclassification.

Adjusted-rates for infant deaths. Annual infant mortality rates were calculated for American Indian infants and for all races in California for the years 1979 through 1993. After adjusting for racial misclassification, infant mortality rates for American Indians exceeded that for all races in California during this 15 year period (Figure 1).

Leading causes of death by age group. Misclassification of race for leading causes of death was identified for American Indian infants under one year of age (Table 1) and for children over one year of age through age 14 (Table 2). The numbers of deaths increased markedly for most categories after adjusting for race misclassification. Infants and children who died from injuries were less likely to be misclassified than children who died of other causes.

Counties with highest rates of misclassification. The 58 counties in California are listed by percent of deaths of American Indian children that were misclassified (Table 4). Counties where misclassification of American Indian youth was demonstrated in over 75% of the matched birth-death files would be the target of greatest concern.

Figure 1. Trends in infant mortality among American Indians and all races in California, 1979-1993.



Conclusions

Health care agencies responsible for monitoring the health status of American Indian populations rely on mortality data for measures of health. In this study of racial misclassification of state mortality reports, we found significant levels of underreporting of deaths of American Indian children in California. Without linking birth and death certificates to control for racial misclassification, the state mortality reports identify only one-quarter to one-third of the deaths among American Indian children under 15 years of age in California.

When adjusted for misclassification, annual infant mortality rates for American Indians continued to remain higher

Table 1. Misclassification of race by leading causes of death among American Indian infants (< 1 year old) in California, 1979-1993, determined by linking birth and death files.

ICD-9 Code	Cause of Death	Race Coded AI/AN	Additional Misclassified Files	Total	% AI/AN Deaths Accurately Reported by State
798	Sudden Infant Death Syndrome (SIDS)	72	140	212	34%
740-759	Congenital Anomalies	52	113	165	32%
769	Respiratory Distress Syndrome (RDS)	11	39	50	22%
765	Disorders related to low birth weight (LBW)	18	29	47	38%
800-949	Accidents	11	15	26	42%
	All Other Causes	115	240	355	32%
	TOTAL	279	576	855	33%

Table 2. Misclassification of race by leading causes of death among American Indian children ages 1-14 in California, 1979-1993, determined by linking birth and death files.

ICD-9 Code	Cause of Death	Race Coded AI/AN	Additional Misclassified Files	Total	% AI/AN Deaths Accurately Reported by State
800-949	Accidents	42	46	88	48%
740-759	Congenital Anomalies	8	20	28	29%
140-208	Malignant Neoplasm	4	13	17	24%
960-978	Homicide	6	8	14	33%
390-398, 402, 404-429	Diseases of the Heart	3	7	10	30%
	All Other Causes	25	40	65	38%
	TOTAL	88	134	222	39%

Table 3. Misclassification of deaths among American Indian children less than 15 years of age in California, by county, 1979-1993.

County of Residence	# Indian Race Code	# Other Race Code	Percent Misclassified	County of Residence	# Indian Race Code	# Other Race Code	Percent Misclassified
Merced	0	4	100	Riverside	24	39	62
San Benito	0	2	100	San Francisco	5	7	58
San Luis Obispo	0	6	100	Siskiyou	5	6	56
Santa Barbara	0	5	100	Tuolumne	4	5	56
Stanislaus	0	16	100	Santa Cruz	14	16	53
Sutter	0	4	100	Shasta	12	13	52
Tehama	0	1	100	Amador	1	1	50
Monterey	1	8	89	Colusa	1	1	50
El Dorado	1	6	86	Mariposa	1	1	50
Orange	6	37	86	Trinity	1	1	50
Yuba	1	6	86	Yolo	4	4	50
San Bernardino	12	55	82	Imperial	5	4	44
Ventura	2	9	82	Alameda	21	13	38
Kern	7	29	81	Humboldt	32	12	27
Sonoma	5	21	81	Tulare	11	4	27
Lassen	1	4	80	Kings	3	1	25
Nevada	1	4	80	Mendocino	22	6	21
San Mateo	2	8	80	Lake	5	1	20
Los Angeles	36	135	79	Del Norte	9	2	18
Fresno	5	18	78	Inyo	11	1	8
Santa Clara	8	28	78	Alpine	0	0	0
Butte	1	3	75	Calaveras	1	0	0
Placer	1	3	75	Glenn	6	0	0
Solano	3	8	73	Madera	4	0	0
Sacramento	15	38	72	Marin	1	0	0
San Joaquin	6	14	70	Modoc	2	0	0
Napa	1	2	67	Mono	2	0	0
San Diego	43	88	67	Plumas	1	0	0
Contra Costa	5	9	64	Sierra	-	-	-

than for all races in California for the period 1979 through 1993. The number of deaths of children ages 1 through 14 was too small to estimate annual mortality rates for these age groups.

The magnitude of the misclassification error determined by this study may still underestimate the number of cases of American Indian children who are misclassified at death. Low-likelihood matches were excluded from our analyses if there was not enough information available to determine a reliable match.

Although the numbers of deaths of American Indian infants and children increased for all causes after adjusting for misclassification, there was no significant shift in ranking of the top five causes of death. Deaths due to accidents or homicide were less likely to be misclassified among our matched records than deaths due to other causes. Deaths of children that occur outside of the hospital are more likely to be investigated, which may lead to greater accuracy of reporting.

Several counties were identified where underreporting due to racial misclassification was notable. Almost two-thirds of the 310,000 American Indians in California reside in urban counties where misclassification of Indian race is much more likely to occur. In more rural CHSDA counties, misclassification was less likely among American Indian children who died after 1986. We are not aware of any procedures that may have led to improved reporting of race in CHSDA counties compared to non-CHSDA counties in California. However, there may have been a more heightened awareness of Indians and Indian culture in reservation counties during this later period.

Health care providers in American Indian communities in California have long suspected that American Indian infants and children were at greater risk of illness and death than the general population, yet state mortality data did not support these concerns. The Indian Health Service is currently conducting a study of racial misclassification using IHS patient registration files to identify deaths of American Indians in all age groups. This report, soon to be released, will provide much needed information about racial misclassification of deaths among American Indians nationwide.

The underreporting of American Indian deaths greatly affects the way federal, state, and local resources are allocated to address morbidity and mortality in these communities. This study of racial misclassification will alert health care agencies to the need to reevaluate funding and resources for health programs serving American Indian children.

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The Provider Will No Longer Be Distributed to Professionals Working for Compacted Tribes

Providers working for tribal programs that have compacted with the Indian Health Service and taken their share of the IHS Clinical Support Center (CSC) budget will no longer receive *The IHS Primary Care Provider* (also referred to as *The Provider*), beginning with the January 1997 issue. This decision was made in response to CSC's shrinking budget due to compacting. Those wanting to continue receiving *The Provider* can make a request through their administrative body or compacting organization to negotiate or amend the tribe's Annual Funding Agreement to accomodate the costs associated with production of *The Provider* for the number of copies to be received by the tribal program. At the time of publication of this notice, the CSC cannot take direct payment or a subscription from a tribe or an individual. We will continue to pursue clarification of this issue and will keep our readers informed if and when any changes occur in this regard.

All others receiving *The Provider* will continue to receive it, as in the past. Thank you for your continued support of the Clinical Support Center and *The Provider.*

Simplifying Health Facilities Planning

Henry Cruz, General Engineer, IHS Division of Facilities Planning and Construction, Rockville, Maryland.

The Indian Health Service is at the midpoint in developing a new version of the Health Facilities Planning Manual (HFPM). The present HFPM is a two-volume set of criteria used for planning and designing space for health facilities. Volume 1 is a guideline for planning and allocating space for health care facilities; formulas in this guideline generally require workload and staffing data as input to generate space allocation for use when preparing Program Justification Documents (PJD) and Program of Requirements (POR). Volume 2 is a reference document containing standard layout plates or drawings of rooms, including recommended standard criteria for room finishes, and electrical, mechanical, and communication equipment.

The HFPM revision includes developing 60 to 80 standardized departmental layouts or templates that vary by increments of staffing and workload. These templates can be assembled manually or through the use of a computerized drafting system to generate the layout of health care facilities sized to meet a specific community's health care demands. Each template will include environmental criteria such as for room finishes, and electrical, mechanical, and communication equipment.

The new version of the HFPM is being developed from the IHS concept of providing comprehensive health care delivery from one facility, and takes into account new IHS facilities being proposed for construction within the next ten years. The use of these templates will shorten and simplify the planning and designing processes, which, in the past, have proven to be lengthy and time consuming. These templates can be rotated, flipped, or stacked to meet the requirements of a campus type facility, a rural setting, or a more complex, multi-story, single building at an urban location.

The development of this new version of the HFPM has involved the participation of an architect and engineering (A/E) firm, an IHS Steering Committee, and numerous individuals and workgroups. The A/E has previously worked with the IHS and private sector health care providers, and has developed a similar template approach to designing health care facilities for the private sector. The A/E brings this experience to the project, as well as their understanding of IHS needs. The IHS Steering Committee has acted to guide the overall direction of the HFPM development. This Committee consists of various IHS staff from the Offices of Health Programs; Environmental Health and Engineering; Planning, Evaluation, and Legislation; and Tribal Activities. Service-oriented workgroups, consisting of professional representatives from Area Offices, service units, tribes, and headquarters, have provided specific recommendations on layout, equipment, room criteria, room sizing, etc.

The new version of the HFPM will include template drawings, equipment lists,and computer software necessary to generate the Program of Requirements, and will allow the designers to leap over the first stages of the design process. It is anticipated that the new version of the HFPM will be completed and ready for distribution late in 1997.

For further information, please contact Henry Cruz, IHS Division of Facilities Planning and Construction, Twinbrook Metro Plaza, 12300 Twinbrook Parkway, Suite 600C, Rockville, MD 20857 (phone: 301-443-1852). □

A Case Report and Review
Oculomotor Nerve Palsy Associated with Diabetes Mellitus

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Abstract

Background. *A longstanding clinical dictum maintains that oculomotor nerve mononeuropathy with pupil sparing is most likely secondary to an ischemic vasculopathy, while oculomotor nerve mononeuropathy with pupillary involvement is considered to be secondary to a subarachnoid aneurysm* until proven otherwise. Notably, this dictum fails to accurately predict etiology in a significant number of cases of oculomotor palsy, as many diabetics suffer ischemic oculomotor palsy characterized by initial pupillary dysfunction.*

Methods. *A case report is presented describing a 62-year-old female diabetic patient taking hormone supplements who developed an acute right third nerve palsy with partial pupillary involvement and periorbital eye pain. Although an ischemic etiology was suspected, magnetic resonance imaging and cerebral angiography were performed to rule out an aneurysmal etiology.*

Results. *Neuro-radiologic testing revealed no intracranial pathology, suggesting an isolated, self-limited, microvascular etiology of the oculomotor palsy. The mononeuropathy gradually resolved over the next eight weeks.*

Conclusions. *This case demonstrates that ischemic oculomotor palsy may present with pupillary dysfunction, contradicting the clinical dictum maintaining that spared pupils are associated with ischemia and affected pupils are associated with aneurysm in acute, isolated oculomotor palsy. The discussion section covers isolated oculomotor palsy and its related pupillary postures and offers possible pathophysiologic explanations for the various relationships.*

Introduction

Type II diabetes mellitus, also known as adult onset diabetes mellitus (AODM) or non-insulin-dependent diabetes mellitus (NIDDM), is an extremely prevalent and often overwhelming disease within the American Indian population in the United States. In the Navajo tribe, the nation's largest Indian tribe, with a population exceeding 200,000, a recent study indicated that one in three people over the age of 44 suffer from Type II diabetes.[1] This prevalence rate is 2.5 to 3 times higher than the rate of the U.S. population as a whole, and continues to increase yearly. Compounding the problem of high prevalence, the age at which diagnosis is being made appears to be steadily decreasing as well. Hoy's study[2] of 400 Navajos with diabetes found that 25% of her subjects were diagnosed with Type II diabetes under the age of 40. Recognizing that 50% of the Navajo population is currently less than 20 years of age,[2] it appears possible that an explosion of new cases of diabetes is imminent for the Navajo Indian population; as the population ages, it is likely that there will also be an increasing amount of diabetic morbidity.

Complication rates secondary to Type II diabetes in the Navajo Indian population have previously been reported[2] including hypercholesterolemia (60%), systemic hypertension (58%), nephropathy (54%), retinopathy (41%), and clinical evidence of heart disease (30%). Remarkably, it has been noted that 50% of myocardial infarctions in Navajos are related to Type II diabetes.[3] Additionally, Navajo diabetic patients are ten times more likely to suffer end stage renal disease (ESRD) than the general population.[4] Further, diabetic eye disease has been implicated as a leading cause of blindness in the Navajo population (personal communication with Jim Hughes, OD; Richard Hatch, OD). In total, these figures not only demonstrate the widespread organ damage that results from diabetes, but also suggest a rather ominous future regarding diabetes mellitus in American Indians and Alaska Natives.

Optometrists and other primary care providers in the Indian Health Service must be ready to play a key role in providing regular eye care for their large group of diabetic patients. Although diabetic retinopathy status deserves close attention, other parts of the visual system also require evaluation, as several other structures besides the retina can be adversely affected by diabetes. The following is a case report covering one of the more common ocular, but non-retinal, diabetic complications.

* There are many other possibilities to consider in the differential diagnosis, such as trauma or tumor.

Case Report

A 62-year-old female was referred from our hospital's general medical clinic with acute complaints of watery eyes, mild right peri-orbital pain, a droopy right upper eyelid, and double vision for four days. Her general medical history was positive for adult onset diabetes mellitus (diagnosed one year previously) and post menopausal status. Medications included chlorpropamide, conjugated estrogen, and medroxyprogesterone. She denied any allergies and any family history of diabetes, hypertension, blindness, or other ocular or medical problems. She further denied any trauma or use of any ophthalmic medications. Her first ever eye examination had taken place four months previously and was remarkable only for mild hyperopic astigmatism, mild atrophic maculopathy, and no sign of diabetic retinopathy in either eye.

Eye examination measured uncorrected visual acuities of 20/60 for the right eye and 20/40 for the left eye, with pinhole acuities improving to 20/50 and 20/40 respectively. Pupils demonstrated a 1 mm inequality in the diameter of the pupils (anisocoria) in dark conditions, and a 2 mm anisocoria in bright conditions, but no afferent defect was noted. The left pupil demonstrated normal function, but the right pupil barely reacted to direct light, accommodation,* or consensual stimulus.† Cover testing‡ revealed a marked right hypotropia§ of 15 prism diopters‖ and an exotropia** of 30 prism diopters, associated with a relatively mild 2 mm drooping (ptosis) of the right upper eyelid. Extraocular muscle function of the right eye demonstrated normal inward rotation on attempted medial gaze and normal abduction, but virtually total paralysis in every other direction of gaze. The left eye demonstrated normal extraocular function and no ptosis. Except for extraocular muscle abnormalities, cranial nerve testing was unremarkable and no other neurologic signs or symptoms could be elicited. Slit lamp examination was normal except for mild diffuse bulbar injection of the right eye, and confrontation fields were full in each eye. The patient's condition was diagnosed as an isolated, complete right oculomotor palsy, with sub-total pupillary involvement. To rule out subarachnoid aneurysm, the patient was referred for immediate magnetic resonance imaging and cerebral angiography studies, which were performed the next day. The radiologist reported no clear sign of any aneurysm, mass, or intracranial bleed, which suggested an isolated ischemic event as the etiology for the third nerve palsy, most likely associated with this patient's diabetes mellitus.

The patient was seen again in the eye clinic one week after her initial visit. She stated her double vision was improving, and her periocular eye pain had decreased. Upon examination, she demonstrated unchanged right eye extraocular deficits, but notably, pupil function was now *normal* in the right eye without apparent anisocoria. Cranial nerve testing, except for the extraocular muscle dysfunction, was again unremarkable, and no other changes were noted from the previous exam.

At follow-up one month later, the patient demonstrated residual restrictions in all but lateral gaze in the right eye, but the restrictions were now only partial. She was able to adduct, supraduct, and infraduct to approximately 50% of normal. She had no ocular or visual complaints, and her ptosis had improved moderately. Pupils were again equal and reactive, and no other changes were noted.

Two months later, the patient demonstrated only minimal superior, inferior, and medial restrictions, ptosis was resolved, and the cover test revealed no primary gaze strabismus, although an alternating exotropia could be elicited with some effort. A mild residual ptosis was present, but practically, the third nerve palsy was resolved.

The most common symptoms of oculomotor palsy are unilateral eye or head pain, and complaint of diplopia.

Discussion

Background. The oculomotor nerve, or third cranial nerve (CN III), is a paired motor nerve that functions in conjunction with the trochlear (CN IV) and abducens nerve (CN XI) to produce eye movement. Its trunk consists of motor neurons that innervate several ocular muscles, including the superior, inferior, and medial rectus muscles, the inferior oblique and levator muscles, as well as the internal eye muscles of the iris sphincter (parasympathetically innervated) and the ciliary body.[5-7] The vast majority of the fibers within the third nerve trunk control extraocular motor fibers, with a minority of fibers designated for internal ocular muscle control. These fibers are located in a bundle and run in the superficial part of the nerve. That is why compression can first give only loss of pupillary reflex with preserved eye movements.

Oculomotor palsy (OMP) is characterized by restricted motility, exotropia, hypotropia, and often an ipsilateral ptosis.[5,7] The globe drifts down and out due to the fourth cra-

* Accommodation: During active accommodation (eyes focusing from far to near), both pupils normally decrease in size.

† Consensual stimulus: Normally, both pupils react similarly to a stimulus (light) applied to only one eye.

‡ Cover testing: This tests for muscle balance. The patient looks at a specific fixation point with both eyes. Then the examiner covers one eye with an opaque card or eye cover and while doing so observes the uncovered eye to see if it moves to fix on the object. The examiner then removes the opaque cover from the covered eye and observes for any movement of the eye just uncovered. Movement of either eye is an indication of weakness in one of the extraocular muscles.

§ Hypotropia: A downward deviation of the visual axis of an eye.

‖ Prism diopters: A unit of prismatic deviation; deflection of one centimeter at a distance of one meter.

** Exotropia: Deviation of the visual axis of one eye away from that of the other, resulting in double vision.

nial nerve (superior oblique muscle control) and the sixth cranial nerve (lateral rectus muscle control) functioning without opposition, resulting in a non-concomitant strabismus. Muscular paresis in OMP can be partial or complete, and some muscles may exhibit more paresis than others.[7] Ptosis occurs because of diminished neural input to the levator muscle. Additionally, pupil deficits may occur depending on the type, severity, and location of the lesion causing the oculomotor nerve deficit. Further, other neurologic signs or symptoms may occur depending on the underlying etiology of the third nerve compromise.

The most common symptoms of OMP are unilateral eye or head pain, and complaints of diplopia (double vision). Although pain is generally more prominent and posteriorly located in aneurysmal OMP, and less severe with periorbital location in ischemic OMP, pain characteristics cannot be relied upon to distinguish between ischemic and aneurysmal OMP.[8,9] Diplopia, although frequently absent initially due to the significant ptosis that occurs in acute OMP, often develops later as ptosis generally recovers more quickly than the extraocular movement palsy.[10,11]

The underlying causes of oculomotor palsy are many, and because some of the entities that cause OMP are life threatening, determination of the underlying etiology of the palsy is urgent and critical. Most important in this effort is thorough assessment of concurrent neurologic signs and symptoms, in an effort to localize the offending lesion. Localization of the third nerve lesion guides the appropriate management and/or consultation, and this diagnostic effort requires thorough comprehension of third nerve anatomy.

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Anatomy. The third nerve nuclei originate in the midbrain at the level of the superior colliculus, just anterior to the sylvian aqueduct. The nuclei consist of paired and unpaired subdivisions, a unique anatomical feature that aids in diagnosis of nuclear lesions. Clinically, nuclear lesions demonstrate bilateral ptosis and superior rectus paresis opposite the side of the lesion. This occurs because an unpaired subnucleus controls bilateral levator function but paired subnuclei control contralateral superior rectus function.[12] Thus, without contralateral superior rectus or levator dysfunction, a nuclear lesion is ruled out. These lesions are rare.[5,13]

From the nuclei, each nerve travels as a diffuse collection of fibers through and around the red nucleus, near the cerebral peduncles. This is known as the fascicular portion of the nerve. A fascicular lesion of the third nerve can be diagnosed by the company it keeps. Two well recognized syndromes occur from lesions in this area. Both are characterized by third nerve dysfunction associated with either contralateral hemitremor (Benedikt's syndrome) or contralateral hemiparesis (Weber's syndrome).[5,12,13]

As the nerve exits the fascicular area, it traverses a very narrow passageway between the inferiorly located tentorium cerebelli and the superiorly located uncal portion of the temporal lobe. This anatomical arrangement creates a very high risk for compression of the third nerve, as the temporal lobe is easily displaced downward in cases of intracranial space occupying pathology.[14] The oculomotor palsy that occurs secondary to this downward displacement and squeezing of the nerve may be the initial sign that a subdural hematoma is present.[5,13]

As the nerve emerges from the midbrain, it enters the subarachnoid space and travels adjacent to the basilar, posterior communicating, and internal carotid arteries. This is, of course, the prime area for aneurysmal compression of the nerve.[5,10] Because of the relatively "wide open" space in this area of the brain, lesions in this area are often isolated neurologic phenomena, except of course for headache.

From the subarachnoid space, the nerve advances to pierce the dura and enter the cavernous sinus, where it runs adjacent to several other cranial nerves. Intracavernous oculomotor lesions usually present with multiple cranial nerve deficits due to the close proximity of the oculosympathetic, fourth, sixth, and the upper division of the fifth cranial nerves.[5,13] Oculomotor compromise in this area often produces only partial palsies, and some muscles may be more affected than others.[10,13] Traumatic or aneurysmal lesions, but not ischemic lesions,[11] within the cavernous sinus can lead to aberrant third nerve regeneration, where sprouting axons innervate anomalous target muscles resulting in lid-gaze dyskinesis or pupil-gaze dyskinesis.[11] Intracavernous carotid aneurysms can occur and often result in carotid-cavernous fistula syndrome.[13] Interestingly, these intracavemous aneurysms are associated with only a small risk of death, which is in contrast to subarachnoid carotid aneurysms, which can and often do progress to subarachnoid hemorrhage and death.[13]

Just before exiting the cavernous sinus and entering the superior orbital fissure, the oculomotor nerve splits into a superior division supplying motor fibers for the levator and superior rectus muscles, and an inferior division supplying the medial and inferior rectus, inferior oblique, pupillary sphincter, and ciliary body muscles.[6] Lesions distal to this split will demonstrate deficits consistent with this division and the most likely etiology is a cavernous or retro-orbital mass lesion.[9] It should be noted that the inferior division is rarely spared in ischemic OMP.[11]

Etiology. The two* most common underlying etiologies

* Other possible etiologies include trauma, tumor (orbital apex, superior orbital fissure),and intracranial hypertension. Though these often occur with other cranial nerve findings, they can affect CN III in isolation.

of OMP are aneurysm in the subarachnoid region, and ischemia, commonly associated with diabetes and thought to occur as a "stroke" to the nerve.[11] Unfortunately, these two types of OMP can be clinically indistinguishable, as both ischemic OMP and aneurysmal OMP often occur acutely, painfully, and completely. Further, these two types of OMP tend to occur in isolated neurologic fashion, unlike many of the other forms of OMP which usually present with associated neurologic findings.[9,11,15] Because of the clinical similarity, the relatively high incidence of these two types as the underlying etiology of isolated OMP, the markedly different follow-up required, and the markedly disparate potential outcomes in each of these etiologies, proper differential diagnosis between these two types is both vital and difficult.

Historically, pupil status has been used to aid diagnosis of OMP etiology. A longstanding clinical dictum maintains that if a patient over the age of fifty years presents with an acute, isolated, complete OMP with an unaffected or spared pupil, the etiology is highly likely to be ischemic in origin, especially if that patient has concurrent hypertension or diabetes mellitus. However, if that patient's pupils are fixed and dilated, the OMP is generally considered to be secondary to an aneurysm at the junction of the internal carotid and posterior communicating arteries until proven otherwise.[11]

Supporting the dictum are pathologic reports that demonstrate that between the brainstem and the cavernous sinus, the third nerve's parasympathetic pupillomotor fibers are situated in a bundle or tract, positioned externally within the nerve trunk at a superior and slightly medial location.[16-18] This superficial location of the pupillary fiber tract allows these fibers to be easily compressed and compromised in any sort of external lesion, such as aneurysm. Conversely, this tract remains relatively "spared" and unaffected in microvascular ischemic events, which strike the core of the nerve, physically distant from the pupillary fibers.[12,17,19]

In addition to the pupillary fibers' peripheral location, two other factors may be associated with the phenomenon of pupil sparing in ischemic OMP. First, and related to the pupillary fiber tract's external/superficial location, it has been proposed that pupil sparing may occur because of a dual blood supply to the oculomotor nerve. Although the third nerve's primary blood supply is via the vasa nervorum vessel that basically runs through the middle of the nerve trunk, it also appears that an additional blood supply from externally located pial vessels may feed the trunk's peripheral nerve fibers, including the parasympathetic pupillary fiber tract.[12] This arrangement would give the pupillary fibers a separate or supplemental blood supply which could preserve relatively normal pupil function during a vasa nervorum occlusive event, assuming a vasa nervorum stroke as the ischemic event in OMP.

Additionally, it has been postulated that because myelin tissue is susceptible to ischemia, and because the extraocular fibers are myelinated and the pupillary fibers are unmyelinated or less myelinated, the extraocular muscle fibers are more likely to suffer functionally significant compromise in ischemic insult when compared to the pupillary fibers.[14,16] Thus, along with factors of external physical location and supplemental blood supply, myelination characteristics of the pupillary fibers may help to explain the phenomenon of pupil sparing in ischemic OMP.

Another explanation of pupil sparing has recently been proposed. Traditionally, it has been commonly and widely believed that the loci of third nerve ischemic insult is within the intracavernous or subarachnoid portions of the third nerve, a belief based on previous pathologic study of just three ischemic OMP patients.[10,19] However, new studies challenge those beliefs and indicate that ischemic OMP may occur more commonly from midbrain/fascicular insult of the third nerve than peripheral insult of the third nerve.[10,20,21] Supporting this hypothesis are documented cases with computerized tomography (CT)-confirmed midbrain ischemic insult and pupillary sparing isolated OMP.[14]

Despite all of the aforementioned support for the pupillary OMP dictum, the reality is that the dictum fails to predict OMP etiology in a significant number of cases. Specifically,

The definition of "significant pupillary dysfunction" however is controversial. . . .

three separate presentations contradict the dictum. First, numerous studies have found that pupil sparing does not occur in 15% to 20% of ischemic OMP cases.[13] Thus, one in five cases of acute, isolated, complete, OMP caused by ischemia demonstrates at least some pupillary dysfunction. Although the pupil dysfunction that occurs in these cases is frequently only partial rather than total,[5,9,11] and returns to normal within a week, pupillary compromise does occur with ischemic OMP and is not an uncommon occurrence. To explain this, it may be that in very severe infarctions of the vasa nervorum, the protection of peripheral location, additional blood supply, and non-myelinated anatomy are not enough to prevent clinical pupillary dysfunction. In fact, the amount of pupillary dysfunction may be an indicator of the severity of the ischemic event.

Secondly, there have been reported cases of isolated, complete OMP secondary to angiography-proven aneurysms at the junction of the posterior communicating and internal carotid arteries (ICA/PCoA) which have presented with initial findings of partial or no pupillary dysfunction.[9,10,22,23] In these cases of aneurysmal OMP with pupil sparing, which are much less common than ischemic OMP with pupil involvement, the "pupil sparing" may be explained by aneurysmal compression initially located away from the pupillo-sphincter fiber tract.[11,14] Notably, although these cases demonstrate initial pupillary sparing, the natural evolution of the cases char-

acteristically results in total pupillary dysfunction within a few days.[9,10]

Thirdly, it is possible to find either a miotic pupil or an equal but non-reactive pupil in association with OMP. These presentations, which should not be confused with pupil sparing, can occur when the pupillo-dilator fibers (oculosympathetic bundle) become compromised in a cavernous sinus lesion.[10,11,14] Fortunately, in these cases, the OMP is usually not of an isolated nature as additional neurologic signs or symptoms are often present to help localize the lesion to the cavernous sinus.

In the presented case, after careful history assessment and clinical examination, the patient was diagnosed with an acute, isolated complete third nerve palsy with partial pupillary involvement. The patient's history of diabetes mellitus and the patient's use of estrogen put her at risk for thromboembolic ischemic events, and an ischemic microvascular compromise was the most likely etiology for her acute onset third nerve palsy. However, due to the finding of pupillary compromise in this patient, immediate referral for further testing was indicated to rule out an ICA/PCoA aneurysm, with its potential for imminent rupture and associated life-threatening sequelae. It is generally accepted that any OMP with significant pupillary dysfunction should receive head CT or MRI (magnetic resonance imaging) and cerebral angiography or MRA (magnetic resonance angiography).[10] The definition of "significant pupillary dysfunction" however is controversial as some doctors choose to image patients with partial pupillary dysfunction and some doctors do not image these patients.

It is recommended that follow-up evaluation should occur every 48 hours for the first 7 to 10 days in cases of acute, complete, isolated oculomotor nerve palsy with pupil sparing in an older patient. During those follow-up examinations, special attention must be paid to new neurologic signs or symptoms which would implicate a non-ischemic etiology, including changes in pupillary function. Evolution of pupillary status can often provide valuable clinical information in OMP as both ischemic and aneurysmal groups display a time course of a few days with progressive worsening of pupillary status in aneurysmal cases, and progressive improvement of pupillary status in ischemic cases.[9,11] Resolution of ischemic OMP is expected to gradually take place within 2 to 6 months.[9] If the condition worsens, no measurable resolution occurs within four weeks, or aberrant third nerve regeneration occurs, further workup is indicated.[9,10] Additionally, if pupil-sparing OMP occurs in a young (less than 50 years old) individual without significant vascular risk factors, if it occurs as an inferior or superior branch OMP, or if it occurs in incomplete fashion, even if pupil sparing is present, further testing is indicated as well.[9-11]

In summary, the only cases of OMP in which clinical monitoring as the sole mode of management is reasonable is in cases occurring in older individuals, particularly those with vascular disease, where the presentation is acute, complete, isolated, and pupil-sparing. All other cases deserve further workup, depending on the particular characteristics of the case. Certainly, any case with significant pupil dysfunction deserves special attention, due to the possibility of a life-threatening underlying lesion.

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MEETINGS OF INTEREST ☐

Diabetes Translation Conference
March 23-26, 1997 San Diego, CA

The National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP) of the Centers for Disease Control and Prevention (CDC) is sponsoring this conference with the theme "Daring to Make a Difference in the 21st Century." The meeting will be held at the San Diego Marriott Mission Valley in San Diego, CA. For more information, contact Laura Timperio at the conference logistics company, Professional and Scientific Association, at 404-633-6869 or the NCCDPHP, CDC, 4770 Bufford Highway, Atlanta, GA 30341 (phone: 770-488-5004).

Southwest Regional Pharmacy Seminar
May 30-June 1, 1997 Scottsdale, AZ

This annual continuing education seminar is held for IHS- and tribal-employed pharmacists working in the IHS Phoenix, Navajo, Albuquerque, Tucson, California, and Portland Areas. More information and an agenda will be available in early 1997. For more information, contact Stephan Foster, PharmD, IHS Clinical Support Center, 1616 East Indian School Road, Suite 375, Phoenix, AZ 85016 (phone: 602-640-2140; fax: 602-640-2138).

Mid-Level Primary Care Providers
June 3-6, 1997 Scottsdale, AZ

This continuing education conference for mid-level providers (nurse practitioners, nurse midwives, physician assistants, and pharmacist practitioners) employed by the Indian Health Service (IHS) or the tribes is designed to meet the learning needs of those providing primary care to American Indians and Alaska Natives. An agenda will be available in Spring 1997. For additional information, contact the IHS Clinical Support Center, 1616 East Indian School Road, Suite 375, Phoenix, AZ 85016 (phone: 602-640-2140; fax: 602-640-2138).

IHS/Tribal Nurse Educators
June 18-19, 1997 Scottsdale, AZ

The fourth annual conference for nurse educators is scheduled to be held in Scottsdale, Arizona, pending funding for the conference. Nurse educators (nurses who provide inservice, continuing education, and/or orientation to nursing staff) employed by the Indian Health Service (IHS) or the tribes, and other interested persons are welcome to attend. It is recommended that those interested in attending begin now to identify funds to cover their transportation and per diem.

An agenda and registration materials will be available in Spring 1997, and can be obtained by writing to the IHS Clinical Support Center, 1616 East Indian School Road, Suite 375, Phoenix,AZ 85016 (phone: 602-640-2140; fax:602-640-2138).

NURSING CONTINUING EDUCATION MATERIALS AVAILABLE

The IHS Clinical Support Center has designed the following home study materials for use by nurses. To obtain continuing education credits, an individual must read the materials in the module, take and pass the post-test, and complete the evaluation form. These activities have been planned and produced in accordance with the criteria established by the American Nurses Credentialing Center Commission on Accreditation (ANCCCA). The Indian Health Service Clinical Support Center is the accredited sponsor.

- *Incorporating Critical Thinking Into Nursing Practice.* At the completion of this learning activity, nurses will be able to (a) define the concept of critical thinking, and (b) apply critical thinking to a clinical setting.

- *Improving Case Management Using Critical Care Pathways.* At the completion of this learning activity, nurses will be able to (a) discuss the generic principles of critical pathways,(b) state the steps to develop and design critical pathways, (c) describe one method to document variances, and (d) apply the concepts of critical paths to diabetic clients from the emergency room through inpatient care.

It is expected that each of these learning activities will take participants approximately 2-3 hours to complete. Participants who complete the learning modules and pass the post test will be awarded 3.0 contact hours.

How to Obtain Materials

Nurses employed by Indian health programs may request these continuing education materials by writing to the IHS Clinical Support Center, 1616 East Indian School Road, Suite 375, Phoenix, Arizona 85016 (fax: 602-640-2138).

NCME VIDEOTAPES AVAILABLE ☐

Health care professionals employed by Indian health programs may borrow videotapes produced by the Network for Continuing Medical Education (NCME) by contacting the IHS Clinical Support Center, 1616 East Indian School Road, Suite 375, Phoenix, Arizona 85016.

These tapes offer Category 1 or Category 2 credit towards the AMA Physician's Recognition Award. These CME credits can be earned by viewing the tape(s) and submitting the appropriate documentation directly to the NCME.

To increase awareness of this service, new tapes are listed in The IHS Provider *on a regular basis.*

NCME #701

Clinical Case Reviews: Management of Severe Chronic Pain (60 minutes) Despite advances in the use of analgesics, adequate pain relief remains a major quality of life issue for patients with severe chronic pain. Primary care physicians often question their ability to control pain in these patients. The use of opioid drugs is gaining acceptance for a selected subpopulation of patients with chronic pain, but many questions remain. How do I safely prescribe these drugs? What about breakthrough pain? Will the patient become addicted? What are the pharmacolegal considerations? Our distinguished faculty (Russell K. Portenoy, MD; Patricia M. LoRusso, DO; and Mathew Lefkowitz) will answer these questions and provide you with the latest clinical information as it explores three challenging cases that illustrate effective pain management in patients with non-cancer, cancer, and HIV-related pain.

NCME #702

Psychiatric Challenges for the Primary Care Physician: Social Phobia (60 minutes) Social phobia — the very nature of the condition makes it a diagnostic and therapeutic challenge for primary care physicians and psychiatrists. Patients avoid scrutiny and have extremely well-developed defense mechanisms that prevent physicians and therapists from seeing the issues that torment them. In this program, two experts on social phobia, Drs. Jonathan R.T. Davidson and Nicholas L. Potts, present three case studies based on actual patients, recreated through the use of actors. The signs and symptoms physicians should use to make the diagnosis of social phobia as per the DSM-IV are presented. Also, the available therapeutic regimens, both pharmacologic and cognitive behavior therapy, are examined.

The following is an updated MEDLINE *search on Native American medical literature. This computer search is published regularly as a service to our readers, so that you can be aware of what is being published about the health and health care of American Indians and Alaska Natives.*

The Clinical Support Center cannot furnish the articles listed in this section of The Provider. *For those of you who may wish to obtain a copy of a specific article, this can be facilitated by giving the librarian nearest you the unique identifying number (UI number, found at the end of each cited article).*

If your facility lacks a library or librarian try calling your nearest university library, the nearest state medical association, or the National Library of Medicine (1-800-272-4787) to obtain information on how to access journal literature within your region. Bear in mind that most local library networks function on the basis of reciprocity and, if you do not have a library at your facility, you may be charged for services provided.

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