

is evidence that measles may circulate in vaccinated populations and cause subclinical infection.²

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1. Parker AA, Staggs W, Dayan GH, et al. Implications of a 2005 measles outbreak in Indiana for sustained elimination of measles in the United States. *N Engl J Med* 2006;355:447-55. [Erratum, *N Engl J Med* 2006;355:1184.]

2. Vardas E, Kreis S. Isolation of measles virus from a naturally-immune, asymptotically re-infected individual. *J Clin Virol* 1999;13:173-9.

THE AUTHORS REPLY: We obtained laboratory confirmation for at least one patient in 9 of 11 families infected with measles. Among these 9 families, 14 of 20 patients had disease that was confirmed by laboratory analysis. The remaining two families (with 10 and 4 patients, respectively) declined to have specimens collected from all family members. The parents of the patient who had measles despite receiving two doses of vaccine declined to have specimens collected from any of their children except one whose disease was confirmed by laboratory testing during hospitalization. All patients had classic clinical symptoms of measles that appeared after the appropriate incubation period after exposure to a patient with laboratory-confirmed disease. The percentage of cases that

were confirmed by laboratory testing in the Indiana outbreak (41%) was similar to that in other outbreaks among groups of persons who had declined to receive vaccination.^{1,2}

Case finding involved contacting persons with a known exposure to measles, physician alerts, and media releases. Although the asymptomatic spread of measles could potentially occur, all but one patient had an identified source. This patient worked in a hospital where patients with measles had been treated within 14 days before the onset of her symptoms. Thus, we believe that asymptomatic transmission was unlikely to have played a major role in the Indiana outbreak.

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1. Hanratty B, Holt T, Duffell E, et al. UK measles outbreak in non-immune anthroposophic communities: the implications for the elimination of measles from Europe. *Epidemiol Infect* 2000; 125:377-83.

2. Siedler A, Tischer A, Mankertz A, Santibanez S. Two outbreaks of measles in Germany 2005. *Euro Surveill* 2006;11:131-4.

Pay-for-Performance Programs in the United Kingdom

TO THE EDITOR: As a general practitioner in England, I and the practice in which I work were directly affected by the changes made in 2004 by the introduction by the National Health Service of a pay-for-performance contract for family practitioners, as reported by Doran and colleagues (July 27 issue).¹ The contract was evidence based, ensuring that the majority of general practitioners approved of its aims.

Doran and colleagues omitted a number of important lessons that can be drawn from that experience. First, the necessity to “tick boxes” to ensure that tasks triggering payment were completed had a major effect on many consultations each day. Second, much bigger than the payments to general practitioners were the increased consequential costs triggered by the quadrupling of prescriptions for statins. Third, there was the effect on the local hospitals of a sudden increase

in referrals for investigative procedures such as echocardiography for heart failure and cardiologic referrals for angina — conditions that previously had often been dealt with without referrals. Fourth, there is the increased medication load for patients — typically, a patient with diabetes has to take 10 different therapies.

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1. Doran T, Fullwood C, Gravelle H, et al. Pay-for-performance programs in family practices in the United Kingdom. *N Engl J Med* 2006;355:375-84.

TO THE EDITOR: In his editorial accompanying the article by Doran and colleagues, Epstein¹ encourages the United States to adopt a system similar to that introduced in the United Kingdom.

Although financial incentives could change doctors' behavior,² it is difficult to ascertain whether the achievement reported by Doran et al. is due solely to incentives or to an improvement in clinical practice in general, since there is no control group and there are no baseline data. In our area, organizational care indicators for diabetes, such as data recording, have increased dramatically, but clinical indicators, such as cholesterol levels and glycated hemoglobin values, have revealed a smooth increase that might be due to other factors, such as the use of national targets and the active dissemination of guidelines. Pay for performance could result in a loss of the holistic approach to patient care,³ and patients with diseases that are not included in the contract could be put at a disadvantage.³ Incentives may need to increase with time to maintain targets. Pay for performance may be a good idea, but it should be implemented with caution. We would recommend that when this approach is introduced into a new area it be started as a pilot, so that some comparisons with conventional care as a control can be made.

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1. Epstein AM. Paying for performance in the United States and abroad. *N Engl J Med* 2006;355:406-8.
2. Chaix-Couturier C, Durand-Zaleski I, Jolly D, Durieux P. Ef-

fects of financial incentives on medical practice: results from a systematic review of the literature and methodological issues. *Int J Qual Health Care* 2000;12:133-42.

3. Roland M. Linking physicians' pay to the quality of care — a major experiment in the United Kingdom. *N Engl J Med* 2004;351:1448-54.

THE EDITORIALIST REPLIES: I agree in general with Tahrani et al. As noted in my editorial, the findings reported by Doran et al. could reflect a number of different factors other than improved performance as prompted by the payment incentives. And surely there are a number of reasons to have modest expectations for the improvement in quality associated with pay-for-performance programs and to be wary of the potentially deleterious side effects they may inspire. There have been relatively few studies of pay for performance in health care.^{1,2} On the whole, their findings are not encouraging, although most of the programs studied may not be comparable to the large efforts now envisioned. Numerous pay-for-performance programs are under way in the private sector, and although few have been formally analyzed, anecdotal information has not pointed to large negative consequences. Many aspects of pay for performance make intuitive sense. Thus, it seems to me to be reasonable to bolster efforts in this direction, so long as we maintain moderate expectations and monitor the programs carefully, with an eye to making appropriate modifications.

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1. Rosenthal MB, Frank RG, Li Z, Epstein AM. Early experience with pay-for-performance: from concept to practice. *JAMA* 2005;294:1788-93.
2. Rosenthal MB, Frank RG. What is the empirical basis for paying for quality in health care? *Med Care Res Rev* 2006;63:135-57.

Causes of Chronic Diarrhea

TO THE EDITOR: In the table about congenital diarrheal disorders in the Perspective by Binder (July 20 issue),¹ congenital sodium diarrhea is attributed to mutations in the gene encoding the sodium-hydrogen exchanger (NHE) isoform 3 (SLC9A3, also known as NHE3). Although NHE3-knockout mice (those deficient in *Slc9a3*) are the only available animal model of congenital diarrhea,² genetic analyses of patients with congenital sodium diarrhea have excluded all mapped NHE loci except

NHE4 (which houses *SLC9A4*), but even this locus has not been firmly implicated.^{3,4}

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1. Binder HJ. Causes of chronic diarrhea. *N Engl J Med* 2006;355:236-9.
2. Schultheis PJ, Clarke LL, Meneton P, et al. Renal and intestinal absorptive defects in mice lacking the NHE3 Na⁺/H⁺ exchanger. *Nat Genet* 1998;19:282-5.