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Group homes vs. institutions

For decades, deinstitutionalization advocacy groups have denounced large institutions as inherently inhumane, and have succeeded in getting many of them closed. Now three studies raise doubts about the purported benefits of closing large institutions.

Christopher Kearney, V. Mark Durand, and Jodi Mindell studied two groups of adults with severe or profound retardation. All originally were living in a large developmental center. Two-thirds of the individuals were relocated directly to small 15-bed facilities, while the other third were moved to another large developmental center while awaiting group home placements.

Kearney et al. found that the individuals moved to the larger facility actually were given more freedom of choice in their daily activities, and that this correlated with a higher level of adaptive behavior among these residents. Residents placed in the smaller homes, in contrast, had more restricted choices, and showed regression in adaptive behaviors.

Kearney, Durand and Mindell found that the individuals moved to the larger facility actually were given greater freedom of choice, and that this correlated with more adaptive behavior.

The researchers say their study indicates that increased choice leads to more adaptive behavior. It also suggests, they say, that "traditional methods of distinguishing residential facilities for persons with severe handicaps [e.g., number of occupants] are not fully appropriate.

"The movement of persons with severe handicaps to smaller community residences does not mean necessarily that they have been 'deinstitutionalized,'" they conclude, if such a move actually results in less freedom to make independent choices.

Comparing "campus," home options

In a related study, British researchers evaluated 50 mentally retarded adults moved from a large hospital to either a "campus" setting or a group home setting. The campuses had an average of 25 residents each, divided into several units (with about 12

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Steroid treatment effective in atypical case of Landau-Kleffner

Bulletin: As we go to press, ARRI has learned of important developments concerning Landau-Kleffner syndrome. Dr. Landau and his colleagues are expected to establish a number of LKS diagnostic and treatment centers in major cities. Update in next ARRI.

Some children diagnosed as having autism or pervasive developmental disorder (PDD) actually have been found to be victims of Landau-Kleffner syndrome, or LKS (see ARRI 5/1, 5/2, 9/2), Identifying such children is important because early treatment of LKS can often partially or even completely correct the language and behavior problems of children with the disorder.

A new report, by LKS researcher Gerry Stefanatos and colleagues, suggests that the number of children responsive to LKS treatment may be greater than thought. The researchers, whose work was featured on national TV earlier this year, have discovered that some autistic-like children without the abnormal EEG patterns typical of LKS, but with other LKS symptoms, may benefit greatly from the corticosteroid treatment commonly used for the disorder.

LKS generally strikes formerly normal children between the ages of one and eight. The children lose their receptive language skills—either gradually or suddenly—while retaining some expressive language. They often develop autistic symptoms including withdrawal, aggression, hyperactivity, resistance to change, and echolalia.

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Simple test developed for fragile X

European researchers have developed a simple blood test for fragile X syndrome, the leading cause of inherited mental retardation and one cause of autistic-like behaviors.

Rob Willemsen et al. say their new antibody test "requires only one or two drops of blood, and can be used for screening large groups of mentally retarded people and [newborns] for fragile X syndrome." Such large-scale screening might be of great value, the researchers say, since fragile X occurs in approximately 1 in 1200 males and 1 in 2500 females.

"In many countries, a small blood sample is taken to do a phenylketonuria [PKU] test," Willemsen and colleagues note. It would be simple, they say, to use a drop of blood from this same sample to test for fragile X.

While the blood test developed by Willemsen et al. detects full fragile X mutations, it does not detect the "premutations" which occur in asymptomatic transmitting males and unaffected carrier females. Thus, the researchers say, the PCR test—another method of testing for fragile X—"might be the choice for a population-based screening program for females carrying a premutation."

Fragile X is caused by a mutation of the FMR1 gene on the X chromosome. While the syndrome generally affects males, females sometimes exhibit symptoms as well. The symptoms of fragile X include retardation, hyperactivity, stereotyped behaviors, and physical abnormalities such as prominent ears, long faces, and poor muscle tone. While a minority of autistic children also have the fragile X genetic defect, it is not yet clear if the two disorders are linked.

New study recommends screening

Markku Ryynanen and colleagues are recommending widespread screening of individuals at risk of carrying the fragile X mutation or premutation. The researchers recently screened 515 "at-risk" individuals, and identified 66 with the full fragile X mutation and 163 with the premutation.

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"Rapid antibody test for fragile X syndrome," Rob Willemsen, Serieta Mohkamsing, Bert de Vries, Didier Devys, Ans van den Ouweland, Jean Louis Mandel, Hans Galjaard, and Ben Oostra; *The Lancet*, Vol. 345, No. 8958, May 6, 1995. Address: Ben A. Oostra, Department of Clinical Genetics, Erasmus University, Rotterdam, Netherlands.

—and—

"Carrier diagnosis of the fragile X syndrome—a challenge in antenatal clinics," Markku Ryynanen, Pertti Kirkinen, Arto Mannermaa, and Seppo Saarikoski; *American Journal of Obstetrics and Gynecology*, Vol. 172, No. 4, April 1995, pp. 1236-1239. Address not listed.