Correspondence

Single-Chamber versus Dual-Chamber Pacemakers

To the Editor: In their report on the Pacemaker Selection in the Elderly (PASE) trial, Lamas et al. (April 16 issue)[1] compare single-chamber pacing (VVIR mode [ventricular pacing, ventricular sensing, inhibition response, rate-adaptive]) and dual-chamber pacing (DDDR mode [atrial and ventricular pacing, atrial and ventricular sensing, dual response, rate-adaptive]) in the elderly and conclude that there is no specific quality-of-life benefit for patients undergoing dual-chamber pacing as compared with those undergoing single-chamber pacing (a conclusion that the accompanying editorial notes will surprise most experts[2]). This conclusion is reached despite the fact that 26 percent of the patients assigned to the VVIR mode crossed over to the DDDR mode after symptoms of the pacemaker syndrome developed. The conclusion is therefore suspect, and it has implications for the selection and implantation of pacemakers that are not considered in the article.

In the PASE trial, dual-chamber pacemakers were implanted in all the study participants. The pacemakers were programmed to the VVIR or DDDR mode before implantation, and the patients were randomly assigned to single- or dual-chamber pacing. Those assigned to the VVIR mode in whom the pacemaker syndrome developed were therefore treated simply by reprogramming the pacemaker to the DDDR mode. In clinical practice, however, the development of the pacemaker syndrome in a patient with VVIR pacing requires upgrading the pacemaker, with implantation of an atrial lead and a new generator.

Experience with this procedure has not been extensively documented, but we have recently reported a retrospective assessment of patients who underwent pacemaker upgrading in our high-volume regional pacing center (where we perform 350 implantations annually).[3] Forty-four patients underwent upgrading in an eight-year period. The mean duration of the procedure significantly exceeded that for implantation of a pacemaker in the VVI or DDD mode. More important, the complication rate was high. Twenty patients (45 percent) had complications, such as pneumothorax, infection, a prolonged procedure, or the need for repositioning of the atrial lead.

Lamas et al. suggest that "many patients who received dual-chamber pacemakers might fare just as well with ventricular systems." This is no doubt true, but we cannot reliably identify these patients. If elderly patients with ongoing atrial activity are given single-chamber ventricular pacemakers, the data reported by Lamas et al. suggest that one quarter of them will need to have their pacemakers upgraded. In view of the morbidity and complication rate that we have documented, the costs of upgrading pacemakers in such a large number of patients would, we suggest, significantly outweigh any potential savings from implanting simpler pacing systems. We believe that patients with atrial activity should not be offered single-chamber ventricular pacemakers in the mistaken belief that the system can be upgraded if necessary at minimal risk.

DAVID J.R. HILDICK-SMITH, M.R.C.P.
JOHN T. WALSH, M.D.
Papworth Hospital
Cambridge CB3 8RE, United Kingdom

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To the Editor: Only a few days after the publication of the article by Lamas et al. and the accompanying editorial

by Mason and Hlatky, newspapers carried headlines such as "Type of Pacemaker Appears Not to Matter"[1] and "Pricey Pacemakers No Better: Study."[2] Although the accompanying editorial emphasized some of the important issues, it called attention to the cost of pacing in such a way as to reinforce the media's claim that dual-chamber pacemakers are overused and too expensive. This was unfortunate, and we take exception to these arguments.

Dual-chamber pacing may have been more expensive than single-chamber pacing 12 years ago, as cited by Mason and Hlatky. But advances in technology and competitive changes in the marketplace bring into question the relevancy of data from 1986. According to the last Bilitch Report,[3] on 20,128 pulse generators powered by lithium cells, there was no significant difference in longevity between dual- and single-chamber devices (70.2 and 73.8 percent of the devices, respectively, were still functional at 10 years). A detailed analysis of the real long-term costs of pacing has shown, surprisingly, that single-chamber pacemakers are actually more costly than dual-chamber devices.[4]

Several retrospective and prospective studies have suggested that subsequent atrial fibrillation and congestive heart failure are much more common with prolonged single-chamber pacing than with dual-chamber pacing. There is also some evidence that patients with dual-chamber pacemakers live longer.[4] These data refute the points about differences in cost between single- and dual-chamber pacing.

To have a full understanding of the advantage of dual-chamber pacemakers, one must also consider that with the passage of time, some patients will require various drugs that produce inappropriate bradycardia or atrioventricular block. It would be advisable to customize the device by programming it appropriately to accommodate these changes in clinical conditions. This can best be done with dual-chamber pacemakers.

As clinicians, we frequently see patients who do not tolerate VVI pacing and whose symptoms improve (or disappear) with dual-chamber pacing. It will be interesting to follow the series of patients described by Lamas et al. for a longer time, with particular attention to the late deleterious results of pacing in either mode. Until there is more convincing evidence supporting VVIR over DDDR pacing, we will continue to implant dual-chamber pacemakers in patients with underlying sinus rhythm.

VICTOR PARSONNET, M.D.
MARC ROELKE, M.D.
New Jersey Pacemaker and Defibrillator Evaluation Center
Newark, NJ 07112

1. Type of pacemaker appears not to matter. USA Today. April 16, 1998.
2. Pricey pacemakers no better: study. Daily News. April 16, 1998.
3. Song SL. Performance of implantable rhythm management devices. Pacing Clin Electrophysiol 1994;17:692-708.
4. Brown Mahoney C, Sharma A, O'Neill PG. Costs of follow-up cardiovascular care in pacemaker patients — correction for mortality. Pacing Clin Electrophysiol 1996;19:622. abstract.

To the Editor: We have reservations about the use of the 36-item Medical Outcomes Study Short-Form General Health Survey (SF-36) in the age group studied by Lamas et al. Its use as a generic quality-of-life measure is well established in culturally and demographically diverse populations, but its usefulness in elderly persons, particularly those with serious disorders, has been questioned.[1] The assessment of quality of life in elderly patients may be improved by the use of instruments that allow patients to select and rate the areas of life that are important to them (e.g., the Schedule for Evaluation of Individual Quality of Life).[2] In addition, cognitive function, which is important both because it is a determinant of quality of life and because of its potential confounding effects on the reliability and validity of the SF-36, was not assessed.

GUY M. GRIBBIN, B.M., B.CH.
STEVE W. PARRY, M.B., B.S.
Freeman Hospital
Newcastle upon Tyne NE7 7DN, United Kingdom

1. Hill S, Harries U, Popay J. Is the short form 36 (SF-36) suitable for routine health outcomes assessment in health care for older people? Evidence from preliminary work in community based health services in England. J Epidemiol Community Health 1996;50:94-8.
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The authors reply:

To the Editor: Drs. Hildick-Smith and Walsh point out that if the pacemaker syndrome develops in 26 percent of patients receiving ventricular pacemakers and an upgrade to dual-chamber pacing is required, then ventricular pacemakers should never be used in patients with sinus rhythm. However, the crossover rate in the PASE trial[1] probably overestimates the incidence of the pacemaker syndrome. In the PASE trial, a crossover from single-chamber ventricular pacing to dual-chamber pacing required only reprogramming, not surgery. The one other published randomized trial of pacemakers[2] found that only 1.7 percent of patients with ventricular pacing had severe pacemaker syndrome. The true incidence of severe pacemaker syndrome is probably considerably lower than the crossover rate in the PASE trial.

Drs. Gribbin and Parry discuss possible problems in accurately measuring quality of life in elderly patients. We used an interviewer-administered SF-36 questionnaire to avoid the problem of a low completion rate among elderly patients. Although the Schedule for Evaluation of Individual Quality of Life is conceptually interesting, there is no compelling evidence that it would provide a better assessment of quality of life than the SF-36 among elderly patients.[3] Indeed, the importance ratings that respondents must provide on the Schedule for Evaluation of Individual Quality of Life are cognitively more demanding than the simple rating scales used in the SF-36. All patients in the PASE trial had to have sufficient cognitive function to provide informed consent, complete the base-line interview, and pass a simple cognitive screening with the Telephone Interview for Cognitive Status. Furthermore, we expect that randomization balanced the confounding influence of mild, undetected cognitive impairment in the two treatment groups.

Drs. Parsonnet and Roelke comment on the cost and longevity of pacemakers and on the clinical outcomes. Dual-

chamber pacemakers require two leads instead of one, and the prices for dual-chamber systems are higher than those for ventricular pacemakers of similar sophistication. The battery drain is greater with dual-chamber pacemakers than with ventricular pacemakers. However, individual variations in pacemaker programming have made these differences more difficult to demonstrate.

The comments on clinical outcomes and cost effectiveness are based on retrospective data, which are confounded by profound biases with regard to the selection of the pacemaker mode. Patients with dual-chamber pacing are younger and healthier, are more likely to be men, and have higher socioeconomic status than patients with ventricular pacing.[4] Therefore, it is not surprising that retrospective studies report that patients with dual-chamber pacing have a better prognosis. The results of prospective studies, such as the study by Andersen et al.,[2] the PASE trial,[1] and the large ongoing trials (the Canadian Trial of Physiologic Pacing, Mode Selection Trial, and United Kingdom Pacing and Clinical Events Trial), will establish the clinical benefits and cost effectiveness of atrial-based pacing.

GERVASIO A. LAMAS, M.D.
Mount Sinai Medical Center
Miami Beach, FL 33140

LEE GOLDMAN, M.D.
University of California, San Francisco
San Francisco, CA 94143

CAROL MANGIONE, M.D.
University of California, Los Angeles
Los Angeles, CA 90024

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Intravenous Immune Globulin and Pseudohyponatremia

To the Editor: A 49-year-old woman admitted with typical Guillain–Barré syndrome was treated with intravenous immune globulin (Gamimune N 10%, Bayer, Elkhart, Ind.), at a dose of 45 g per day (0.4 g per kilogram of body weight) for five days.

The sodium concentration, measured with the use of an ion-selective electrode in diluted serum specimens ("indirect potentiometry"), decreased from 143 mmol per liter on day 1 to 127 mmol per liter on day 4. On day 5, when the serum sodium concentration was 122 mmol per liter, urine osmolality was 474 mOsm per liter, the urinary sodium concentration was 45 mmol per liter, and the serum protein value was elevated (10 g per deciliter), but serum glucose and lipid concentrations and thyroid function were normal. Plasma exchange was started on day 6 because of progression and complete flaccid quadriplegia; the serum sodium and protein concentrations promptly returned to normal.

Hyponatremia occurs in up to 26 percent of patients with the Guillain–Barré syndrome, usually because of inappropriate antidiuretic hormone secretion.[1] Pseudohyponatremia is a laboratory artifact due to hyperlipidemia or hyperproteinemia.[2] Intravenous infusion of immune globulin increases the protein-containing nonaqueous phase of plasma, with a consequent relative decrease in plasma water volume. Since sodium is present only in the aqueous phase, each unit volume of plasma measured has less sodium-containing water, and this is interpreted as hyponatremia. The automated method of measurement used by most laboratories involves the use of an ion-selective electrode with prediluted serum or plasma specimens. This method measures the sodium concentration per liter of serum but does not correct for elevated protein or lipid concentrations. Ion-selective–electrode systems are available that analyze whole-blood specimens with no dilution required. These systems are not affected by the concentrations of protein or lipids in solution.

Pseudohyponatremia has been reported after the administration of intravenous immune globulin in patients with other neurologic diseases, but it has not been as pronounced as in this case.[3] True hyponatremia with normal or hypertonic plasma may be seen in association with hyperglycemia as a result of the movement of water from cells into the extracellular fluid space.

The recognition that hyponatremia during intravenous infusion of immune globulin may be an artifact may prevent changes in management that could have deleterious effects. Unnecessary fluid restriction may lead to dehydration, which may impair renal function or exacerbate the effects of dysautonomia.

NICHOLAS LAWN, M.D.
EELCO F.M. WIJDICKS, M.D.
MARY F. BURRITT, PH.D.
Mayo Clinic
Rochester, MN 55905

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Expression of Renin in Glomerular Nonjuxtaglomerular Cells in a Child with a Hypertensive Bartter's-Like Syndrome

To the Editor: Bartter's-like syndromes are characterized by hypokalemia, hypochloremia, metabolic alkalosis, and normal blood pressure despite hyperreninemia and hyperaldosteronism.[1] We report on an infant with a Bartter's-like syndrome characterized by hypertension, hypercalcemia, and the ectopic expression of renin in glomerular nonjuxtaglomerular and adrenal vascular smooth-muscle cells.

The patient was a girl who was delivered at 30 weeks' gestation by cesarean section because of fetal tachycardia. The infant's maternal great-grandmother and maternal great-aunt had hypertension. She weighed 1620 g. The neonatal course was characterized by hyponatremia, hypochloremia, hypophosphatemia, metabolic acidosis, hypercalcemia, hypomagnesemia, and anemia. The blood urea nitrogen and serum creatinine concentrations were normal. The serum parathyroid hormone concentration was increased, but the concentrations of serum vitamin D metabolites were normal. Urinalysis revealed glycosuria, aminoaciduria, and increased urinary electrolyte, calcium, phosphorus, and bicarbonate excretion. The serum aldosterone concentration and plasma renin activity were elevated. Renal ultrasonography revealed nephrocalcinosis. Hypertension (blood pressure, approximately 125/95 mm Hg) was noted during the first week of life and treated with captopril (0.3 mg per kilogram of body weight per day). Failure to thrive developed, and the infant required nasogastric feeding and large supplements of sodium, potassium, and phosphate. At five months of age, she died after the development of aspiration pneumonia and sepsis. Relevant autopsy findings included parathyroid hyperplasia, biventricular hypertrophy of the heart, nodular hyperplasia of the adrenal cortex, nephrocalcinosis, hyperplasia of the juxtaglomerular apparatus, and a rickets-like lesion in the ribs.

Immunohistochemical studies of the distribution of renin were performed with a renin antibody in 4-μm paraffin-embedded renal-tissue sections, as described previously.[2,3] In situ hybridization studies were performed with digoxigenin-labeled renin RNA probes (a gift from Dr. K. Murakami, University of Tsukuba, Tsukuba, Japan),[4] with the use of a kit from Boehringer Mannheim Biochemica. In contrast to the findings in control sections of renal tissue from 14 children with conditions such as sepsis, congenital heart disease, renal-artery stenosis, adrenal insufficiency, the hemolytic–uremic syndrome, and human immunodeficiency virus–associated nephropathy, renin protein and messenger RNA were detected in the child's glomerular mesangial and endothelial cells (Fig. 1) and in vascular

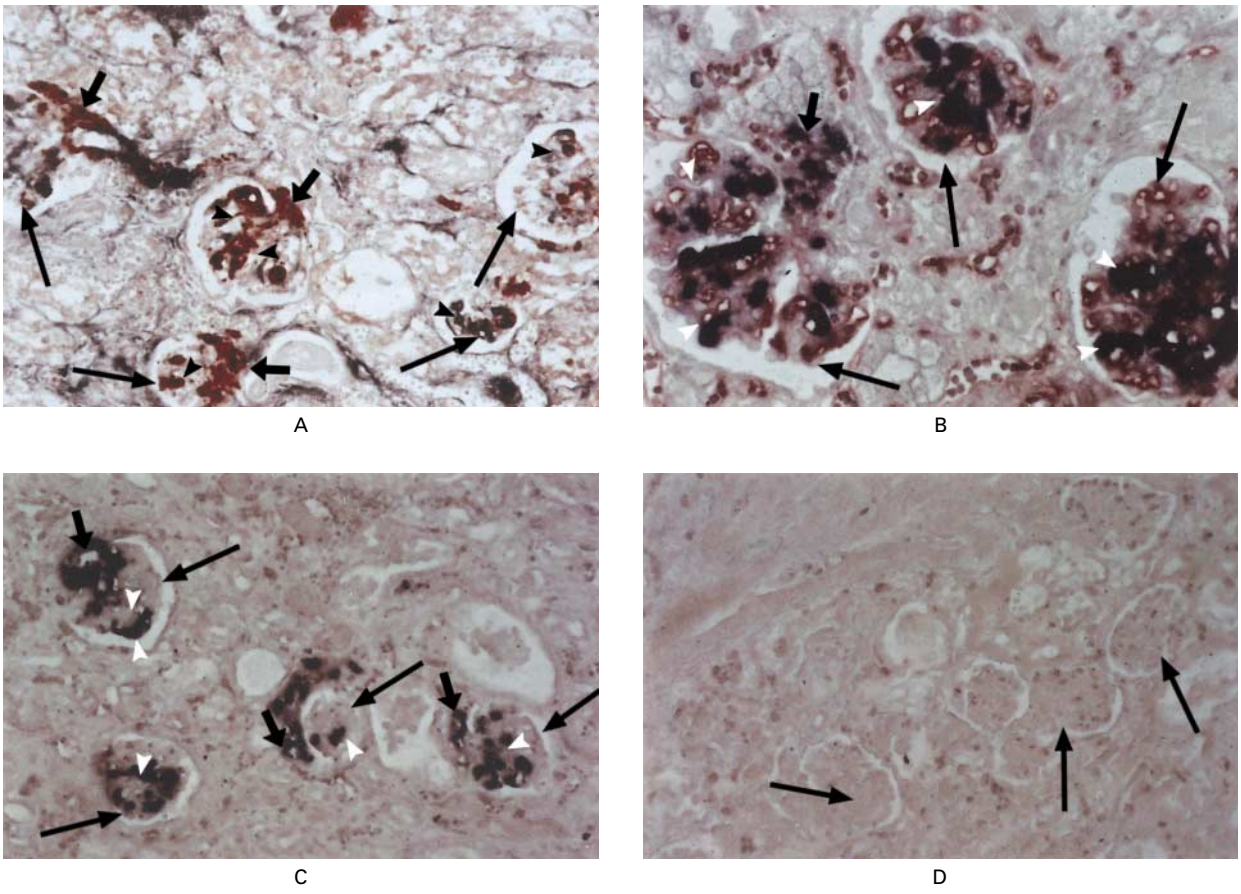

Figure 1. Detection of Renin in Glomerular Nonjuxtaglomerular Cells in an Infant with a Hypertensive Bartter's-Like Syndrome.

Panel A shows colocalization of renin protein (dark red) and smooth-muscle α-actin (stained dark blue [appears black here] with a monoclonal anti–smooth-muscle α-actin antibody from Sigma) in glomeruli (long arrows), juxtaglomerular cells (short arrows), nonjuxtaglomerular cells in glomeruli (arrowheads), and renal vessels (×200). Panel B shows colocalization of renin messenger RNA (black) in glomeruli (stained dark red with an anti-CD34 antibody from Vector Laboratories, arrowheads) (×500). Panels C and D show renal tissue hybridized with the human renin antisense and sense RNA probes. Renin messenger RNA (black) is detectable within glomerular juxtaglomerular and nonjuxtaglomerular cells with the renin antisense probe (Panel C, ×200). Only background staining is detectable with the renin sense probe (Panel D, ×200).

smooth-muscle cells in the adrenal glands. This aberrant expression of renin has not been reported in other patients with Bartter's-like syndromes or renal disease.[2,3,5] The ectopic production of renin and hypercalcemia may explain the presence of hypertension, cardiac hypertrophy, nephrocalcinosis, and failure to thrive in this infant. These findings suggest that the ectopic expression of renin in nonjuxtaglomerular cells may be involved in the pathogenesis of other neonatal forms of Bartter's-like syndromes associated with hypertension.

PATRICIO E. RAY, M.D.
XUE-HUI LIU, M.D., PH.D.
Children's National Medical Center
Washington, DC 20010

TADASHI INAGAMI, PH.D.
Vanderbilt University Medical Center
Nashville, TN 37232

ROMA S. CHANDRA, M.D.
Children's National Medical Center
Washington, DC 20010

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More on Dangerous Dilution of 25 Percent Albumin

To the Editor: The letter by Steinmuller (April 23 issue)[1] reports the problems, including hemolysis, that occurred when sterile water was used to dilute 25 percent albumin. The problems occur because although the albumin is concentrated about fivefold in 25 percent albumin, the electrolytes are not, so that the resulting solution is close to isotonic and therefore can be safely administered intravenously. When diluted with water or dextrose in water, it becomes proportionally low in electrolytes.

Although the response by the representatives of the Food and Drug Administration (FDA)[2] is correct with regard to the recommendation that dilution with 5 percent glucose, instead of water alone, would prevent hemolysis, use of this solution would not prevent the possible development of hyponatremia and brain swelling if the solution were used rapidly in large volumes, as in plasma exchange. Owing to its oncotic pressure, the albumin would tend to remain in the plasma compartment, whereas the glucose would rapidly leave the circulation, enter the interstitial fluid and then enter cells, become metabolized, and no longer exert an osmotic effect. This would not be a problem if the solution were given in small enough amounts or slowly enough to allow the kidneys to excrete the excess water before it accumulated, but in the case of plasmapheresis, it would certainly be hazardous, since the extra water would dilute the extracellular electrolytes and then shift to the intracellular compartment.

Glucose solutions should not be used to replace plasma or other extracellular fluids. Sodium chloride 0.9 percent (154 mmol per liter) is a reasonable alternative, but a more physiologic solution, one that more closely resembles plasma, would be even better.

RICHARD E. KRAVATH, M.D.
Kings County Hospital Center
Brooklyn, NY 11203-2098

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To the Editor: The letter by Dr. Steinmuller is misleading in that it takes out of context advice from the ninth edition of the *Handbook on Injectable Drugs*[1] concerning the dilution of 25 percent albumin. That edition states, "A 5% solution may be prepared from the 25% product by adding 1 volume of the 25% albumin to 4 volumes of an infusion solution such as dextrose 5% in water or sodium chloride 0.9%. If sterile water for injection is the diluent, the tonicity of the diluted solution must be considered. Substantial reduction in tonicity creates the potential for hemolysis."

It is common knowledge that large volumes of very hypotonic solutions should not be administered intravenously. Although the FDA recommended in its *Federal Register* notice[2] that sterile water for injection be used as a diluent for albumin when a lower sodium concentration is indicated, this is undoubtedly suitable only for small volumes of albumin, for partial dilutions, or in the presence of other diluents. There are no pharmaceutical products that should be given in large volumes as very hypotonic solutions. This principle was apparently disregarded in the cases described by Pierce and colleagues.[3]

In 1995, Forte et al. reported hemolysis in a patient undergoing plasmapheresis.[4] The information on albumin in the *Handbook on Injectable Drugs* was revised as noted in the edition published in 1996. The January 14, 1998, issue of *ISMP Medication Safety Alert,*[5] published by the Institute for Safe Medication Practices and sent to virtually every hospital pharmacy in the United States, called attention to the dangers of hypotonic albumin solutions.

This situation points to the need for health care practitioners to use up-to-date references. We are continuing our efforts to alert practitioners through collaboration with the Institute for Safe Medication Practices and notices in upcoming issues of the *ASHP Newsletter* and the *American Journal of Health-System Pharmacy* as well as on the Web site of the American Society of Hospital Pharmacists (http://www.ashp.org). The 10th edition of the *Handbook on Injectable Drugs,* released in June 1998, contains a stronger notice about the dilution of albumin.

The *Handbook on Injectable Drugs* focuses on the stability and compatibility of drugs. Each specific clinical situation

requires practitioners to use their training, knowledge, and common sense when interpreting and applying information from any reference.

LAWRENCE A. TRISSEL, F.A.S.H.P.
University of Texas M.D. Anderson Cancer Center
Houston, TX 77030

HENRI R. MANASSE, JR., PH.D., SC.D.
American Society of Health-System Pharmacists
Bethesda, MD 20814

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The authors and a colleague reply:

To the Editor: We agree with Dr. Kravath that the use of a replacement solution of albumin (human) 5 percent, prepared by diluting albumin (human) 25 percent with dextrose 5 percent injection (in water), would generally be inappropriate in the context of plasma exchange or plasmapheresis or in other situations involving the administration of very large volumes, because of the attendant risk of hyponatremia. Physicians must always take the patient's fluid and electrolyte balance into consideration in deciding what intravenous replacement solutions to administer. Our statement in responding to Dr. Steinmuller that acceptable diluents for albumin (human) 25 percent include sodium chloride 0.9 percent injection and dextrose 5 percent injection was intended to address avoidance of the risk of hemolysis. Physicians may use normal saline as a diluent for albumin (human) 25 percent in situations in which the fluid and electrolyte status of the patient permits the administration of normal saline. Conversely, in situations in which dextrose 5 percent injection may be safely administered in like volume, the dilution of albumin (human) 25 percent with dextrose 5 percent injection may be appropriate.

Plasma exchange or plasmapheresis represents a unique circumstance because, in formulating the replacement solution, one must take into account not only the loss of endogenous plasma proteins (principally, but not exclusively, albumin) but also the fact that significant quantities of electrolytes such as sodium and chloride are being removed by the procedure. As was evident in our reply to Dr. Steinmuller's letter, the sodium concentration of albumin (human) 5 percent prepared by dilution with dextrose 5 percent injection is only 26 to 32 mmol per liter. We are aware that normal saline is commonly used as a diluent for albumin (human) 25 percent in preparing replacement solutions for plasma exchange or plasmapheresis. In other settings in which sodium load is a concern and the volume to be administered is more limited, dextrose 5 percent injection may be an acceptable choice of diluent for albumin (human) 20 or 25 percent solutions.

We wish to emphasize the final statement by Trissel and Manasse. It is unlikely that any reference book, compendium, or guideline can anticipate all clinical situations. Accordingly, practitioners must continue to use their training, knowledge, and experience when interpreting and applying information from such sources.

L. ROSS PIERCE, M.D.
JOHN S. FINLAYSON, PH.D.
JAY S. EPSTEIN, M.D.
Food and Drug Administration
Rockville, MD 20852

Treatment of Port-Wine Stains

To the Editor: Van der Horst et al. (April 9 issue)[1] report no additional benefit from early treatment of port-wine stains with the pulsed-dye laser, which is in contradistinction to our experience[2] and that of others. Several issues should be raised about the methods of the study, which may account for the unexpected results.

The authors enrolled consecutive patients in their treatment groups without controlling for the types or locations of the lesions. There were more "hypertrophic" lesions in the younger treatment groups (29 percent) than in the older treatment groups (18 percent). Hypertrophy of port-wine stains in childhood has not been reported previously, suggesting that lesions with an arterial or venous component may have been treated. Such lesions are known to be poorly responsive to pulsed-dye laser treatment. Van der Horst et al. also failed to control for the location of lesions on the head and neck, which we have reported[3] to affect the response to pulsed-dye laser treatment.

The use of ice cubes to cool lesions during treatment may have led to chilling of dermal vessels in younger patients, who have thinner skin, thereby interfering with the efficacy of laser treatment. By treating port-wine stains only partially at each visit and by requiring several sessions to treat the entire port-wine stain, van der Horst et al. provided few complete treatments early on, potentially missing a therapeutic window of opportunity, when the skin is thinner and the stain smaller.

The treatment technique and technology that were used are outdated. Improved therapeutic outcomes[4] have been demonstrated with the use of larger spot sizes — 7 to 10 mm — rather than the 5-mm spot size used by van der Horst et al. Additional benefit has been derived from the use of longer wavelengths (595 nm, as compared with 585 nm) and longer pulse durations (1.5 msec, as compared with 0.45 msec), in particular for hypertrophic lesions. Selective epidermal cooling can be achieved with cryogen-spray cooling, which is now being utilized in conjunction with 585-nm and 595-nm pulsed-dye laser treatment, decreasing the pain of treatment and the time needed for recovery.[5]

ARIELLE N.B. KAUVAR, M.D.
ROY G. GERONEMUS, M.D.
317 E. 34th St.
New York, NY 10016

1. van der Horst CMAM, Kos ter PHL, de Borgie CAJM, Bossuyt PMM,

van Gemert MJC. Effect of the timing of treatment of port-wine stains with the flash-lamp–pumped pulsed-dye laser. N Engl J Med 1998;338:1028-33.
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The authors reply:

To the Editor: At the start of our study it was clear from other series involving small numbers of patients that port-wine stains in children could be treated safely with the flash-lamp–pumped pulsed-dye laser.[1] Our prospective study, using objective color measurements and validated instruments to measure disfigurement, tested the hypothesis that port-wine stains in children could be treated more effectively than those in adults.[2,3]

We included patients with capillary malformations alone. We defined hypertrophic port-wine stains in this population as lesions that were abnormally swollen in comparison with the healthy skin on the contralateral side. Patients with combined malformations (venous, arteriovenous, and lymphatic) were excluded. In our opinion, patients with Sturge–Weber syndrome have capillary malformations, and there is no evidence that treatment of port-wine stains that are part of such a syndrome is less effective than treatment of port-wine stains that are not part of a syndrome.

Before beginning treatment, we recorded the extent and location of the port-wine stain in each patient. We made an anatomical diagram of the face and neck in 64 regions and then regrouped these regions into 18 principal regions. We evaluated the response to treatment of the port-wine stain in relation to these regions within the age groups. We did not observe differences in responses that were related to the anatomical locations of the port-wine stains. In determining the response to treatment, we found that the initial size and depth of the lesion were more important predictive factors than the location of the lesion.

We only used gauze drenched in ice water, not ice cubes, for a short period after the laser pulses. The question is whether the vascular response to this type of pain relief differs from the vasoconstriction induced by a eutectic mixture of lidocaine and prilocaine (Emla cream), which is not known to interfere with the efficacy of treatment.[4]

Therapy was standardized; therefore, we did not change the laser settings during the study period.[5] The use of a faster laser with a larger spot size (7 mm) would not have influenced the use of general anesthesia in our youngest age groups. In children with port-wine stains that could be treated partially during one visit, the entire lesion was treated within six to eight weeks. We included only a few children under the age of one year; therefore, it is still unclear whether such patients will ultimately have better treatment results than those treated at an older age. The additional benefit of the use of longer wavelengths and pulse durations remains to be established. Nonetheless, our main conclusion — that treatment of port-wine stains in early childhood is effective, but not more so than treatment at a later age — is unlikely to change.

CHANTAL M.A.M. VAN DER HORST, M.D.
CORIANNE A.J.M. DE BORGIE, M.SC.
PATRICK M.M. BOSSUYT, PH.D.
Academic Medical Center
1100 DE Amsterdam, the Netherlands

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Case 15-1998: Elevated *N*-Acetylaspartic Acid Activity in Canavan's Disease

To the Editor: In the discussion of Case 15-1998 (May 14 issue),[1] Canavan's disease is said to be characterized by "the absence of *N*-acetylaspartic acid activity on magnetic resonance spectroscopy." In actuality, *N*-acetylaspartic acid is elevated in Canavan's disease.[2,3]

JONATHAN BRESLAU, M.D.
Radiological Associates of Sacramento
Sacramento, CA 95814

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Should We Accept Mediocrity?

To the Editor: I find it interesting that all of Manian's DEFs (deficiencies, errors, and frustrations) were due to problems with hospital staff and payers (April 19 issue)[1]; none dealt with the multiple problems physicians create daily. I have compiled a list of PDEFs (physicians' deficiencies, errors, and frustrations) — the times when a physician's decision results in an adverse outcome for a patient, unnecessary cost, or both.

PDEF 1

I am a family physician, and I refer a patient to an infectious-disease doctor for vaccines required for overseas travel. The infectious-disease doctor finds the patient has dyspepsia in addition to needing immunization against yellow fever and refers the patient to a gastroenterologist. The gastroenterologist sees the patient limp into the office (he took a spill playing basketball the night before) and refers

the patient to an orthopedic surgeon, who puts the patient on a regimen of ibuprofen and ice. The gastroenterologist prescribes the same medicine the patient was already taking and charges $146 for an initial office visit. The orthopedic surgeon charges only $92.

The Problem: Each physician thinks only inside his or her own box and does not value the relationship the patient has with his family doctor, hence costing the medical system nearly $250 more than necessary but providing no better care. (Using the Ottawa Rules for determining the appropriate medical intervention, I would have managed the ankle injury over the phone.)

PDEF 2

Physician A wants to check a patient's potassium level because the patient is taking a diuretic for high blood pressure. The physician wants to do some screening tests and therefore orders a 22-test chemistry panel and a complete blood count. The bill for the laboratory tests is $146, but only the potassium test is covered by the diagnostic code on the billing form, so the patient is left to pay the remaining $124.

The Problem: The physician uses a "shotgun" approach to diagnosis and screening because it is easier to circle the SMA22 series on the procedure form than each necessary test. The physician is also under the mistaken impression that the use of screening laboratory tests prevents morbidity and reduces mortality.

PDEF 3

Physician B sits in a box seat at a baseball game and eats a catered meal supplied by a pharmaceutical-company representative. Physician B keeps a well-stocked cabinet of drug samples containing all the newest drugs. Physician B takes a full course of clarithromycin from the sample cabinet for his own upper respiratory infection. Physician B prescribes clarithromycin for patients with bronchitis.

The Problem: Physicians are influenced in their prescribing patterns by the easy availability of drug samples. Physicians treat themselves with drug samples. But the newest, most expensive drugs are not always the best medicines for the patient.

PDEF 4

Patient A comes to the emergency department after four hours of chest pain and with ST-segment elevation on the electrocardiogram. The patient is admitted to the intensive care unit and given oxygen and heparin, and serial cardiac-enzyme levels and electrocardiograms are recorded.

The Problem: Despite years of evidence, a considerable percentage of patients with acute heart attacks do not receive the appropriate treatment with aspirin, beta-blockers, and thrombolytic drugs. Changing physicians' behavior is very difficult, even when the evidence of improved outcomes is clear.

Physicians are just as culpable as the parties Manian blames for creating the quagmire in which medical care finds itself. Physicians have profited for years from expensive and inefficient care. I agree that we should never accept mediocrity, but we must take responsibility for cleaning our own house before we begin inspecting others'.

JOHN M. WESTFALL, M.D., M.P.H.
University of Colorado Health Sciences Center
Denver, CO 80220

1. Manian FA. Should we accept mediocrity? N Engl J Med 1998;338:1067-9.

To the Editor: I would like to add a few DEFs of my own to Dr. Manian's list:

DEF 12

Ever since the radiology department has been transformed into the hospital's "imaging department," the access the attending physician has to his or her patients' actual films has deteriorated greatly. I dread my daily sally into the imaging department because, in spite of my best efforts, I have only about a 50 percent chance of being able to find a patient's recent films. Thus, half the time I feel foolish for having wasted time and effort trying to be a compleat physician.

DEF 13

Ever since the medical records department has been rechristened the "health information department," service to patients and physicians has declined. The health information department is now primarily devoted to providing documentation of billable services to payers and various government agencies. Because patients change physicians every time their employers change health plans, nearly every patient admitted is someone I have never met before. Obtaining the old charts of these patients seems to require an act of Congress. It seems more important for billing clerks, nurse case managers, and other bureaucrats to have the charts, rather than the poor doctor trying to obtain a few scraps of reliable information about his or her new patient's history.

DEF 14

Hospital transcription (soon to become "voice processing"?) is so backed up that operative reports take nearly a week to appear on the chart. If I request that mine be processed "stat" they will appear in 24 to 36 hours, but this backs up the system for everyone else. Histories and the results of physical examinations take three to five days to appear on the chart. This is lots of fun when I'm on weekend call and my partner admitted the patient on the previous Thursday or Friday.

What can we do as physicians? Retire early? Find another profession? What we really need is a Hundred Thousand Doctor March on Washington, D.C.

JAMES M. DAVID, M.D.
Idaho Falls Clinic
Idaho Falls, ID 83404

To the Editor: As a patient who happens to have a medical background (albeit in the animal world), I believe that, overall, the quality of actual doctor care (that is, when you get to see one) remains excellent. On the other hand, the quality of office and hospital care seems to be deteriorating as unqualified people attempt the work that used to be done by more highly trained people.

One would like to assume that, as a patient, one should not have to clarify to laboratory personnel what tests were actually ordered by one's physician, explain to one's nurse

what the actual flow rate on a total-parenteral-nutrition infusion should be, or monitor the patency of one's own intravenous line. One would also like to assume that an adult patient undergoing elective surgery requiring anesthesia would have signed the consent form before being heavily sedated. One would even like to assume that a patient could use the call button from his or her hospital bed and get a compassionate response rather than being greeted with "Next time, wait and call me after you've finished vomiting."

Dr. Manian's conclusion that "patients will suffer directly . . . if they haven't already" as a result of "mindless cost cutting" is right on target. The downward spiral that is occurring in our health care system is alarming and should be of concern to every consumer of health care. Unfortunately, many patients are afraid to complain or question those in the medical profession.

SHEILA ADENWALLA, D.V.M.
Fox Valley Technical College
Appleton, WI 54913

To the Editor: . . . Dr. Manian, like many physicians, views his coworkers as barriers to improving quality instead of as partners with valuable contributions to make in improving care.

The principles of quality management are total employee involvement, data-driven decision making, and continuous improvements in all aspects of quality. Physicians who view themselves as part of a team of providers and part of a larger system of care have the opportunity to make dramatic improvements that will benefit patients and the medical profession. Physicians who assign blame to everyone from those in the boardroom to those in the treatment room for poor quality and remain aloof from improvement efforts erode the system and our profession.

CHARLES LEVENBACK, M.D.
University of Texas M.D. Anderson Cancer Center
Houston, TX 77030

Dr. Manian replies:

To the Editor: Dr. Westfall presents several of his own DEFs but does not seem to disagree with any of the DEFs presented in my article. He suggests that because some physicians have faults of their own (many of which are not new), they have no right to express their frustration with a new system of health care delivery that in many instances has made what was once standard care now a luxury (e.g., orders executed properly and experienced health care workers taking care of patients). However, Dr. Westfall himself admits that "changing physicians' behavior is very difficult," and to suggest that physicians should tolerate substandard and mediocre care for their patients until the behavior of their colleagues is perfected is absurd. It is interesting to note that three of the four DEFs listed by Dr. Westfall concern the cost, not the quality, of patient care. Such inordinate emphasis on cost is music to the ears of managed-care administrators and is an unfortunate tribute to those who have successfully divided our profession by placing cost ahead of quality.

I have also experienced the DEFs listed by Dr. David, and many other physicians probably have also. These DEFs further illustrate how the basic support system needed by physicians to provide high-quality care to our patients has been eroded.

Dr. Adenwalla's personal experience with inexperienced and less-than-caring health care workers is a vivid example of the way many institutions have underestimated the importance of having competent staff care for patients during what is one of the most vulnerable periods of their lives.

Finally, if this were the early 1990s, I might agree with Dr. Levenback's idealistic views. However, having endured nearly a decade of managed-care policies driven purely by the bottom line, without regard for the suggestions of physicians, I have become more realistic and less sanguine about the prospect of teamwork.

I agree with the principles of quality management as outlined, but the fact remains that as long as these principles can be applied only under what are often arbitrary budgetary rules imposed by short-sighted businesspeople who have no contact with patients, true teamwork is unlikely. If we don't speak up against mediocre patient care, then we don't deserve to be called physicians.

FARRIN A. MANIAN, M.D., M.P.H.
Infectious Diseases Consultants
St. Louis, MO 63141

Cybermedicine

To the Editor: Readers of the *Journal* should be aware that, in Massachusetts and perhaps other states, some physicians are providing diagnostic and treatment services over the Internet, without having seen or examined patients. This practice involves providing patients with prescribed medication after the patient completes a questionnaire and an on-line, physician-generated "conference" regarding symptoms and medical history. Ostensibly, the provision of this service is rationalized as a way of improving access to medical care and counteracting the lack of physicians in underserved areas, and as a simple convenience to patients. Curiously, one on-line service claims to provide "real time, online, confidential emergency medical care and advice," but it is implied that the service would be most helpful to those "experiencing symptoms of an apparently minor medical illness for which you would like some medical advice and/or initial treatment."[1] The same service recommends that its users "obtain timely medical follow up with a physician, in person," and affirms that "we are not a substitute in any way for conventional medical care." It does not appear to require follow-up by the doctors providing the on-line service itself.

Although I do not wish to impugn the motives of the physicians who are involved in such "cybermedicine," I do want to express my deep concern about both the medicolegal implications and the clinical wisdom of this service. Although a detailed questionnaire may suffice in straightforward cases of, say, ear infections, such tools are no substitute for a physical examination, face-to-face with a patient.[2] In my own field of psychiatry, it is not uncommon for someone to sound quite reasonable on the phone as he or she requests an "urgent" prescription for benzo-

diazepine. When the person is actually seen and examined, however, the clinician may observe needle tracks, signs of self-injurious behavior, or other signs that the person is a poor candidate for such drugs. Or consider the person reporting insomnia and requesting a "simple prescription" for a hypnotic drug. An Internet conference cannot rule out the possibility of a primary sleep disorder, such as obstructive sleep apnea, which would be worsened by conventional hypnotic drugs.

In medically underserved areas, telemedicine — in which the physician can at least see the patient electronically — shows promise, though many questions regarding the quality of care with telemedicine have been raised.[3] But cybermedicine over the Internet is another matter, and it represents a trend all physicians should view with concern.

RONALD PIES, M.D.
297 Bedford St.
Lexington, MA 02420

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Garden of Heaven and Earth, Museum of Fine Arts, Boston RAYMOND WAITEKUS, D.O.