Abstracts in the advertising sections

The New England Journal of Medicine

Established in 1812 as The NEW ENGLAND JOURNAL OF MEDICINE AND SURGERY

VOLUME 317 OCTOBER 22, 1987 NUMBER 17

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Duchenne's muscular dystrophy. We suspect that several of our patients have an autosomal disease with the same phenotype as Duchenne's muscular dystrophy but with a different genetic defect.[4]

Whatever the cause, recombination is an important source of error in the use of DNA probes for genetic counseling of families with X-linked dystrophy. As with any diagnostic test, it is important to have reliable data on the accuracy of these probes if they are to be used clinically. On the basis of our information, the error rate with even the best probes is at least 7 percent.

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1. Goodfellow PN, Davies KE, Ropers H-H. Report of the committee on the genetic constitution of the X and Y chromosomes. Cytogenet Cell Genet 1985; 40:296-352.
2. Fischbeck KH, Ritter AW, Tirschwell DL, et al. Recombination with pERT87 (DXS164) in families with X-linked muscular dystrophy. Lancet 1986; 2:104.
3. Ott J. Estimation of the recombination fraction in human pedigrees: efficient computation of the likelihood for human linkage studies. Am J Hum Genet 1974; 26:588-97.
4. Fischbeck KH, Ritter A, Kunkel LM, et al. Genetic heterogeneity in Duchenne dystrophy. Neurology 1987; 37:Suppl 1:116.

The above letters were referred to the authors of the article in question, who offer the following reply:

To the Editor: Although we agree with Dove and colleagues that the normal distribution of creatine kinase levels has to be carefully considered when serum creatine kinase determinations are used to assign carrier status, we disagree with their opening statement that "the use of DNA probes for prenatal diagnosis of carrier status . . . relied, in the absence of an affected male, on measurement of serum creatine kinase activity." This is not true of the families presented in our paper. In both families that fell into this category, Families A and B, the maternal grandmothers of the male fetuses at risk were obligate carriers. One had two affected sons, and the other had an affected brother and an affected son. The task at hand was to determine by DNA analysis whether the pregnant woman had passed on her maternally derived, possibly mutant, or her paternally derived, normal Xp21 region to her fetus. The creatine kinase results were irrelevant to the diagnosis in both cases, especially since values in the normal range do not exclude carrier status.

Fischbeck and Ritter provide additional data on the frequency of recombination between the phenotype for Duchenne's muscular dystrophy and polymorphisms detected by intragenic probes, which agree with our conclusions that recombination within the gene is an important source of error. Their 7 percent error rate, however, appears to be based on the use of a single intragenic marker. As we pointed out in our paper and documented in the families in our study, this type of error can be avoided by using flanking DNA markers outside the gene in addition to intragenic markers. If the entire Xp21 haplotype is transmitted without apparent recombination, the risk of error is much lower than 7 percent. On the other hand, if haplotype analysis reveals a crossover event, the study is uninformative if the site of the mutation with respect to the site of the crossover point is unknown.

The issues discussed here, however, are superseded by a recent breakthrough with the cloning of the entire gene for Duchenne's muscular dystrophy.* The 14-kb cDNA, when used as a molecular probe, detects deletions in at least 50 percent of all patients with Duchenne's or Becker's muscular dystrophy* (and Darras BT, et al.: unpublished data), in contrast to the detection rate of less than 10 percent with previously available random DNA probes. These deletions represent the molecular basis of the disease and are detectable in affected males and in carrier females. Thus, the cDNA method allows direct detection of the mutation in DNA, eliminating the need for linkage testing that is mostly applicable to large families and has inherent problems due to recombination. Most patients with Duchenne's muscular dystrophy are from families with a single proband, and in 50 percent of these, diagnosis of potential carriers and unborn fetuses can now be made accurately (Darras BT, et al.: unpublished data).

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SIMPLIFIED CALCULATION OF BODY-SURFACE AREA

To the Editor: Values for body-surface area are commonly used in the practice of internal medicine, particularly to calculate doses of chemotherapeutic agents and index cardiac output and stroke volume. Body-surface area (BSA) is generally calculated from height (Ht) and weight (Wt), according to equations such as the classic 1916 Du Bois formula[1]: $BSA\ (m^2) = 0.007184 \times Ht\ (cm)^{0.725} \times Wt\ (kg)^{0.425}$. Although many nomograms based on such equations have been published, some are inaccurate,[2] and one is not always readily available when a determination must be made. At our institution, we use a simple, easy-to-remember modification of an equation by Gehan and George,[3] which requires the use of a calculator with a square-root key:

$$BSA\ (m^2) = \sqrt{\frac{Ht\ (in) \times Wt\ (lb)}{3131}}$$

or, in metric:

$$BSA\ (m^2) = \sqrt{\frac{Ht\ (cm) \times Wt\ (kg)}{3600}}$$

If a 73-inch-tall, 175-lb patient is used as an example, the keystroke sequence on most calculators would be:

$$73 \times 175 = \ \div 3131 = \ \sqrt{\ } = 2.02\ m^2.$$

Although a slight degree of accuracy has been lost in making the above equations easy to remember, deviations from accepted values derived from other formulas[1,3,4] are generally less than 2 percent. We have made good clinical application of these equations and believe they may be of benefit to other physicians.

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2. Turcotte G. Erroneous nomograms for body-surface area. N Engl J Med 1979; 300:1339.
3. Gehan EA, George SL. Estimation of human body surface area from height and weight. Cancer Chemother Rep 1970; 54:225-35.
4. George SL, Gehan EA. Methods for measurement of body surface area. J Pediatr 1979; 94:342.

BOOK REVIEWS

NEUROANATOMY: AN ATLAS OF STRUCTURES, SECTIONS AND SYSTEMS

Second edition. By Duane E. Haines. 236 pp., illustrated. Baltimore, Urban and Schwarzenberg, 1987. $22.50.

The second edition of this atlas has been considerably improved over the first, and new material has been added. Included are more