

Continuity in Clinical Education

TO THE EDITOR: I was glad to learn of the pilot project in longitudinally integrated clinical education for third-year medical students at Harvard Medical School and the Cambridge Health Alliance, as reported by Hirsh et al. (Feb. 22 issue).¹ It will be interesting to see whether outcome data from this and other efforts to reform undergraduate medical education by increasing continuity of care, curriculum, and supervision confirm the reasonable expectation of the authors that “enhanced learning, greater patient satisfaction, and more efficient and effective medical care” will result.

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1. Hirsh DA, Ogur B, Thibault GE, Cox M. “Continuity” as an organizing principle for clinical education reform. *N Engl J Med* 2007;356:858-66.

TO THE EDITOR: We studied the effect of an “e-learning” tutorial, on reading electrocardiograms (ECGs); the tutorial, held in October 2006, was for third-year medical students at Imperial Col-

lege London. One group of 83 students was told that the tutorial was compulsory, whereas a second group of 263 students was simply encouraged to use it. Participation rates were 97.5% in the first group and 64.6% in the second. A quiz about ECGs was administered 3 months later; the mean score in the first group was 75.1%, as compared with 60.1% in group 2.

E-learning encourages a deeper approach to learning,¹ and it can be used by students in distant clinical placements to supplement knowledge obtained on the wards. High-quality interactive e-learning will help lessen the discrepancy in knowledge due to different learning opportunities at different sites.²

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Chikungunya Outbreaks

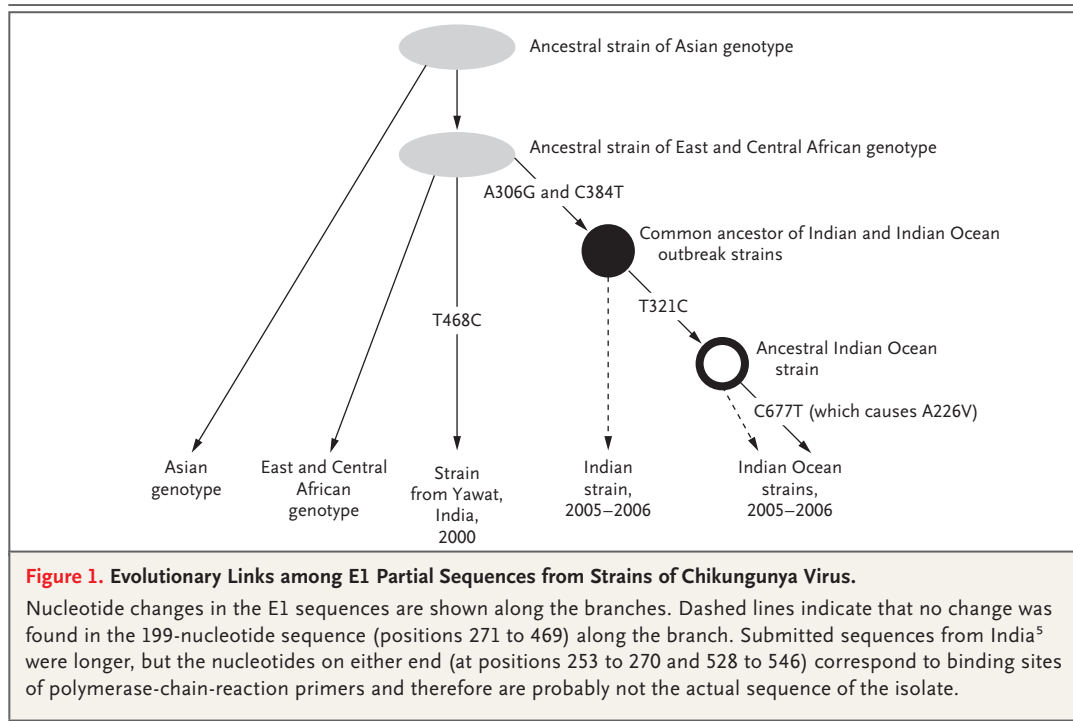
TO THE EDITOR: In their Perspective article on outbreaks of chikungunya fever, Charrel et al. (Feb. 22 issue)¹ suggest that the recent outbreak in India² could have been caused by the same viral strain that caused the Indian Ocean outbreak.³ Recent data do not support this speculation.^{4,5} We previously described sequence microheterogeneity in the viruses causing the Indian Ocean outbreak.⁴ We have compared E1 sequences from viruses involved in the Indian and Indian Ocean outbreaks (Fig. 1).^{4,5} The strains differed only in a single nucleotide change (T321C), which was found in all Indian Ocean isolates. Isolates from India retained the ancestral T321 nucleotide present in all other African and Asian strains, making it unlikely that the outbreak in India was caused by a strain originating from the Indian Ocean outbreak. Nevertheless, in contrast to se-

quences from all other available chikungunya viruses, the Indian and Indian Ocean isolates shared two nucleotide changes: A306G and C384T. These shared derived characters indicate common ancestry. The strain isolated from Yawat, India, in 2000⁵ retained the ancestral nucleotide at both positions but had a distinctive T468C change, arguing against a direct link between this strain and the strain implicated in the 2005–2006 Indian outbreak.

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TO THE EDITOR: I am concerned about the accuracy of the Perspective article by Charrel et al. regarding the distribution of *Aedes albopictus* in Australia and the number of deaths attributed to chikungunya in the recent outbreaks on Indian Ocean islands. The map that accompanies the article indicates that *A. albopictus* is widespread in Australia. This is incorrect. *A. albopictus* has not been found on mainland Australia.¹

In April 2005, *A. albopictus* was discovered in the Torres Strait of Australia.² However, an eradication program is currently restricting any further progression into the mainland. The map also fails to indicate that *A. albopictus* is present in much of western Indonesia, whereas it shows populations in most of the western United States.

I am also concerned about the statement that chikungunya was “implicated in 237 deaths.” The National Arbovirus and Malaria Advisory Committee believes that much of the data on human deaths attributed solely to chikungunya are very contentious and have not been properly evaluated.

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1. Gratz NG. Critical review of the vector status of *Aedes albopictus*. *Med Vet Entomol* 2004;18:215-27.
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THE AUTHORS REPLY: Brisse et al. contribute a new phylogenetic tree that includes strains involved in the chikungunya outbreaks in 2005 and 2006. Their data confirm that strains implicated in both the Indian Ocean and Indian outbreaks originated from a common ancestor that has not yet been identified. Moreover, they show that the current Indian outbreak involves a different strain from that found in Yawat, India, in 2000. This suggests that the huge outbreak of 2005–2006 followed the appearance of a new variant that subsequently evolved in India and the Indian Ocean.

Hall may be right to emphasize that the mosquito *A. albopictus* is not established in mainland Australia. However, it is established in the Torres Strait region in northern Australia¹ and has been repeatedly identified in Australian and New Zealand harbors for the past 20 years.^{2,3} Hall is also concerned about the reported number of deaths. The number of deaths per month on Reunion Island increased by 34% in February 2006 and 25% in March 2006, as compared with the number in the same month in 2005.⁴ These increases represent a total of 170 to 180 additional deaths, just for these 2 months, with many of them in patients older than 75 years. A total excess of 260 deaths was reported by the French National Institute for Public Health Surveillance⁴ for the outbreak. This corresponds roughly to a 1% case fatality rate for estimated cases of chikungunya (based on seroprevalence studies). Indeed, the disease outbreak paralleled a significant increase in mortality, and no other cause of death was identified during this

period. Few outbreaks have modified significantly the death ratio for a whole country. In the Reunion outbreak in 2006, chikungunya was a significant cause of death, perhaps because of the higher proportion of elderly patients infected during this outbreak than during previous outbreaks.

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Case 8-2007: A Man with Chest Pain Followed by Cardiac Arrest

TO THE EDITOR: In discussing the Case Record of a patient with *Helicobacter cinaedi* myopericarditis, presented by Lewis et al. (March 15 issue),¹ Dr. Butterton notes that the 16S ribosomal DNA sequencing used to identify this organism is not commercially available. Fortunately, that is not so: DNA-sequence-based identification of bacterial and fungal pathogens is integral to our clinical laboratory, and we routinely perform testing on referral specimens. For example, we recently identified *H. cinaedi* in a blood culture and *Bartonella henselae*, *Ureaplasma urealyticum*, *Tropheryma whipplei*, and *Coxiella burnetii* in aortic-valve biopsy specimens from patients with repeatedly negative cultures. The usefulness of DNA sequencing for identifying medically important bacteria,² yeasts,³ and molds⁴ is increasingly appreciated for several types of organisms: those lacking well-established phenotypic identifiers (as illustrated in the Case Record) or displaying phenotypic variability that defies conventional methods of identification; those that are slow-growing or fastidious or that cannot be cultured; and those present in fixed or paraffin-embedded tissue samples or in archival material

or samples of tissues for which resampling is undesirable or contraindicated (e.g., brain-biopsy specimens). Finally, molecular methods have proved cost-effective for routinely identifying elusive pathogens by substantially reducing the total cost of laboratory operations, turnaround time, and error rates.

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THE DISCUSSANT REPLIES: The patient described in the Case Record was admitted to the hospital in