predictive of poor outcome.[3] Hypothermia can decrease the rise in NSE levels and may lead to reduced sensitivity for poor outcome, but the drop in NSE levels in patients who have had hypothermic treatment may also reflect increased neuroprotection.[4] It seems unlikely that NSE levels would rise after hypothermic treatment to produce a higher rate of false positives for poor outcome; elevated serum levels of NSE should still have prognostic value for poor outcome. The clearance rate of some drugs may be affected by hypothermia, but this effect would not significantly compromise the results of testing in patients paralyzed with cisatracurium or sedated with propofol (drugs commonly used during hypothermia), which are still cleared quickly, especially once patients are normothermic.

Prospective data are needed to examine the validity of the AAN practice parameters in a group of patients who receive hypothermic treatment after having cardiac arrest. It is highly likely that the factors that have been shown to be reliable predictors in the past — such as loss of pupillary and corneal reflexes and of somatosensory-evoked responses — will be validated. However, the timing of the testing of some variables may require adjustment.

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Older Age and a Reduced Likelihood of 2009 H1N1 Virus Infection

TO THE EDITOR: Early epidemiologic reports regarding the 2009 pandemic influenza A (H1N1) virus suggest that cases of infection and deaths are concentrated in adults between the ages of 20 and 40 years.[1] This finding could reflect age-related differences in susceptibility or differential testing and diagnosis in this age group. Increased susceptibility to infection in young persons is characteristic of influenza pandemics and has important implications for disease-control policy.[2] We examined whether the reported excess of cases in younger persons derives from testing practices or reflects a differential risk of infection in Ontario, Canada.

Our study sample included all persons who were tested for 2009 H1N1 virus infection under an enhanced, provincewide, laboratory-based surveillance regimen from April 20, 2009, to June 10, 2009. Patients with confirmed infection were compared with those who tested negative for the 2009 H1N1 virus. Using multivariate logistic regression and zero-inflated Poisson regression, we evaluated the association between age group (which was defined according to the relationship between birth year and the predominant circulating influenza strains) and the risk of infection with the 2009 H1N1 virus.

Of 11,560 patients who were tested, 1819 (15.7%) had positive results for the 2009 H1N1 virus. Persons who were born before 1957 had a reduced risk of infection, and estimates did not substantially change after adjustment for travel to Mexico, public health unit of residence, or calendar week (adjusted odds ratio for older age group, 0.15; 95% confidence interval [CI], 0.12 to 0.18; unadjusted odds ratio; 0.17; 95% CI, 0.14 to 0.21). Persons who were born between 1957 and 1975 were at intermediate risk for infection (adjusted odds ratio, 0.42; 95% CI, 0.37 to 0.48). Similar effects were seen in zero-inflated Poisson models that used testing volumes and population as model offsets. There was no significant relationship between age group and the risk of infection with seasonal influenza A viruses (either H3N2 or H1N1) (Fig. 1).

Among persons who were at risk for infection with 2009 H1N1 virus, being born before 1957 was associated with a lower infection risk. The reduced

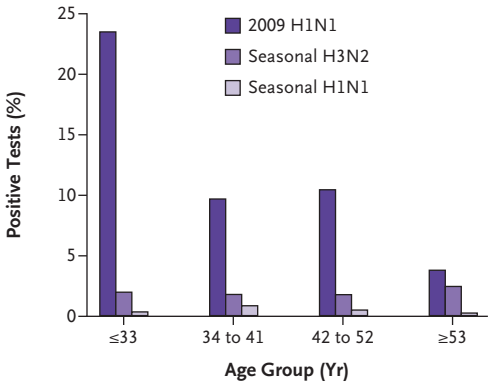

Figure 1. Age-Related Probability of Seasonal Influenza A and 2009 H1N1 Influenza in 11,560 Tested Patients.

Patients who were born after 1957 (i.e., ≤53 years of age) have an increased risk of infection with the 2009 pandemic influenza A (H1N1) virus. The results of testing show no significant relationship between age group and the risk of infection with seasonal influenza A viruses (either H3N2 or H1N1).

number of infections was not simply a reflection of decreased testing in this group. The mechanism for this association is unclear but is compatible with the reported age-related increase in the prevalence of neutralizing antibody titers against the 2009 H1N1 virus[3] and may reflect some immunity to infection as a result of exposure to similar viruses in early life. Maximally effective host immune responses to influenza may be generated by early-life infections.[4] These findings are consistent with the high frequency of outbreaks of 2009 H1N1 influenza in schools[5] and the decreased frequency of outbreaks in long-term care facilities that have been associated with this pandemic virus to date.

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Pathological Changes Associated with the 2009 H1N1 Virus

TO THE EDITOR: Between April 23, 2009, and May 15, 2009, we performed 15 autopsies on deceased patients in whom probable influenza had been diagnosed either clinically or macroscopically. Small samples of lung tissue were obtained and taken for analysis to the Institute of Epidemiological Diagnosis and Reference in Mexico City. Five infections with the 2009 pandemic influenza A (H1N1) virus were confirmed with the use of a real-time reverse-transcriptase–polymerase-chain-reaction assay, after it was determined that these patients were seronegative for influenza B virus, respiratory syncytial virus, parainfluenza virus (types 1, 2, and 3), and adenovirus.[1] From these five patients, organ samples were collected, fixed in 10% formalin, embedded in paraffin, and stained with hematoxylin and eosin. In the remaining 10 patients in whom the 2009 H1N1 virus was not detected, histopathological analyses identified bacterial pneumonia.

All five patients with diagnosed 2009 H1N1 influenza had been residents of Mexico City. Four of them were young adults (ages 22, 26, 28, and 37 years) who were hospitalized with the presump-