Clinical management of nutritional nightblindness

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Abstract
Night blindness is a frequent patient complaint. Vitamin A deficiency has long been known to cause night blindness. More recently, zinc deficiency has also been shown to cause night blindness. Diagnosis of nutritional night blindness is made difficult however, by the lack of associated clinical signs, especially in the case of marginal deficiencies. In order to identify those tests the practitioner should conduct when nutritional night blindness is suspected, results from several diagnostic tests were compared between a group of patients with the complaint of night blindness, and an age and sex matched group without the complaint. Dark adaptometry testing, the classical test for night blindness, confirmed that the patient complaint, when properly elicited, is of high validity in diagnosis of night blindness. The American Automobile Association's Night Sight Meter, a screening device, was found to produce results which correlated significantly with dark adaptometry results, but was of little value as a screening test for night blindness. Results from nutritional testing showed no differences between the two groups for serum vitamin A, concentration vitamin A intake as assessed by diet survey, or zinc levels from hair analysis. It was concluded, therefore, that dietary supplementation may be the test of choice in evaluation of nutritional sufficiency.

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Clinical Management of Nutritional Nightblindness

Roger R. Hirons

A Thesis submitted to the Pacific University College of Optometry
in partial fulfillment of the requirements for the Degree of Doctor of Optometry

1982

Diane P Yolton, Ph.D.
Faculty Advisor

Night Vision

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NOTE:

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ABSTRACT

Night blindness is a frequent patient complaint. Vitamin A deficiency has long been known to cause night blindness. More recently, zinc deficiency has also been shown to cause night blindness.

Diagnosis of nutritional night blindness is made difficult however, by the lack of associated clinical signs, especially in the case of marginal deficiencies.

In order to identify those tests the practitioner should conduct when nutritional night blindness is suspected, results from several diagnostic tests were compared between a group of patients with the complaint of night blindness, and an age and sex matched group without the complaint.

Dark adaptometry testing, the classical test for night blindness, confirmed that the patient complaint, when properly elicited, is of high validity in diagnosis of night blindness. The American Automobile Association's Night Sight Meter, a screening device, was found to produce results which correlated significantly with dark adaptometry results, but was of little value as a screening test for night blindness.

Results from nutritional testing showed no differences between the two groups for serum vitamin A, concentration vitamin A intake as assessed by diet survey, or zinc levels from hair analysis. It was concluded, therefore, that dietary supplementation may be the test of choice in evaluation of nutritional sufficiency.
Night blindness or lowered visual function in dim lighting is a frequently elicited patient complaint. Often, the complaint is only rather vague such as difficulty indentifying objects at dusk or trouble in driving a car at night, but none the less may constitute a serious reduction of the patient's visual function. Known causes of night blindness (nyctalopia) may be congenital (retinitis pigmentosa, hereditary optic atrophy) or acquired (glaucoma, optic atrophy, cataract, retinal degeneration, vitamin A deficiency, zinc deficiency).

Dark adaptometry, wherein visual threshold of light is determined sequentially as the visual system dark adapts from photopic to scotopic function, is the classical diagnostic test for night blindness. Unfortunately, dark adaptometry requires expensive instrumentation as well as lengthy, tedious examination and so has remained primarily a research tool.

Since dark adaptometry testing is only infrequently utilized, diagnostic conformation of night blindness is commonly restricted to manifestation of clinical signs or familial history. However, in cases of early or marginal vitamin A or zinc deficiency, clinical signs are lacking. Given the ubiquitous and perhaps vague nature of the night blindness complaint, it may be diff-
icult for the practitioner to know if nutritional testing or treatment is appropriate without additional diagnostic conformation.

The purpose of this investigation was to determine the validity of the subjective complaint of night blindness; that is, of those with the complaint, how many demonstrate diminished visual function on objective testing. Further, to determine what proportion of the patients have vitamin A or zinc deficiencies as specific etiological causes of their problems. Lastly, to evaluate the use of a low-cost, rapid night vision screening device, the Night Sight Meter (developed by the Automobile Association of America (AAA)) as a tool in the diagnosis of night blindness.

To facilitate an understanding of the experimental design of this investigation, vitamin A and zinc metabolism and diagnostic tests for night blindness will first be reviewed.

**Vitamin A/Zinc Metabolism and Vision**

Night blindness was probably the first nutrient-deficiency disease clearly recognized. Ancient Egyptians, as early as 2500 BC, treated night blindness with liver extract (now known to be rich in Vitamin A) applied topically to the eye. Throughout history, liver consumption has remained as both a therapeutic and preventative measure against night blindness.

Only during this last century has vitamin A (retinol) been identified as the active agent in liver and its role in
vision been revealed, predominantly thru the Nobel Prize winning work of Wald. In addition to its importance in vision, vitamin A plays an important role in maintenance of epithelial tissues (including the conjunctival and corneal epithelium) and in bone formation.

Two dietary sources exist for vitamin A; retinol from animal sources (liver, egg yolk, and dairy products) and carotenoids from plant sources (carrots, tomatoes, sweet potatoes, and leafy green vegetables), with beta-carotene being the primary source. Carotene is split and reduced to yield two retinol molecules in the small intestinal mucosa, bound preferentially to palmitic acid and then transported via the lymphatics to the liver. Since it is fat soluble, retinol is stored throughout the body but retinyl palmitate stored in the liver is the body's primary reserve. Retinol is transported in the plasma to peripheral organs and tissues bound to a specific plasma protein (retinol-binding protein or RBP). Adequate serum levels of retinol are maintained through controlled synthesis and release of RBP.

Once at the retina, retinol is oxidized to retinal by the enzyme retinol dehydrogenase, isomerized from all trans to 11-cis retinal, and bound through Schiff base linkage to the apoprotein opsin to form the photolable visual pigments. With light capture, the retinol component is isomerized (photoisomerization) back to the all-trans form and separated from the opsin, initiating an electrical response leading ultimately to vision.
separated from the opsin, the retinal is rapidly reduced to retinol, most of which is recycled to reform the active photopigment. However, there is some loss due to diffusion from the retina, peroxidative degradation, or through oxidation to retinoic acid. Due to this and other losses, constant dietary replacement of vitamin A is required for maintenance of visual function.

Insert figure 1 about here

The importance of assessing vitamin A nutriture as a cause of a patient's night blindness is that in cases of inadequate vitamin A nutriture (hypo vitaminosis A), night blindness is the initial symptom in a sequence that may eventually lead to total blindness as a result of xerophthalmia. The sequence of clinical signs and symptoms lead from night blindness to conjunctival xerosis (possibly including Bitot spots) and finally to corneal xerosis. Clinical intervention prior to advanced corneal xerosis with appropriate vitamin A therapy will, except in cases of severe malnutrition, reverse this progression. While the U.S. population is not considered one at risk of these advanced sequelae, vitamin A deficiency is considered a major cause of blindness in the third-world nations with an estimated 100,000 new cases yearly.

Although the portion of bodily retinol in the eye is quite small (0.01%) and the liver store is sufficiently large to maintain body needs for 7 months to 1 year, there are a
number of evidences to suggest that vitamin A nutriture may be inadequate in a substantial portion of the U.S. population. Several recent autopsy studies have indicated low liver reserves of vitamin A in upwards of one-third of a normal population. In the largest, most comprehensive dietary survey of the U.S. population to date, mean vitamin A intake was found to be only slightly above the survey's standard for dietary intake (3,500 international units). However, the distribution of vitamin A intake is highly skewed, reducing the meaningfulness of mean data, and the current recommended daily dietary allowance is higher than the value used as the standard in the survey. Reanalysis of the data using today's standard (5,000 I.U. for men, 4,000 I.U. for women) reveals that not only is the mean intake for the entire population substandard, but that for some subsets of the population, in excess of 75% were undernourished in vitamin A. Additionally, trend analysis of vitamin A intake shows per capita intake to be declining. Despite recent warnings of increasing incidence of vitamin A toxicity due to megavitamin regimens of healthfood faddists, hypovitaminosis A or marginal vitamin A nutriture would seem to be much more common.

A patient's vitamin A nutriture can be assessed by either diet evaluation or serum analysis. Diet evaluation, most commonly employing computerized analysis of a patient compiled diet survey, is probably the poorer method of determining
biological sufficiency due to inherent inaccuracies and a host of factors affecting vitamin A absorption once in the intestine. Unfortunately serum levels of vitamin A, which are of value in evaluating populations, are of limited value in assessing deficiency in the individual. While low serum vitamin A levels indicate a high probability of impaired visual function, serum levels in the "normal" range, due to an apparent logarithmic rather than linear relationship of serum vitamin A and night vision, do not exclude reduced visual function/vitamin A deficiency.

Zinc has only much more recently been found to be critical in human visual function. Every tissue throughout the body contains zinc and its functions are many and varied. Two factors of vitamin A metabolism are contingent on adequate zinc nutriture. First, zinc is required for mobilization of retinol from the liver. While the exact mechanism is unknown, retinol-binding protein synthesis is retarded in animals fed zinc deficient diets, resulting in lowered serum vitamin A levels. Secondly, at the retina, zinc is involved in retinol dehydrogenase enzyme activity which is required for conversion of incoming retinol from the blood and recycling that retinol which is present from past use. Recently, in a study of zinc deficient rats, retinol dehydrogenase enzyme activity was found to be significantly reduced. (see figure 1).

Additional evidence of zinc's role in vitamin A metabo-
lism was gained in a study of patients with alcoholic cirrosis of the liver. When these patients with night blindness were unresponsive to vitamin A supplementation, zinc supplementation was found to be effective in restoring normal visual function.

Presently, it appears that marginal zinc nutriture is more common than is frank deficiency. While prevalence data of zinc deficiency in the U.S. population is lacking, Hambridge, in 1976, found 68% of a low growth percentile sample of children from low economic background to have inadequate zinc nutriture. Primary zinc sources include liver and muscle meats. Other than liver, which is not included in many people's diets, zinc and vitamin A come from different sources: A well balanced diet, therefore, is required for optimal dietary supply of both zinc and vitamin A.

Adequacy of zinc nutriture is much more difficult to assess than is vitamin A. Serum levels, which are useful in identifying chronic vitamin A malnutrition (after depletion of liver reserves), are of less value in identifying depressed zinc nutriture as homeostatic mechanisms are thought to exist that serve to keep serum zinc levels remarkably normal by shunting zinc from one tissue to another, thus preserving the most vital functions. For this reason, hair zinc concentration may be the best measure of chronic zinc deficiencies. Ironically, due to retarded hair growth, zinc deficiency is identified by excessive hair zinc levels.
**Darkadaptometry and Night Blindness**

Dark Adaptation is the transition of the retina from the light-adapted or photopic state to the dark-adapted or scotopic state. During this adaptation, the eye increases in sensitivity about 100,000 fold. In dark adaptometry, this change in photosensitivity is determined by measurement of lumenance threshold as a function of elapsed time in darkness and plotted to obtain a dark adaptation curve (see figure 2)

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Insert figure 2 about here

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The upper portion of the curve represents cone adaptation with the first plateau indicating the cone threshold. The inflection in the curve (at 6 to 8 minutes) is called the rod-cone break and indicates the retina's shift from cone to rod vision. The lower portion of the curve is attributed to rod vision. The lower portion of the curve is attributed to rod function and indicates final rod threshold (for practical testing purposes) at approximately 30 minutes. Because of the sensitivity of dark adaptometry, it has been utilized to assess vitamin A storage in liver disease, vitamin A nutriture in malnourished populations, and in establishing recommended levels of vitamin A intake.

Since night blindness is the first clinical manifestation of deficient vitamin A nutriture, dark adaptometry has become an important tool in diagnosis of hypovitaminosis A.
Retinal is incorporated in all photopyments, therefore, dark adaptometry testing of hypovitaminosis patients can reveal an increase of both rod and cone thresholds as well as a delay in the transition from cone to rod vision (see figure 2). Final rod threshold, however, is the most repeatable of the three findings and is consequently the measure commonly used for diagnostic confirmation of night blindness.

Consistent dark adaptometry results have been shown to depend upon patient age and careful control of several variables, including preadaptation brightness and duration, the test spot's size, color, and location, pupil size, and test technique. The Goldmann-Weekers Darkadaptometer allows for precise control of these variables and represents the state-of-the-art in dark adaptation testing.

While the Goldmann-Weekers Darkadaptometer has yielded reliable and repeatable results, its expense, fragility, and lengthy exam sequence have limited its utilization outside the research laboratory. Consequently, screening tests have been developed as alternatives to dark adaptometry for diagnosis of hypovitaminosis A. While often considered too subjective to be of value, the complaint of night blindness (or "chicken-blindness" in many third world countries) has been shown to be of high diagnostic utility, particularly when testing young children in field situations.

Thornton has recently described a screening test based
on the Purkinje shift. This test requires the patient to sort photopically lumance balanced red and blue buttons under scotopic lighting conditions. Correct sorting is impossible until after the retinas shift from cone to rod vision when the Purkinje shift allows sorting based on luminance differences. Since hypovitaminosis A may cause an increased time to the rod-cone break, vitamin A deficient individuals may take significantly longer to correctly complete the sorting task.

The AAA Night Sight Meter is a screening device that tests visual recognition in the mesopic range. Measurement of glare vision, night vision, and glare recovery are obtained by having the patient identify the orientation of a series of Landolt C's (20/300) as they rotate past a small window at the rate of 45 per minute. The glare source and test target were engineered so as to simulate the headlights of an oncoming car at 150 feet with a pedestrian walking on the edge of a 20 foot pavement ahead of a driver. Hunter, in a sample of 100 clinic patients, found 26% to have reduced visual function as measured by the Night Sight Meter. When supplemented with vitamin A and D, 90% of these patients returned to normal ranges, suggesting that the Night Sight Meter may be of value in diagnosis of vitamin A deficiency.
METHODS

Eighteen experimental subjects were selected from respondents to local newspaper articles requesting subjects for a research project involving night vision difficulties and or night blindness (3 male, 15 female/ ages 24 to 46, median age 30).

Respondents were first screened on the basis of their visual complaint. Subjects were considered to have a valid night blindness complaint when, in addition to difficulty driving at night (all respondent's chief complaint), they complained of not being able to see objects at all (rather than not seeing objects clearly) and difficulty while walking outdoors at night (e.g., tripping over unseen objects, difficulty in negotiating stairs, etc.). All subjects were required to have no family history of eye disease (excepting senile cataracts) or night blindness and be in good health. Best correctable visual acuity of 20/20 or better and an age limit of 50 was imposed so as to reduce confounding effects due to reduced ocular media clarity. Additionally, an ophthalmoscopic examination was conducted so as to exclude from participation those with evidence of ocular disease.

A control group, comprised of healthy individuals without night vision complaints was then selected. To control for differences in dark adaptation and with age and sex, the control group was both age and sex matched to the experimental
As an additional measure to identify night myopia as the basis for the night vision complaint, a bichrome test, using an isolated 20/40 line without room-illumination was administered. In no case, was the red-green balance in excess of -.25D from the subjective to test visual acuity refraction or -.50D from the habitual correction.

The subjects were next tested using the Night Sight Meter. The test was conducted in a darkened room, using the procedure suggested by the AAA.

Glaire vision was tested first. With the glare source on, and the target illumination at maximum, the subject was instructed to watch Cs as they passed into view and call out the orientation of the opening. Target-illumination was then reduced until the subject could no longer correctly identify the orientation. Night vision was next tested using the same procedure except with the glare source off. Lastly, glare recovery was tested by starting with the glare source and the target illumination at maximum. After a minimum of 10 seconds, the glare source was extinguished and, simultaneously, target illumination reduced to the previously determined night vision level. In each phase of the testing, the subject was given one practice trial with the average of three subsequent trials recorded as the test result.

Monocular dark adaptometry testing, utilizing a Goldmann-
Weekers Darkadaptometer was conducted next. Pupil size was controlled by dilation of each subject utilizing 1.0% tropicamide.

After pupil dilation to 7 - 8 mm, subjects were preadapted to a diffuse white light of approximately 200 millilamberts (mL) for 5 minutes. Threshold determination began immediately following preadaptation, utilizing a uniform white test field of the standard size (retinal subtense of 11'). Retinal area tested was controlled by having the subjects fixate a small red spot 20' above the test field.

Threshold determinations were taken in a series of ascending (subject first able to detect) and descending (subject no longer able to detect) trials using an intermittent illuminance test field. The average of ascending and descending trials was used in determining final cone and final rod thresholds, with visual inspection of the averaged curves used to determine rod-cone break time. Testing continued until the threshold values had stabilized (usually 30-35 minutes).

A one gram hair sample was then obtained. Only the most recent (closest to the scalp) two inches of hair, gathered from the nape of the neck, was used for analysis. Analysis for zinc content was conducted by Mineral Labs Inc., Havard, Ca.

Subjects were given a dietary survey form to complete at their leisure. The survey vehicle used required the subjects estimate their monthly, weekly, or daily consumption of 232
food types and, additionally, includes the subjects (vitamin) supplement history. The diet survey was then computer analysed by Mineral Labs, Inc. for vitamin A and zinc intake.

Finally, blood samples were drawn and analysis of serum vitamin A were conducted, using the trifluoroacetic acid method of Neeld and Pearson.

RESULTS

Means and standard deviations on all vision tests and nutrition tests were calculated for experimental and control groups. These data are presented in table 1.

Insert Table 1 about here.

One-tailed t-test analysis revealed significant differences on dark adaptometry final rod threshold, Night Sight Meter glare vision, and Night Sight Meter night vision tests (.05 level of confidence was used in all statistical analyses. Due to the markedly skewed distribution of Night Sight Meter glare recover results, the Mann-Whitney U-test was used for analysis of this result. Glare recovery was found to be significantly reduced in the experimental group. Differences between experimental and control groups for dark adaptometry final cone threshold and rod-cone break time, serum vitamin A, diet intake vitamin A, and hair zinc were not significant.

Regression analysis was conducted to correlate serum vitamin A levels, vitamin A intake, and hair zinc with scotopic visual function (dark adaptometry final rod threshold). Addi-
itionally, vitamin A intake was correlated with serum vitamin A. Scatter plots of correlational tests are shown in figures 2-5. Linear correlation to final rod threshold was found to be significant for Night Sight Meter glare vision and night vision tests. Significant correlations were not found for serum vitamin A, vitamin A intake, hair zinc, or Night Sight Meter glare recovery and final rod threshold. Although serum vitamin A and vitamin A intake were found to be significantly correlated, removal of the three subjects who had abnormally high levels of vitamin A supplementation (i.e. restricting analysis to normal range) resulted in a nonsignificant correlation.

Insert figures 2-5 about here

DISCUSSION

The significant difference found between the experimental and control group final rod threshold supports the hypothesis that the patient complaint is of diagnostic value in diagnosis of night blindness. While this may seem rather obvious, many of the experimental subjects remarked that they had, in the past, been assured by various health care providers that their problems were shared by most everyone and that they should not be concerned by it. If diagnosis of night blindness were based on final rod threshold being in excess of 1 SD from mean normal rod threshold, the patient complaint
would, as a screening instrument for night blindness, have a sensitivity of 82% and specificity of 79% (see table 2).

The most probable explanation for significant differences in final rod threshold but not for cone threshold or rod-cone break time rests in the nutritional status of the population tested (see figure 6). Reduced Rod sensitivity is the first dark adaptometer result affected with vitamin A depletion. While increased cone threshold and delayed rod-cone break time have been frequently reported, most of these changes involve individuals with a variety of diseases resulting in gross vitamin A deficiency. The population studied in this investigation were of good health and, presumably, had only marginally deficient vitamin A/zinc nutriment.

The AAA Night Sight Meter was found to be of limited value in diagnosis of night blindness. While significant differences were seen between the experimental and control groups for all three of the individual test, the proximity of the group means and the overlap of ranges make establishment of pass-fail criterion that yield adequate test sensitivity and specificity impossible (see figure 7).
Although not directly tested in this investigation, it is probable that Thornton's screening test for night blindness would have also proved of limited value with this population since Thornton's test is essentially a measurement of rod-cone break time which was found to be of significant difference between experimental and control groups. Again, this reflects the marginal rather than gross nutritional deficiencies expected in the experimental group.

**Nutritional Assessment**

That significant difference was not found between experiment and control groups for serum vitamin A is attributable, in part, to the large variation seen within both groups (see figure 8) and the lack of correlation of serum vitamin A and final rod threshold. This lack of correlation, consistent with most research, is the result of a wide range of serum vitamin A levels required to fulfill physiologic function amongst individuals. While one recent study (including diseased subjects) demonstrated a logarithmic relationship between serum vitamin A and final rod threshold, the nature of the curve was such that correlation would not be expected in ranges of normal or marginal vitamin A nutriture. Thus, it appears, as has eloquently stated by Carney and Russell, serum vitamin A levels are best used as probability indicators rather than clear-cut designators of night blindness.

Diet surveys for vitamin A intake are subject to the same
constraints of individual variability as serum concentration measurements are as indicators of night blindness and so, as would be expected, were not found to correlate with final rod threshold. Diet surveys, however, suffer from the additional weakness of being rather poor predictors of serum vitamin A concentrations. Inherent inaccuracy, inability to assess available liver stores, and a myriad of variable absorption factors amongst individuals, are demonstrated by the lack of correlation of vitamin A intake and serum levels in "normal" ranges of intake.

These factors of individual variability in eating habits, absorption, and serum concentration required for physiological function result in inability of either dietary intake or serum concentration assessment to give conclusive evidence of vitamin A adequacy. Vitamin A supplementation with subsequent testing remains as the only adequate means of evaluating vitamin A sufficiency except in cases of gross deficiency.

Hair zinc levels were also not found to differ significantly between experimental and control groups, nor to correlate with serum vitamin A or with final rod threshold. It remains unclear whether these findings reflect an individual variability of zinc concentrations required for physiological function as was seen with vitamin A or shortcoming of the relatively new hair analysis techniques. Like vitamin A, the best method of evaluating potential of deficient zinc nutrition as the basis for night blindness in the individual
patient may be dietary supplementation.

CONCLUSION

When dark adaptometry test is either unavailable or impractical, a properly elicited history is a fairly sensitive and specific tool in the diagnosis of night blindness. The screening test discussed, the AAA Night Sight Meter and Thorntons reapid screening technique, while of potential value in advanced cases, are of limited value in milder cases of night blindness. When marginal nutritional deficiencies of zinc or vitamin A are suspected, dietary supplementation with subsequent monitoring is preferable to serum vitamin A determination, diet analysis for vitamin A intake, or hair zinc analysis.
REFERENCES


27. Vinton, N E: Evaluation of Rapid Test of Dark Adaptation, (Thesis, the Yale Universitv School of Medicine.)


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<tr>
<th>Test</th>
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E = experimental
C = control
NS = not significant

TABLE 1: Results of Statistical Analysis
Evaluation of the patient complaint as a screening test for night blindness. Diagnosis of night blindness based on dark adaptometry final rod threshold \( > 1 \text{ S.D. from control group mean} \).

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<tr>
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<tr>
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Sensitivity  = 82%
Specificity  = 79%

TABLE 2
Figure 1

Schematic diagram of vitamin A metabolism. Pathways dependent upon zinc metabolism are indicated with an asterisk.
Figure 2

Typical biphasic dark adaptation curve (solid line). Dashed line indicates changes seen with night blindness.
Figure 3

Scatter graph showing correlation of dark adaptometry final rod threshold and Night Sight Meter glare vision. (r=.423, p<.05)
Figure 4

Scatter graph showing correlation of dark adaptometry final rod threshold and Night Sight Meter night vision. (r = .503, p < .05).
Figure 5

Scatter graph showing correlation of Serum Vitamin A concentration and vitamin A dietary intake. (r=0.461, p<0.05)
Figure 6

Scatter graph showing correlation of serum vitamin A concentration and hair zinc. ($r = 0.356$, $p < 0.05$)
Envelopes containing all dark adaptometry curves for experimental and control groups. Significant difference was found only for final rod threshold.
Figure 8

Distribution of Night Sight Meter results for experimental and control groups. Arrows indicate group means, bars indicate ± standard deviation.
Figure 9

Distribution of serum vitamin A results. Arrows indicate group means, bars indicate ± 1 standard deviation.