Letters

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A New Range for the Anion Gap

To the Editor: The anion gap, the difference between routinely measured anions and cations of the serum, or $NA^+ - (Cl^- + HCO_3^-)$, is a useful tool for deciphering simple and mixed acid–base disorders. When low, the anion gap can be used to indicate the presence of occult cancer or drug poisoning. The traditional normal value for the anion gap has been 12 ± 4 mEq/L (normal range, 8 to 16 mEq/L), values that textbooks continue to reiterate (1, 2). However, as noted by Winter and colleagues (3) and others (4), the range for the anion gap is much narrower than previously thought because of the use of the ion-selective electrode. Thus, we retrospectively analyzed the serum anion gap in 222 patients with normal serum albumin and creatinine levels. We attempted to eliminate the effects of renal failure and hypoalbuminemia, two factors known to modify anion gap values. The results (mean $\pm$ SD) of our analysis were as follows: serum sodium level, 140 ± 2.7 mEq/L; potassium level, 4.6 ± 0.4 mEq/L; chloride level, 105 ± 4 mEq/L; CO_2 level, 28 ± 2.9 mEq/L; and anion gap, 6.6 ± 2 mEq/L (range, 2.6 to 10.6 mEq/L). This range is almost identical to the value reported by Winter and colleagues (3).

The exact reason for the lower anion gap value is not entirely clear. One possible explanation may be that, with ion-selective electrode technology, serum chloride levels tend to be higher, whereas the serum sodium levels closely resemble serum water levels. These methods are widely used by clinical laboratories, some of which correct this chloride overestimation and use the typical anion gap range as a reference value. Knowledge of the lower range of the anion gap is not only of theoretical interest; it also has practical bedside applications. In a recent study of lactic acidosis in critically ill patients (5), all patients with blood lactate levels higher than 10 mmol/L had anion gap values exceeding 16 mEq/L; 50% of patients with lactate levels between 5 and 9.9 mmol/L and 79% of those with lactate levels between 2.5 and 5 mmol/L had anion gap values less than 16 mEq/L. I believe that this finding results from the fact that the current anion gap value is excessive; if a lower range is adopted, nearly all cases of metabolic acidosis could be easily identified. Also of note, the use of ion-selective electrodes has made pseudohyponatremia obsolete, although use of the term continues.

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External Influences on Left Ventricular Diastolic Pressure

To the Editor: Dauterman and colleagues (1) described the effect on the left ventricular pressure-volume relation of a sudden interruption in inferior vena caval inflow to the right heart during the first 10 to 15 seconds. Although the authors' observations are interesting from a physiologic standpoint, I fail to appreciate their claim regarding the clinical significance or relevance of these observations. First, the reduction in right ventricular filling in the clinical setting is rarely, if ever, as sudden and dramatic as was dictated by the experimental design of the study. Second, the subsequent resumption of flow to the right side of the heart is rarely that abrupt. Third, observations recorded under experimental conditions (which last only for several seconds) cannot be expanded to the clinical continuum. The authors showed that immediately after the interruption of inferior vena caval inflow, left ventricular diastolic pressures decreased. End-diastolic volume (preload), however, did not change, presumably because of the preservation of left ventricular inflow by right ventricular and pulmonary circulation reservoirs. Within a few additional seconds of inferior vena caval obstruction, however, the left ventricular pressure-volume relation deteriorated, and stroke volume decreased. It is inappropriate to discuss practical implications and provide general therapeutic guidelines on the basis of results of such abrupt, short-lived, and artificial experimental conditions. The authors' concluding remarks stating that "... by careful volume reduction ... a considerable portion of the resting left ventricular diastolic pressure can be lowered without necessarily compromising cardiac output" are true (cardiac output is indeed increased by lowering excessive preload); however, these observations have little to do with the results of the study: There was nothing "careful" about volume reduction in the experimental design, and cardiac output was uncompromised for only a few seconds.

When low-output states are being addressed, it is a well-established bedside practice to manipulate various determinants of cardiac output (preload, afterload, contractility, and heart rate), depending on which factors are responsible, and to gauge the response by measuring changes in cardiac output. On the basis of the results, strategies to optimize cardiac output can then be designed. Results of experiments based on a sudden interruption of right ventricular filling and its consequences during the first several seconds, although interesting from physiologic and experimental standpoints, are not relevant to clinical situations.

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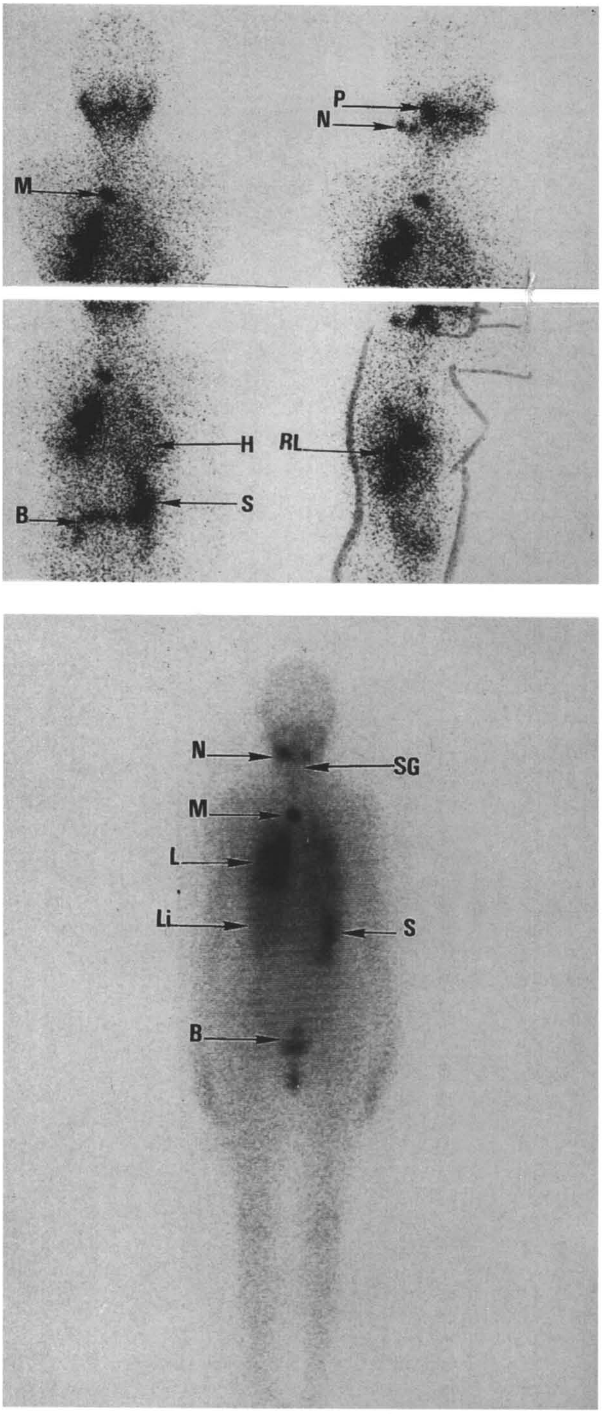

Figure 1. Top. Whole-body scan 48 hours after 4.0 mCi of I^{131}. Abnormal accumulation of the isotope is present in the posterior part of the right side of the neck (*N*), in the superior mediastinum just to the right of the midline (*M*), and in the right lung (*RL*). Normal activity in the blood pool outlines the body and the heart (*H*). Radioiodine is also present in the salivary glands, parotid gland (*P*), the paranasal sinus, the stomach (*S*), and the bowel (*B*). **Bottom. Whole-body scan 1 week after therapy with 314 mCi of I^{131}.** The therapy dose was used as the tracer material. Radioiodine-labeled thyroxine in the circulation shows the body. The image of the liver (*Li*) reflects the enterohepatic circulation of thyroxine. Abnormal uptake is now seen in both lungs (*L*) and in a mass in the superior mediastinum (*M*). The increased uptake of the right side of the neck reflects the nodule in the back of the neck (*N*) and overlying salivary gland. Residual inorganic iodine can be seen in the salivary glands (*SG*), the stomach (*S*), the bladder (*B*), and the genital region below the bladder.

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In response: Dr. Sahebjami suggests that because our study used a rapid transient intervention to identify the physiologic contributors to resting left ventricular diastolic pressure, the results are not relevant to clinical situations. We respectfully disagree. The resting end-diastolic pressures of the left ventricle reflect both intrinsic properties of the heart and extrinsic forces from pericardial and interventricular interactions. The former links altered transmural pressure to volume changes; the latter is associated with no change in transmural pressure but rather with altered pressures at the same volume. Our use of a rapid right heart unloading technique (such as obstruction of inferior vena caval inflow) was aimed at temporally separating the two factors. Near-complete separation did exist for only several seconds, but it was sufficient to yield an accurate quantitative assessment of the external forces other than filling of the left heart chamber. Importantly, this physiologic finding is not an artifact of the particular intervention. It is more precisely and cleanly delineated, however, by use of a rapid intervention.

Many previous studies (1–4) support the notion that right heart filling can be gradually decreased to obtain stable reductions of external cardiac forces; left ventricular pressures can therefore be substantially decreased without commensurate changes in filling. For example, Alderman and Glantz (1) decreased diastolic pressures by 43% with nitroprusside while stroke volume was unchanged; end-diastolic volume decreased by only 14%. The reduction in left ventricular diastolic pressure at preserved preload was not caused by enhanced ventricular performance as Dr. Sahebjami seems to imply, but rather by a decline in the loads external to the left heart. As with our data, these stable responses were principally due to a downward displacement of the diastolic pressure-volume relation. It is not always easy to selectively reduce right heart load without compromising left ventricular filling and transmural pressures. However, gentle titration of pharmacologic agents that are selective for systemic and pulmonary venous beds will probably achieve this result. Our data provide a real physiologic and relevant target at which to aim because they quantify the component of resting measured pressure that is probably due to factors other than direct myocardial stretch within the left ventricle. Successful translation of our results to the routine clinical setting depends primarily on the choice of agents and the care with which they are administered.

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Reducing Suppressive Therapy in Patients with a History of Thyroid Cancer

To the Editor: It is now recommended that levothyroxine doses be reduced when the drug is used to suppress thyroid-stimulating hormone (TSH), so that a thyrotoxicosis-induced increased risk for osteoporosis is minimized in patients with a history of thyroid cancer who have been apparently tumor-free for many years (1–5). However, no data address whether low but detectable TSH levels can achieve the clinical benefit that was seen when TSH was extinguished. Furthermore, a tumor-free state is difficult to definitively ascertain.

We are concerned that some patients with a history of thyroid cancer may be harmed when TSH is unclamped unless they are first evaluated for dormant and unsuspected persistent or recurrent tumor. This concern was heightened by our recent discovery, incidental to a plan to reduce the levothyroxine dose, of unmanifested thyroid cancer that was metastatic to the lung in a 57-year-old woman with asymptomatic osteoporosis and TSH suppression. Thirty years before, papillary thyroid carcinoma had been treated surgically, followed by I^{131} scanning. Whole-body scans in 1965, 1973, and 1981 were negative. The current thyroid sonogram, chest radiograph, and magnetic resonance imaging scan were physiologic, and the test for thyroglobulin (border-line high level) was invalid because of interfering antibodies. Withdrawal of levothyroxine, hypothyroidism, and a whole-body I^{131} scan were needed to reveal the metastatic cancer (Figure 1).

Because of the excellent prognosis when TSH is undetectable, the frequency of late recurrence or of prolonged dormancy of metastatic thyroid cancer has not been examined critically. This patient and other experiences have led us to conclude that each patient should be evaluated for evidence of hidden cancer before the levothyroxine dose is reduced. Reducing the dose without this evaluation could expose the thyroid cancer cells to TSH, the patient to an unknown but perceived increased risk for activating the cancer, and society to increased and probably unnecessary cost.

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Reevaluation of Amiodarone

To the Editor: In his comprehensive review of amiodarone, Dr. Podrid (1) discusses minor thyroid abnormalities, particularly the elevation of thyroid-stimulating hormone (TSH) levels. He does not mention the more common and more problematic elevation in thyroxine levels. Elevation of TSH levels is only transient, and levels gradually return to pretreatment values (2, 3). In contrast, thyroxine and free thyroxine levels remain elevated (2, 3). This condition can be confused with hyperthyroidism, which also occurs in a few patients receiving amiodarone. It can be difficult to distinguish between a euthyroid patient receiving amiodarone and an amiodarone-treated patient who has become hyperthyroid (3, 4). Ultrasensitive TSH assays and measurement of triiodothyronine and free triiodothyronine levels can be useful in this setting (3, 4).

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To the Editor: I read with interest the recent article by Podrid (1). Some important issues arising from this review warrant discussion. First, the mechanism of the observed toxicity may result from the accumulation of substances such as iodine or amiodarone, given that 37.5% of the weight of the drug is iodine. It has been postulated that because both amiodarone and angiographic dyes contain iodine, the combination may cause an additive toxic effect and subsequent death after pulmonary angiography (2). In addition, Sadek and colleagues (3) recently described amiodarone-induced epididymitis and reviewed 12 additional cases in the literature.

Reports of endocrine testicular dysfunction have been published, including one in which 44 men had higher serum concentrations of follicle-stimulating hormone and luteinizing hormone. Serum total and unbound testosterone concentrations correlated inversely with the cumulative amiodarone dose, culminating in a higher incidence of atrophic testes (4).

Dr. Podrid states that warfarin, digoxin, other antiarrhythmic drugs, and anesthetic agents have shown drug interactions with amiodarone. Phenytoin and lithium, two important and widely used agents, should be added to that list. Amiodarone inhibits the metabolism of phenytoin, thereby causing phenytoin toxicity. The phenytoin dose should thus be halved (5).

Thyroid disorders are much more common when lithium and amiodarone are used concomitantly. Clinicians should also take note of patients who are receiving ophthalmic β-blocker eye drops concomitantly with amiodarone. Amiodarone increases serum cholesterol concentrations, an effect that occurs independently of changes in thyroid function.

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In response: I appreciate the comments made by Drs. Ahmed and Goldman and by Dr. Ahmad. They correctly note other toxicities caused by amiodarone not specifically mentioned in my recent review. I recognize that many side effects were not mentioned, but my intent in the review was not to focus on the many toxicities caused by the drug. Its side-effect profile is well known and has been described in several extensive review articles (1–5). Rather, my purpose was to critically examine the many reported clinical trials in which this agent has been used. Certainly, before any antiarrhythmic drug is administered, the risks of therapy must be weighed against the benefits. Indeed, the recommendations for amiodarone use were based on both these concerns.

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Trimethoprim–Sulfamethoxazole Prophylaxis of Spontaneous Bacterial Peritonitis

To the Editor: Singh and colleagues (1) reported that trimethoprim–sulfamethoxazole was effective for preventing spontaneous peritonitis in patients with cirrhosis. They also argued that this drug has a lower cost and fewer associations with selection of gram-positive bacteria than norfloxacin. Although I agree with their arguments, their results may lead to a different conclusion regarding the efficacy of prophylactic trimethoprim–sulfamethoxazole. Their results appear impressive; 8 of 30 controls and 1 of 30 patients receiving prophylaxis developed spontaneous peritonitis or spontaneous bacteremia. However, two controls had enterococcal peritonitis, and one had *Klebsiella pneumoniae* bacteremia, conditions that one would not expect to be prevented by trimethoprim–sulfamethoxazole. If these three episodes are excluded from the analysis, the difference between the two groups is no longer statistically significant, whether or not the patients are kept in the analysis ($P = 0.19$ and 0.09, respectively; Fisher exact test). Assuming that the *Klebsiella* strain was sensitive to trimethoprim–sulfamethoxazole, the difference between the two groups remains nonsignificant if the two episodes of enterococcal infection are excluded ($P = 0.10$); however, the difference is marginally significant if these 2 patients are excluded from the analysis ($P = 0.048$). This lack of statistically significant differences may be the result of the study's relatively small sample size; however, until further studies confirm Singh and colleagues' findings, one must be careful when recommending long-term chemoprophylaxis with trimethoprim–sulfamethoxazole to patients with cirrhosis.

It would be interesting to know the results of an analysis of only those patients who had previously had spontaneous bacterial peritonitis. Long-term chemoprophylaxis may be more clearly beneficial for such patients.

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In response: Dr. Nishioka states that three episodes of peritonitis in the control group could not have been prevented by trimethoprim–sulfamethoxazole. This is incorrect. Trimethoprim–sulfamethoxazole is active against *Klebsiella pneumoniae* with minimum inhibitory concentrations ranging from 0.05 to 3.1 μg/mL (1). The isolate in the control patient with *K. pneumoniae* bacteremia was sensitive to trimethoprim–sulfamethoxazole, as are 83% of the *K. pneumoniae* isolates in our institution. Trimethoprim–sulfamethoxazole also has in vitro activity against enterococci (2); however, the activity of the drug is inconsistent, and this agent cannot be recommended for treating serious enterococcal infections.

A history of spontaneous bacterial peritonitis represents one of several risk factors for peritonitis in patients with cirrhotic ascites. The number of patients with previous spontaneous bacterial peritonitis in our study, however, was too small (27% in the prophylactic group and 17% in the control group) to allow statistical analysis of infections in these patients alone.

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Renal Aneurysms and IgA Nephropathy

To the Editor: We describe a patient with previously diagnosed IgA nephropathy presenting with persistent macrohematuria and iron-deficiency anemia, in whom angiography showed multiple bilateral renal aneurysms.

In January 1993, a 31-year-old woman was hospitalized because of persistent macrohematuria and moderate iron-deficiency anemia. She did well until 1987, when she experienced two episodes of gross hematuria. A percutaneous renal biopsy specimen showed a moderate focal and segmental mesangial proliferation associated with a discrete increase in the mesangial matrix in 75% of the glomeruli. Tubulointerstitial changes were minimal, and there was no evidence of vasculitis. The immunopathologic pattern was characterized by diffuse deposits of IgA in the mesangial areas. Special attention was placed on excluding the many diseases commonly associated with mesangial IgA deposits.

In 1992, the number of recurrent episodes of gross hematuria increased, becoming persistent macroscopic hematuria. Blood pressure and renal function remained normal. At hospital admission, urine was grossly hematuric, but erythrocyte casts could not be detected on microscopic examination. The 24-hour urine protein excretion was 0.5 g, and the creatinine clearance was 102 mL/min per 1.73 m^2 body surface area. Laboratory findings included microcytic hypochromic anemia. The blood hemoglobin level was 8.4 g/dL. A renal biopsy specimen showed only minimal glomerular changes, and immunofluorescence studies were negative. Cystoscopy indicated that gross hematuria originated from the left ureter, but retrograde ureteropyelography did not detect the cause of bleeding. Abdominal aortography with bilateral selective renal angiography showed multiple intraparenchymal aneurysms in the left and right kidneys. All aneurysms were located in the interlobar arteries or arteries distal to the interlobar arteries. The aneurysms were small and relatively uniform in size, with diameters of 2 to 5 mm.

Renal aneurysms in medium- or small-sized arteries have long been recognized in patients with systemic necrotizing vasculitis of the polyarteritis nodosa group (1, 2). However, they have recently been seen, although more rarely, in patients with severe arterial hypertension, rheumatoid arthritis, temporal arteritis, and diabetes mellitus (2, 3). Our patient did not have any evidence of the systemic diseases known to be associated with angiographically established visceral aneurysms (1–3). This report is the first description of bilateral renal aneurysms in primary glomerulonephritis.

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Percutaneous Transcatheter Arterial Embolization for Hypersplenism

To the Editor: Portal hypertension is commonly associated with splenomegaly and, at times, with functional hypersplenism and its attendant peripheral blood cytopenias. Splenectomy has been the most frequently used procedure for hypersplenism, but it is associated with a 30-day mortality rate that approaches 20% (1). In patients with underlying cirrhosis, infections developing after splenectomy are also a prominent problem (2, 3). Other surgical procedures, such as splenic artery ligation or portosystemic shunting, produce limited clinical improvement.

We report a case of successful percutaneous transcatheter arterial embolization for treatment of a patient with hypersplenism associated with cirrhosis and hepatocellular carcinoma. A 54-year-old nonalcoholic Arabic man who had had idiopathic thrombocytopenic purpura for 4 years was evaluated for a 2-week history of right upper-quadrant pain. Physical examination, a computed tomographic scan of the abdomen, and a subsequent biopsy specimen showed multicentric hepatocellular carcinoma. Initial laboratory studies showed a platelet count of 32 000 cells/mm^3, a leukocyte count of 5400 cells/mm^3, and a hemoglobin level of 13.5 g/dL; results of serum biochemical studies were within normal limits except for an elevated serum alanine aminotransferase level (108 IU/L; normal, 7 to 56 IU/L). No platelet-specific antibodies were seen. The patient had serologic reactivity to hepatitis B and C viruses but not to human immunodeficiency virus type 1 or 2. A bone marrow biopsy specimen showed normal elements and 60% cellularity. Hepatocellular carcinoma was treated with chemoembolization with cisplatin, doxorubicin, mitomycin C, and polyvinyl alcohol foam, followed by systemic 5-fluorouracil and recombinant human interferon-α. During the next 18 months, the thrombocytopenia became resistant to intravenous immunoglobulins, and the patient developed persistent neutropenia when not receiving therapy. The patient had three splenic embolizations with Gelfoam particles and cefoperazone through a right femoral arterial approach over 4 weeks, and his platelet count increased to more than 190 000 cells/mm^3 without further therapy.

Splenic embolization with subtotal infarction is usually well tolerated and represents a potential alternative for patients with hypersplenism secondary to portal hypertension who may not be surgical candidates (4, 5).

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SIADH in a Patient Receiving Sertraline

To the Editor: Selective serotonin re-uptake inhibitors are better tolerated than older antidepressant agents but are associated with side effects of which internists may not be aware. One such side effect is the syndrome of inappropriate antidiuretic hormone secretion (SIADH). Life-threatening hyponatremia due to SIADH has been described with both fluoxetine (Prozac, Dista Products Co., Indianapolis, Indiana) (1) and paroxetine (Paxil, SmithKline Beecham Pharmaceuticals, Pittsburgh, Pennsylvania) (2). Recently, several reports in the psychiatric literature have described the occurrence of SIADH due to another selective serotonin reuptake inhibitor, sertraline (Zoloft, Roerig, New York, New York) (3–5). We describe an additional case of sertraline-induced life-threatening hyponatremia and wish to alert internists to this possible complication.

A 59-year-old woman with a history of depression and chest pain presented with progressive lethargy. Her medications included aspirin, metoprolol, and nitroglycerin patches, which she had been receiving for several years without incident. Three weeks before admission, the patient began receiving sertraline, 25 mg/d. Her serum sodium level was normal at that time. At hospital admission, her vital signs were normal and no edema was noted. Results of a neurologic examination did not show focal neurologic abnormalities, but the patient was lethargic and nonverbal. Her serum sodium level was 117 mEq/L, her blood urea nitrogen level was 4 mg/dL, her serum creatinine level was 0.4 mg/dL, and her urine sodium level was 189 mmol/L. The patient's serum osmolality was 233 mOsm/kg, and concurrent urine osmolality was 465 mOsm/kg. A diagnosis of SIADH was made, and sertraline was stopped. Intravenous normal saline and furosemide were started. The patient's serum sodium level returned to normal by hospital day 4, and no recurrence of hyponatremia has been noted during 7 months of follow-up.

The development of hyponatremia shortly after the start of sertraline therapy strongly implicates this drug. The onset of hyponatremia within several weeks of starting sertraline and the degree of hyponatremia are both consistent with the findings of previous reports (3–5). Internists should be aware of the risk for hyponatremia with this class of medication, as well as other recognized side effects and drug interactions related to selective serotonin reuptake inhibitors.

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Not for Money, but for Life

To the Editor: One day, one of our critical care nurses asked me for help: Her mother had been admitted to a hospital in Nigeria because of heart block. The pacemaker necessary for her survival was unavailable, and no fluoroscopy tubes were functional. Several attempts were made to contact the International Red Cross, the World Health Organization, and Doctors without Borders, but lack of funding or insufficient organization quickly made us realize the helplessness of these associations in solving this emergency. I then called our Medtronic representative and received a free pacemaker unit. To obtain a visa for the patient, a colleague in Lagos sent me a fax stating the patient's medical condition. I submitted this fax to the urgent attention of a U.S. senator, along with a personal affidavit supporting the need for an emergency pacemaker implantation. A few days later, the patient arrived in the United States and was admitted to a local community hospital. At that time, she was lightheaded and had symptoms of congestive heart failure. An electrocardiogram showed a complete heart block with an escape rate of 28 beats/min. The pacemaker unit was delivered to the hospital, where two colleagues, a cardiologist and a surgeon, agreed to implant it free of charge. The patient was discharged in good condition 2 days later.

The saving of this human life raised several questions: Should it be made easier to contact an international organization that would be able or willing to help in similar situations? Should not health care spending be better allocated and redistributed in Third World countries? Should the industrialized nations help in structuring an improved health care system for less privileged countries? Will the more cost-conscious approach of managed care render this kind of generous enterprise impossible? If so, is a human life a cost or a sheer benefit?

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Prioritizing or Rationing Health Care

To the Editor: Dr. Caplan (1) presents for our serious consideration a recent Swedish approach to setting priorities for health care resources. Dr. Caplan suggests that adopting the simple ethical principles of this Swedish approach would provide an "enormous boon to health policy," that is, an orderly approach to reform of the U.S. health care system.

Dr. Caplan's recommendation has two disabling flaws. The first concerns the centrality of the value of solidarity. Put aside for the moment that the use of this language in Europe—and Canada—may be a device to paper over cultural and moral pluralism in a misguided nostalgia for cultures in which everyone is essentially alike. Solidarity is not a fundamental U.S. value. The United States consists of a far more pluralistic, contentious, regional, even neighborhood people and culture than Sweden—or any other country, for that matter. As a consequence, there can be found in the United States serious, thoughtful advocates of divergent views of the obligations of society to its vulnerable members and about the priority that should be given to controlling health care costs. We cannot simply import consensus about health care; if U.S. history is a reliable guide, we may never be able to achieve it, short of a national emergency. Proposals that we look to other countries' responses, as if we could simply import them here, are therefore unrealistic. We must build consensus, which is hard work (2).

Second, we do not have a health care system to reform, because we do not have a health care system. We are nonsystematic, perhaps even antisystematic, in the manner in which we provide health care and nearly every other basic social service (3). The failure to appreciate this fact explains far better the failure of the Clinton health care legislation than anything having to do with rationing. Dr. Caplan's continuing attachment to the misconception that we have a system in the first place shows how deeply ingrained this failure is.

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2. **Moreno JD.** Deciding for Others: Bioethics and Moral Consensus. New York: Oxford University Pr; 1995.
3. **McCullough LB.** Should we create a health care system in the United States? J Med Phil. 1994;19:483-90.

In response: Dr. McCullough has peeked out from his bunker near the Alamo long enough to espy cultural homogenization creeping toward him under the cover of my editorial on rationing (1). However, I fear that his paranoia at the suggestions that 1) any other nation might have something to teach us about health reform or 2) that talk of solidarity will grind out the cultural, religious, and ethnic differences that make the United States a great nation may have distorted his perception of my views.

I could not agree more with Dr. McCullough that the United States does not have a health care system to reform. One of the few things that the Clinton administration did understand in its bungled effort at reform was this fact. I do not believe, as Dr. McCullough suggests, that if Americans could simply import the loving, neighborly value of solidarity from the Swedes into our collective moral psyches, our health care costs would dip and the quantity and quality of services available here would improve. Health care in Sweden costs a lot, and despite the country's relative cultural homogeneity, those who dwell there spend endless hours contentiously arguing about what to do about this expense.

My point in citing the Swedish approach to setting priorities was that in trying to figure out what is good to pursue in health care, debates about rationing in the United States bog down. They probably do so for some of the reasons Dr. McCullough cites. However, if we could follow the Swedes, not so much in importing the content of their values but in their moral focus, by starting our debates about rationing and setting priorities over what is not allowed and what is morally off the table, we will get a lot further in reforming our mishmash of a health care system than we have to date.

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Reference

1. **Caplan AL.** Straight talk about rationing [Editorial]. Ann Intern Med. 1995;122:795-6.

Screening Surgeons for HIV Infection

To the Editor: Owens and colleagues (1) conclude that counseling and testing all U.S. surgeons for human immunodeficiency virus (HIV) at a cost of $57 for each surgeon would not be a cost-effective approach for HIV control. However, the prevalence of HIV varies by more than 10-fold among U.S. states (2, 3). We agree that routine HIV testing in low-prevalence areas does not make much sense, but we remain unconvinced that a more thoughtful, geographically targeted approach in severely affected states, districts, or cities might not be cost-effective. Furthermore, as we reported for the U.S. military (4), HIV screening programs cost less than $10 per test. The outcome of the analysis by Owens and colleagues might have been different if actual contract-negotiated costs rather than free-market, inflated cost figures had been used. We encourage Owens and colleagues to revise their cost-effectiveness model to address real costs, limited to geographic regions selected on the basis of HIV prevalence data.

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References

1. **Owens DK, Harris RA, Scott PM, Nease RF Jr.** Screening surgeons for HIV infection. A cost-effectiveness analysis. Ann Intern Med. 1995;122:641-52.
2. **Burke DS, Brundage JF, Goldenbaum M, Gardner LI, Peterson M, Visintine R, et al.** Human immunodeficiency virus infections in teenagers: seroprevalence among applicants for US military service. JAMA. 1990;263:2074-7.
3. **Wasser SC, Gwinn M, Fleming P.** Urban-nonurban distribution of HIV infection in childbearing women in the United States. J Acquir Immune Defic Syndr. 1993;6:1035-42.
4. **Brown AE, Burke DS.** Cost of HIV testing in the U.S. Army. N Engl J Med. 1995;332:963.

In response: Drs. Burke and Brown suggest that test costs may be lower than the estimates we used and that geographically targeted screening may be more cost-effective than screening all surgeons. We agree that some purchasers will be able to negotiate for relatively low-cost HIV tests. The military HIV screening program is particularly efficient (1). Our estimate of $57, however, represents the total cost per patient screened and includes the cost of the enzyme immunoassay and any necessary confirmatory tests, counseling before and after the test, and administrative overhead. The cost of the tests alone is a minor component of the overall expenditures per person screened. Our costs reflected those paid by a large purchaser of health care. If, however, the total cost per person screened could be reduced from $57 to $10, the cost-effectiveness of one-time screening program improves from $458 000 to $402 000 per year of life saved.

As noted by Drs. Burke and Brown, if a screening program were targeted geographically, the prevalence among the surgeons might be higher than that in a national screening program. Our base-case analysis assumed that the prevalence of HIV infection among surgeons was approximately 0.1%. If the prevalence of HIV infection among screened surgeons in a targeted program were 1% (10 times higher than our base-case estimate and substantially higher than any published estimates of HIV infection among physicians) and if the total program costs were reduced to $10 per person screened, one-time screening costs $382 000 per year of life saved. Thus, the cost of tests and counseling and the prevalence of HIV infection among surgeons only modestly influence the cost-effectiveness. Their effect is modest because the induced costs (the cost due to the decreased services provided by surgeons identified as having HIV infection) of a program to screen surgeons are high (2). In contrast, the prevalence of HIV infection in the screened population and the test and counseling costs have a more marked effect on the cost-effectiveness of programs to screen patients because these programs do not incur similar induced costs. In summary, even an efficient, geographically targeted screening program for surgeons would cost substantially more per year of life saved than most commonly accepted medical interventions.

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References

1. **Brown AE, Burke DS.** Cost of HIV testing in the U.S. Army. N Engl J Med. 1995;332:963.
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Preventing Firearm Violence

To the Editor: The American College of Physicians (1) has established its position in the gun control morass but stands on a precarious notion: that firearm violence is a public health issue in the sense that tuberculosis control and childhood immunizations are public health issues. Firearm violence is clearly a public safety issue, but I cannot envision our local health department workers regulating firearm ownership while they are arranging WIC services. I believe that labeling this social and moral problem as a public health failure is at best a well-intentioned but misguided effort to save lives. At worst, it is an intentional manipulation of the College members into tacit support of gun control as a solution to a distressingly complex problem.

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Reference

1. **American College of Physicians.** Preventing firearm violence: a public health imperative. Ann Intern Med. 1995;122:311-3.

To the Editor: The College's position paper (1) does not represent the opinion of many College members. Most people would agree that firearm safety, responsible storage of unloaded firearms, and respect for the possible hazards of firearms are reasonable and necessary. However, to say that "regulatory oversight" of guns or ammunitions is desirable misrepresents the need for a safer society. History tells a different story. In Nazi Germany, for example, Hitler instituted a gun registration policy.

Has any nation ever benefited from gun control? The College's support of legislative and regulatory measures that would limit the availability of firearms overextends the bounds of the organization.

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Reference

1. **American College of Physicians.** Preventing firearm violence: a public health imperative. Ann Intern Med. 1995;122:311-3.

To the Editor: In reviewing the College's position paper on preventing firearm violence (1), we are struck by the fact that although it is written by persons with careers in science, it lacks the attributes of a scientific approach to problem solving. The most basic strategy in science is to collect and analyze all available facts and data. This was simply not done; for every controversial assertion in the position paper, there is an egregious lack of balancing argument.

In their haste to link guns causally to the deaths of young black males, the authors did not mention that the groups in the United States for whom firearms ownership is the highest have the lowest homicide rate (2). The data showing that firearms, including handguns, can save lives (3), prevent the completion of crime, and limit personal injury (2) were not reviewed. Instead, the authors cite flawed and criticized (4) public health research and an essay written by an employee of Handgun Control, Inc. In depicting firearm owners as unsafe, the position paper omitted the facts that the death rate from firearms-related accidents continues to decrease (5) and that hunting—a sport that requires all participants to be armed—was the safest outdoor participant sport in the last year for which data are available.

We would expect the true scientist, wishing more than anything to know the truth, to be delighted by disparate facts or data; it is the reconciliation of these data that constitutes discovery. Such a scientist should be eager to bring these data out and share them with colleagues, because shared views result in a splendid intellectual quality that we know as insight. Unfortunately, the treatment in the medical literature of the subject of firearms and violence, as typified by the position paper, lacks these qualities; brought to the fore are only those data that support a particular agenda.

The marriage of scholarship and objectivity is that which separates the sciences from less progressive endeavors. When neither is present, we are simply a group with a political opinion.

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2. **Kleck G.** Point Blank: Guns and Violence in America. Hawthorne, NY: Aldine de Gruyter; 1993.
3. **Kleck G, Gertz M.** Armed resistance to crime: the prevalence and nature of self-defense with a gun. Journal of Criminology and Criminal Law. 1995;86:[In press].
4. **Kates DB, Schaffler HE, Lattimer JK, Murray GB, Cassem EH.** Guns and public health: epidemic of violence or pandemic of propaganda? University of Tennessee Law Review. Summer 1995.
5. Accident Facts: 1992 edition. Chicago: National Safety Council; 1992.

To the Editor: I believe that the College's recent recommendation on gun ownership (1) is fundamentally flawed. The recommendation is contrary to long-standing criminologic experience, violates the fundamental rights enjoyed by a free people, and, if enacted as law, might increase firearm-related deaths.

Cesare Beccaria (2), the founder of scientific criminology, understood the folly of gun control laws in practical terms:

> False is the idea of utility that sacrifices a thousand real advantages for one imaginary or trifling inconvenience. . . . The laws that forbid the carrying of arms are of such a nature. They disarm those only who are neither inclined nor determined to commit crimes. . . . Such laws make things much worse for the assaulted and better for the assailants; they serve rather to encourage than to prevent homicides, for an unarmed man may be attacked with greater confidence than an armed man.

The framers of the U.S. Constitution understood the fundamental right of a free people to keep arms. The Second Amendment uses the phrase "a well-regulated militia" to describe the people who enjoy this right. This phrase, in historical context, defined the armed volunteer adult male citizenry.

Finally, the recommendation may increase the risk for firearm-related injury and death. The National Academy of Sciences concluded that "self-defense gun use . . . is associated with a reduced risk of physical attack and injury" (3). Indeed, a restriction on handguns might actually increase the number of deaths related to firearm injuries. Kleck (4) has estimated that rifles or shotguns could easily be substituted for handguns in 54% to 80% of all homicides. Because the ballistics of long guns render them inherently more lethal than handguns, a prohibition of handguns might increase the mortality rate from armed crimes.

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2. **Beccaria C.** On Crimes and Punishment. Indianapolis, IN: Bobbs-Merrill; 1963:87-8.
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In response: Gun-related injury in the United States claimed 38 000 lives in the 12 months preceding October 1994, a number 5 to 10 times higher than that in other developed countries. Since 1980, more than 50 000 U.S. children have died of gunshot wounds; for African-American male youths, gun-related homicide is the leading cause of death. Certainly Drs. DeHart and Nacol would agree that these potentially preventable deaths are a national tragedy that must be addressed.

Violence prevention, building on the older field of injury control, is a dynamic new field of public health recognized by the American College of Physicians, the American Medical Association, the American Public Health Association, and the Centers for Disease Control and Prevention (1). The quality and quantity of the science is growing as more researchers enter this field.

We agree with Drs. Waters and Roosevelt that efforts to reduce firearm injuries should be informed by scientifically sound evidence. We acknowledge, as Kleck agrees, that epidemiologic and experimental research in gun injury is complex, and we urge that the field expand (2). The conflicting claims that firearm ownership enhances personal safety or increases the chance of death fourfold are at least equally subject to methodologic critique. Dr. Roosevelt argues that legal firearm possession deters crime and that gun use in self-defense reduces injury, but a recent study of the easing of laws for carrying concealed firearms suggests that in five cities in three states, the overall incidence of homicide increased after the laws went into effect (3). The recent *JAMA* report stating that persons who had more training in gun safety used less safe practices with guns in the home, particularly where children were living, leads to the question of whether gun safety training is enough (4).

Finally, it is important to recognize that in the United States, more than half of all gun-related deaths are suicides, not associated with issues of self-defense or criminal deterrence. The field of suicide prevention must deal more directly with handguns, the most common instrument of suicide. We believe it is important to explore gun control as one possible way in which the disputes between domestic partners and acquaintances that lead to violence might be made less lethal.

The College urges a comprehensive approach to gun violence prevention, including defining and modifying risk factors and educating the public and policymakers about this important risk to American lives.

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American College of Physicians

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1. Violence and the Public's Health. Health Affairs Special Issue. 1993; Winter.
2. **Reiss AJ, Roth JA, eds.** Understanding and Preventing Violence. Washington, DC: National Academy of Science Pr; 1993.
3. **Kleck G.** Point Blank: Guns and Violence in America. Hawthorne, NY: Aldine de Gruyter; 1993.
4. **McDowall D, Loftin C, Weiersema B.** Easing concealed firearm laws: effects on homicide in three states. Journal of Criminal Law and Criminology. 1995.
5. **Hemenway D, Solnick SJ, Azrael R.** Firearm training and storage. JAMA. 1995;273:46-50.

Correction: Error in Citation

The work of Dr. A.J. Bollet was incorrectly cited in a recent article (1). The following is the correct citation for Dr. Bollet's work:

Bollet AJ. Politics and pellagra: the epidemic of pellagra in the U.S. in the early twentieth century. Yale J Biol Med. 1992;65: 211-21.

Reference

1. **Elmore JG, Feinstein AR.** Joseph Goldberger: an unsung hero of American clinical epidemiology. Ann Intern Med. 1994;121:372-5.

Correction: Vienna 1938

In a recent paper on the damage caused by the Nazis to the Medical Faculty in Vienna (1), the French writer considered to be a progenitor of racist Nazi theories was incorrectly listed as Duke Gibenau. The writer's name is Count Joseph-Albert de Gobineau.

Reference

1. **Ernst E.** A leading medical school seriously damaged: Vienna 1938. Ann Intern Med. 1995;122:789-92.