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Helicobacter pylori and Symptomatic Relapse of Gastro-esophageal Reflux Disease

ABSTRACT & COMMENTARY

Synopsis: *This elegant study indicated that a group of international patients with GERD and concomitant infection with Helicobacter pylori had definite GERD improvement when H pylori was successfully eradicated.*

Source: Schwizer W, et al. *Lancet.* 2001;357:1738-1742.

This article addresses the highly controversial relationship between *Helicobacter pylori* infection and gastroesophageal reflux disease (GERD). Schwizer and colleagues studied 70 GERD patients as defined by either erosive esophagitis and/or abnormal esophageal acid exposure. All patients received lansoprazole 30 mg b.i.d. for 10 days, followed by 30 mg daily for an additional 8 weeks. Patients with documented *H pylori* infections were randomized to receive placebo or antibiotics for the first 10 days (clarithromycin 500 mg b.i.d. and amoxicillin 1000 mg b.i.d.). Clinical follow-up was performed for 6 months at 2-week intervals, and endoscopy and pH monitoring were again performed at the end of the study. A total of 58 patients completed the trial, and 16 patients were *H pylori* positive at the study conclusion (14 placebo recipients and 2 eradication failures). Thirteen patients were negative due to successful *H pylori* eradication and there were 29 controls who completed the study. The *H pylori*-positive patients relapsed earlier (54 days) than those in whom *H pylori* had been eradicated (100 days; $P = .046$). *H pylori*-negative controls relapsed after the longest interval (110 days). Esophagitis grade also affected relapse. Those with no esophagitis relapsed in 127 days and grade III or IV esophagitis led to relapse after only 18 days. Corrected by esophagitis grade, *H pylori*-positive patients relapsed earlier than *H pylori*-negative patients and controls. ($P = .001$).

COMMENT BY MALCOLM ROBINSON, MD, FACP, FACG

Gastroesophageal reflux is clearly caused by acid contact with esophageal mucosa although the pathophysiology is complex,

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INSIDE

including motility and acid secretory factors as well as local mucosal defenses. Some previous studies have suggested that *H pylori* might protect against both GERD and its more severe complications. Eradication of *H pylori* in duodenal ulcer disease has led to development of GERD in one study although other studies suggest that GERD symptoms might improve after cure of *H pylori* infection. Issues that could be involved in such divergent outcomes might include differing *H pylori* strains, their differing intragastric distributions, and the varying severities of accompanying gastritis.

The present study does not allow us to answer the basic question of how *H pylori* will affect reflux disease that already exists or the triggering of new GERD symptoms or damage by either its presence or its eradication. It is unfortunate that this study was small and that it did not permit subdivision of possible organism virulence and anatomic involvement characteristics. It seems likely that patients who relapsed sooner in conjunction with *H pylori* infection eradication must have had relative hypersecretion of acid, well known to occur in a subgroup of *H pylori* infections. Had other patients been included with gastritis and impaired acid secretion resulting from *H pylori* infections, eradication would have produced the opposite result. Schwizer et al found little gastric atrophy in patients entering or leaving this study. Although Schwizer et al recommend *H pylori* eradication in patients with GERD, this applies only to the specific patient population and characteristics involved in this study. Other patients probably would demonstrate quite different results, and many experts (including this one) still believe that the only rational approach to *H pylori* in patients presenting with GERD signs or symptoms should remain, "Don't ask, don't tell." ❖

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In-Hospital CPR: When to Stop

ABSTRACT & COMMENTARY

Synopsis: *Survival to hospital discharge following in-hospital cardiac arrest and CPR could be predicted with 99% sensitivity using a clinical decision aid that incorporated whether the arrest was witnessed, whether the initial cardiac rhythm was ventricular tachycardia or ventricular fibrillation, and whether a pulse was regained within the first 10 minutes.*

Source: van Walraven C, et al. *JAMA.* 2001;285:1602-1606.

Van walraven and colleagues sought to validate a previously derived clinical decision aid to reliably predict patients who would have a poor outcome from in-hospital cardiac arrest. They used data from a large registry of in-hospital resuscitation attempts at a community-teaching hospital to determine whether the answers to 3 questions could be used to predict survival to hospital discharge:

1. Was the arrest witnessed?
2. Was the initial cardiac rhythm either ventricular tachycardia or ventricular fibrillation?
3. Was a pulse regained during the first 10 minutes of chest compressions?

If the answer to any one of the above questions was "yes," the patient was classified as having a reasonable

likelihood of survival following resuscitation. If none of the questions could be answered in the affirmative, the patient was classified by the decision aid as having no chance for survival.

Data used were from 2181 cardiac resuscitation attempts (in 1884 patients) at the 550-bed hospital from 1987 through 1996. In 15.1% of resuscitations (327/2181), the patient survived to hospital discharge. For 99.1% of these successful resuscitations (324/327), the decision aid would have placed the patient in the favorable prognostic group (95% confidence interval, 97.1-99.8%). Only 3 of 269 patients (1.1%) who were predicted by the decision aid to have no chance of survival did survive to hospital discharge (negative predictive value, 98.9%), and none of these 3 individuals was able to live independently following discharge. van Walraven et al; conclude that this decision aid can be used to help physicians to identify patients who have an extremely small likelihood of benefiting from continued resuscitative efforts.

COMMENT BY DAVID J. PIERSON, MD, FACP, FCCP

A number of factors have been shown to be associated with poor outcomes from attempted resuscitation from in-hospital cardiopulmonary arrest. These include such pre-arrest patient characteristics as hypotension, renal failure, metastatic cancer, pneumonia, and low functional status. Initial cardiac rhythms other than ventricular tachycardia or ventricular fibrillation are associated with unsuccessful resuscitation attempts. In addition, the longer the duration of attempted cardiopulmonary resuscitation, the less the likelihood of patient survival. Previous decision aids based on these and other factors have proven either too cumbersome for quick clinical application or have not been validated in relevant patient populations. This study appears to overcome these problems.

In their discussion, van Walraven et al point out that clinical decision aids should meet several strict methodological standards. These include a clinically important and easily determined outcome (in this case, survival and the ability to live independently) and the requirement that the aid itself be clinically sensible, using components that have been associated with survival in other studies. The latter is met by the present study, since whether an arrest is witnessed, whether ventricular tachycardia or ventricular fibrillation is the initial rhythm, and the duration of resuscitative efforts have all been independently associated with survival in previous studies. In addition, the aid should be validated in a well-defined, appropriately described population.

Use of the simple 3-factor decision aid described in this article could help clinicians to decide when resuscitative efforts should be discontinued after in-hospital cardiac arrest, thus preventing prolonged but ultimately futile resuscitations and the unfruitful use of critical care resources. As van Walraven et al point out, it could also be helpful in discussions with patients about resuscitation in the event of cardiac arrest: For patients who wished not to be subjected to invasive life support without a reasonable likelihood of benefit, the decision aid could be used to assure them that their wishes would be honored. ❖

Dr. Pierson is Professor of Medicine, University of Washington, Medical Director, Respiratory Care, Harborview Medical Center, Seattle, Wash.

Pioglitazone Improves Lipid Profiles More Effectively than Rosiglitazone

ABSTRACT & COMMENTARY

Synopsis: *When switching from troglitazone to other insulin sensitizers, pioglitazone improved lipid profiles significantly more than rosiglitazone.*

Source: Gegick CG, Altheimer MD. *Endocr Pract.* 2001; 7:162-169.

The objective of this study was to compare short-term glycosylated hemoglobin (HbA1c), lipid, weight, tolerability, and hepatic effects after switching patients with type 2 diabetes from troglitazone to either pioglitazone or rosiglitazone treatment.

A total of 144 and 125 patients met the criteria for comparison of HbA1c and lipids, respectively. HbA1c decreased 0.08% for each treatment group, after a mean 3.2 months of observation. Mean cholesterol, triglyceride, and low-density lipoprotein (LDL) cholesterol levels decreased in the pioglitazone group by 4.7%, 11.3%, and 7.3% but increased 8.4%, 38.4%, and 8.1%, respectively, in the rosiglitazone group. Mean high-density lipoprotein (HDL) increased 2.6% with pioglitazone and decreased by 6.3% with rosiglitazone.

Patients receiving a statin concomitantly when switched to rosiglitazone treatment had a 51.9% increase in mean triglyceride levels vs. a 25.7% increase in those not receiving a statin, whereas the patients

switched to pioglitazone therapy had respective decreases of 14.2% and 6.2%. Both drugs were generally well tolerated and had similar slight weight increases and no hepatic dysfunction.

Switching to pioglitazone caused a trend toward lipid profile improvement, but switching to rosiglitazone therapy caused a significant increase in all lipids, except HDL, which was lowered. Patients receiving statins when switched to rosiglitazone had particularly notable triglyceride worsening. Whether these effects will lead to changes in cardiovascular outcome or will be maintained over a longer period of time remains to be established.

Table

Lipid Changes After Changing From Troglitazone

	Before	After
	Pioglitazone	
Lipid	mg/dL	mg/dL
Total Cholesterol	190.6	181.6
Triglycerides	208.5	184.9
HDL	46.7	47.9
LDL	104.6	97.0
	Rosiglitazone	
Total Cholesterol	180.0	195.0
Triglycerides	178.7	247.4
HDL	44.1	41.3
LDL	100.0	108.1

COMMENT BY RALPH R. HALL, MD, FACP

Despite the short-term nature of this study and the lack of patient randomization, these are remarkable changes in risk patterns. Further, this is how it happens in our practices. Other medications such as metformin, monotherapy with troglitazone, sulfonylurea treatment, and others were similar for each group of patients.

The changes seen in HDL cholesterol and triglycerides suggest that the patients switched to rosiglitazone had an increase in the more atherogenic small dense LDL. With these changes and the availability of pioglitazone, it would be difficult to maintain treatment with rosiglitazone until more safety and long-term studies were completed.

One other item of note is that these patients had been treated for their HbA1c and lipid goals prior to the change from troglitazone to the other thiazolidinediones. The study also demonstrates that the national goals for HbA1c and lipids can be met in clinical practice. ❖

Bone Mineral Density in Subjects with Mild Asthma

ABSTRACT & COMMENTARY

Synopsis: *This current report found that there was no change in bone mineral density over 2 years in patients with mild asthma who were taking inhaled corticosteroids vs. noncorticosteroid treatment. Furthermore, asthma control was better in patients taking inhaled corticosteroids.*

Source: Tattersfield AE, et al. *Thorax.* 2001;56:272-278.

The development of osteoporosis is a major concern with oral corticosteroids.[1] Whether inhaled corticosteroids (ICS) significantly affect bone density is not clear. ICS are clearly beneficial in patients with moderate to severe asthma. They are absorbed to some extent and although systemic effects are less than with oral steroids, potential long-term adverse effects have to be considered when considering risks and benefits of their use in mild asthma.

Tattersfield and associates performed a prospective, randomized, open trial in 19 centers in France, New Zealand, and the United Kingdom. Patients with mild asthma were randomized to receive either inhaled budesonide at a median daily dose of 389 mcg, inhaled beclomethasone dipropionate at a median daily dose of 499 mcg, or noncorticosteroid (nonICS) treatment for 2 years. After initial assessment and screening, subjects were seen in the clinic every 4 weeks for the first 3 months and then every 3 months.

There were no significant differences in the change in the mean bone mineral density between the 3 groups. There was no difference in markers of bone metabolism between budesonide and the nonICS group. However, the beclomethasone group had lower osteocalcin levels than the nonICS group (104 vs 141; $P < .05$). The beclomethasone group also had higher urinary deoxypyridinoline levels than the budesonide group (105 vs 92) and higher urinary pyridinoline levels than the budesonide group (101 vs 90; $P < .05$). In subjects taking ICS, the mean dose of ICS correlated with the fall in bone mineral density over 2 years at the lumbar spine ($P < .01$) but not at the femoral neck. To investigate whether asthma severity might be contributing to bone mineral density by requiring more doses of corticosteroids, FEV_1 was added to the prediction model. Little change, however, was observed in the regression coefficient when FEV_1 was added as compared to without

FEV_1 (-0.80 vs -0.82; *P* = .016) and the findings remained statistically significant. Asthma control was better in patients treated with ICS as determined by reduction in day-time and night-time symptoms and in rescue bronchodilator use. The increase in morning peak flow was more rapid and more marked in subjects receiving ICS, with a mean increase in morning peak flow of 48, 36, and 20 L/min in the budesonide group, beclomethasone group, and the nonICS group, respectively. The change in FEV_1 followed a similar pattern with an early increase in the 2 groups receiving ICS.

COMMENT BY DAVID OST, MD, & AAMIR AWAN, MD

ICS are clearly beneficial in patients with moderate to severe asthma. The extent to which the ICS affect bone metabolism and bone density is less clear since the few prospective studies in asthma patients have been small and most cross-sectional studies have not controlled adequately for the prior use of oral corticosteroids.[2,3] In the current study, participants had mild asthma, were taking beta-2 agonists only, and had not used corticosteroid treatment by any route during the previous 3 months.

Although no significant differences were observed in bone mineral density in the 3 groups, there was a relationship between the dose of ICS and the fall in bone mineral density over 2 years at the lumbar spine but not at the femoral neck. This could be explained by a direct effect of corticosteroids on bone or an indirect effect due to asthma severity since patients with lower peak flow and FEV_1 might have been taking higher doses of inhaled corticosteroids. However, when adjusted for FEV_1, there was no significant change in the relationship between ICS and bone mineral density, with a regression coefficient without adjustment for FEV_1 of -0.82 and after adjusting for FEV_1 of -0.80 (*P* = .016). Asthma was better controlled in the ICS group, with patients in the nonICS group taking twice as many courses of oral corticosteroids than patients in the ICS group.

On analyzing the data, some caution is needed in interpreting the results. Bone mineral density can be influenced by many factors like menopause and change in weight and smoking, although these did not differ between the 3 groups at baseline. More patients in the nonICS group discontinued the study and this may have introduced bias. These findings would also be influenced by the type of treatment selected for the nonICS group. ❖

Dr. Awan is a Fellow in Pulmonary and Critical Care Medicine, North Shore University Hospital, Manhasset, NY.

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Practice Parameter: Bell's Palsy

ABSTRACT & COMMENTARY

Synopsis: *Bell's palsy remains a disease in search of a proven effective therapy.*

Source: Grogan PM, Gronseth GS. *Neurology.* 2001;56: 830-836.

Various treatment options exist for bell's palsy, some undeniably useful, some unequivocally pointless. Artificial tears, lubricating ophthalmic ointment, and eyelid taping prevent corneal drying. Massage and facial nerve electrical stimulation provide psychological support, but little else. Within this spectrum, wither steroids, acyclovir, and facial nerve surgical decompression?

A special article by the Quality Standards Subcommittee of the American Academy of Neurology addresses this question. A MEDLINE search of the National Library of Medicine's database from 1966 to June 2000, and review of the references of these articles to identify other relevant reports on Bell's palsy, uncovered 230 articles examining steroids (only 9 were prospective), 92 addressing acyclovir (3 prospective), and 104 discussing surgical decompression (4 prospective). None were adequately powered class I studies, defined as a randomized, controlled trial with 1) clearly defined primary outcomes and exclusion and inclusion criteria; 2) equivalent baseline characteristics among treatment arms; and 3) satisfactory accounting of dropouts and crossovers. Results of class I and II (3 of 4 above criteria) were pooled where possible.

No definite benefit could be established for steroids, acyclovir, or surgical decompression. Probable benefit from steroids, with acyclovir possibly effective when combined with prednisone, was suggested by the available evidence. No recommendation could be made regarding surgical decompression. Bell's palsy remains a disease in search of a proven effective therapy.

COMMENT BY MICHAEL RUBIN, MD

Herpes simplex virus (HSV) type 1 is reportedly the major cause of Bell's palsy,[1] but HSV type 6 is also a com-

mon culprit. Using polymerase chain reaction (PCR), type 6 HSV DNA was detected in the tear fluid of 35% of patients (7 of 20) with Bell's palsy.[2] Varicella zoster virus (VZV) reactivation (Ramsay Hunt syndrome), which may appear without skin lesions and mimic Bell's palsy, was found in 10% (2 of 20). VZV is more resistant to acyclovir, and may be responsible for some treatment failures. If suspected, higher doses of acyclovir are recommended.

Transmastoid decompression may benefit severe Bell's palsy. Among 101 adults with significant denervation following prednisone therapy for Bell's palsy, defined as > 95% amplitude drop in compound muscle action potential on facial motor nerve stimulation, 58 underwent decompression and 43 were followed conservatively. Two months following surgery, the operated group demonstrated a significantly better House-Brackmann grade than the nonsurgical group.[3] Further studies are warranted, however, before recommendation of this procedure is justified. ❖

Dr. Rubin is Associate Professor of Clinical Neurology, New York Presbyterian Hospital-Cornell Campus, New York, NY.

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Pharmacology Update

Yasmin—A Novel New Oral Contraceptive

By William T. Elliott, MD, FACP, and James Chan, PharmD, PhD

The fda has approved berlix's "yasmin," a unique new monophasic oral contraceptive. The product contains ethinyl estradiol and drospirenone—a new progestogen. Drospirenone is an analog of spironolactone, an aldosterone antagonist, and like spironolactone, drospirenone has antimineralocorticoid activity. As such, its antiandrogenic and antimineralocorticoid actions are closer to progesterone than to other progestogens such as desogestrel or levonorgestrel. Drospirenone is also being touted as a more "natural" progestogen.

Indications

Ethinyl estradiol/drospirenone (DRSP/EE) is indicated for the prevention of pregnancy in women who elect to use an oral contraceptive.[1]

Dosage

DRSP/EE is taken once daily. It may be started the first day of the menstrual period or on the first Sunday after the onset of the menstrual period. Each blister pack contains 21 active tablets and 7 inert tablets. Each active tablet contains 30 mcg of ethinyl estradiol and 3 mg of drospirenone.

Potential Advantages

In addition to its antiandrogenic activity which reduces symptoms such as acne, seborrhea, and hirsutism, drospirenone also has antimineralocorticoid activity that reduces ethinyl estradiol-induced sodium and water retention.[2]

Potential Disadvantages

Drospirenone in Yasmin is comparable in its antimineralocorticoid activity to 25 mg of spironolactone.[1] It has the potential to cause hyperkalemia and should not be used in patients at risk for this condition. These include patients with renal insufficiency, adrenal insufficiency, and hepatic dysfunction. DRSP/EE must be used with caution in patients taking medication that may cause elevation of serum potassium (eg, angiotensin converting enzyme inhibitors, angiotensin II receptor antagonists, potassium sparing diuretics, and NSAIDs). Drospirenone increases the concentration of plasmin renin activity and plasma aldosterone.[3]

Comments

DRSP/EE has been shown to be an effective contraceptive with good cyclic control compared to ethinyl estradiol/desogestrel.[4] Drospirenone is an analog of spironolactone and its pharmacologic profile closely resembles that of progesterone.[2]

DRSP/EE is an oral contraceptive that may slightly reduce body weight and blood pressure and improve pre-existing acne and seborrhea.[2-4] In addition, it does not adversely affect lipid or carbohydrate metabolism.

DRSP/EE costs about $27 per cycle.

Clinical Implications

Yasmin is a new monophasic oral contraceptive with unique properties. Since weight gain has been cited as a concern in patients who discontinue the use of oral contraceptives,[5] DRSP/EE provides an alternative to other oral contraceptives in patients with a tendency to gain weight due to water retention. In addition, DRSP/EE is an alternative to the use of a progestogen with a low androgenic potential (eg, norgestimate or low-dose norethindrone) in patients with acne. ❖

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CME Questions

3. *Helicobacter pylori* has which one of the following effects on gastroesophageal reflux disease (GERD)?
   a. Invariably worsens existing GERD
   b. Invariably moderates existing GERD
   c. Eradication of *H pylori* may lead to emergence of previously unrecognized GERD
   d. May lead to any or all of the above results

4. In patients with primary GERD symptoms, the prudent physician will:
   a. search for and eradicate *H pylori*.
   b. always follow a laissez faire or "don't ask, don't tell" policy.
   c. decide on management based on *H pylori* subtype and on the distribution of gastritis present.
   d. admit that the appropriate course of action is still unknown.
   e. consider inoculation of the patient with a nonpathogenic strain of *H pylori*.

5. Which one of the following statements is *incorrect*?
   a. The change in HDL cholesterol was more favorable with pioglitazone than with rosiglitazone.
   b. The increase in triglycerides that occurred is probably not important.
   c. Hepatic dysfunction was not a problem with either pioglitazone or rosiglitazone.
   d. The rise in triglycerides and the fall in the HDL lipoproteins in the rosiglitazone group is consistent with a change to small dense LDL cholesterol, which is more athergenic.

6. All of the following can affect bone mineral density *except*:
   a. menopause.
   b. cigarette smoking.
   c. change in weight.
   d. steroid use.
   e. asthma severity.

7. For the treatment of Bell's palsy:
   a. prednisone is of proven benefit.
   b. acyclovir is of proven benefit.
   c. combining prednisone with acyclovir is of proven benefit, moreso than individually.
   d. surgical decompression is of proven benefit.
   e. prednisone and acyclovir may help, but controlled trials are still needed to prove this conclusively.

By Louis Kuritzky, MD

The Canadian CT Head Rule for Patients with Minor Head Injury

Minor head injury (mhi), defined as injuries associated with loss of consciousness, amnesia, or disorientation, and a Glasgow Coma Scale (GCS) score of 13-15, are an important public health concern, since as many as 1 million such cases are reported annually in North America. CT scans used in the course of evaluation for MHI have an extraordinarily low yield of pathology (0.7-3.7%), and, hence, represent a substantial financial burden.

In this prospective study, Stiell and colleagues developed a CT head decision rule based upon experience involving adult patients (n = 3121) with MHI and GCS scores of 13-15. CT was deemed merited only in persons who had positive responses to screening of high-risk and medium-risk criteria.

High-risk (for likelihood of neurologic intervention) criteria were: GCS score < 15 at 2 hours after injury, suspected open or depressed skull fracture, signs of basal skull fracture, > 2 vomiting episodes, or age > 65; medium-risk (for brain injury detected on CT) screening criteria were: amnesia before the impact lasting > 30 minutes, or what is described as a "dangerous mechanism" of injury, such as a pedestrian struck by a car, passenger thrown from a vehicle, or fall from height > 3 feet. Using the CT Head Rule should effectively reduce the ordering of CT scans by 32-54%. ❖

Stiell IG, et al. Lancet. 2001;357: 1391-1396.

Validation of Clinical Classification Schemes for Predicting Stroke: Results from the National Registry of Atrial Fibrillation

Hypertension is responsible for the highest attributable stroke risk, but of individual risk factors, atrial fibrillation (AF) is the most potent. Since antithrombotic therapies (ie, ASA, warfarin) have consistently demonstrated benefit for stroke prevention in AF, but the risk profiles for the 2 therapies are quite different, it is necessary for clinicians to have appropriate stratification schema to provide guidance in how best to apply such treatment.

Gage and colleagues examined data during 2121 patient-years of follow-up for AF patients, during which there were 94 strokes. To stratify patients, they incorporated information from the Atrial Fibrillation Investigators (AFI) and Stroke Prevention and Atrial Fibrillation (SPAF) investigators to form a composite risk classification scheme called CHADS2, which includes a single risk point for each of the following: congestive heart failure, hypertension, age > 75, and diabetes; stroke (or TIA) was assigned 2 points. According to this scoring system, for every point increase in CHADS2, the risk of stroke increases 1.5 fold.

ASA provides less stroke risk reduction than warfarin, but also provides less adverse event risk. Gage et al suggest that for patients with a CHADS2 risk of 0, aspirin would be the clearly preferred treatment. ❖

Gage BF, et al. JAMA. 2001;285: 2864-2870.

High Density Lipoprotein Cholesterol and Ischemic Stroke in the Elderly

The linear relationship between cholesterol and CHD end points has not been clearly established with stroke. On the other hand, use of statins for persons with CAD has demonstrated impressive reductions in stroke, prompting closer scrutiny of the relationship between lipids, especially lipid subfractions, and cerebrovascular end points.

Sacco and colleagues used a population (n = 688) of persons older than age 39 suffering their first cerebral infarction, and compared these individuals on a case-control basis with 905 controls.

Higher levels of high-density lipoprotein (HDL; > 50 mg/dL) cholesterol were associated with a 0.5 odds ratio for stroke. The relationship of HDL to stroke did not change when LDL, triglycerides, ethnicity, gender, or race were factored in through multivariate analysis.

Previous trials have shown that use of statins for stroke prevention is of greater benefit for those with lower baseline HDL levels. Another recent trial using gemfibrozil in patients with isolated low HDL (LDL not elevated) resulted in favorable effect upon stroke and other vascular outcomes associated with improvements in HDL. Sacco et al suggest that greater attention to HDL as a cerebrovascular risk factor, and subsequent modification, may significantly affect the burden of stroke. ❖

Sacco RL, et al. JAMA. 2001;285: 2729-2735.

In Future Issues: Maybe We Should Put Statins in the Water