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INSIDE

Hip Pointers

ABSTRACT & COMMENTARY

Synopsis: *Thiazide use for at least 1 year is associated with a decreased incidence of hip fractures, but that decrease disappears 4 months after discontinuation.*

Source: Schoffs MW, et al. *Ann Intern Med*. 2003;139:476-482.

THE ROTTERDAM STUDY, A PROSPECTIVE, POPULATION-BASED cohort study, began in 1990 with an invitation to all people of Ommoord, The Netherlands, who were 55 years or older to participate in a study of disease and disability in the elderly. There were 10,275 eligible Ommoordians and 7983 signed up. Before the study started, 84 persons died and 8 had hip fractures, leaving 7891 (3071 men and 4820 women). Their medical histories, physical examinations, laboratories, and demographics were recorded initially and at follow-up visits. The researchers had access to all of their prescription data, including which drugs were prescribed, in what doses, the number dispensed, and the daily frequency.

Schoffs and colleagues looked at the occurrence of hip fracture (excluding pathologic fractures and fractures in prosthetic hips) from 1991 to 1999 and their relationship to thiazide diuretics and chlorthalidone, which is not strictly a thiazide, but behaves like one.

As might be expected, there were fewer fractures in men (60, 2%) than in women (221, 5%), and persons with hip fractures were older than the group as a whole (78.2 vs 68.9 years). As patients aged, the more likely they were to sustain a fracture (0.7% in the group aged 55-64 vs 11.8% in those 85 years or older).

When use of thiazide diuretics was examined, ever-use decreased the risk of fracture (hazard ratio, 0.94 [95% confidence interval, 0.72-1.24]). Since the confidence interval (CI) includes 0, this is not statistically significant. Current use (without regard to duration of use) was similarly insignificant (HR, 0.71 [CI, 0.47-1.06]. However, when duration of current use was

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factored in, there was a statistically significant inverse relation to risk of hip fracture (HR, 0.99 [CI, 0.97-0.99]). At the top end of duration of use (> 365 days), the HR was 0.46 (CI, 0.21-0.96). The risk returned to baseline 120 days after discontinuing the medication. There was an additive effect among patients with higher calcium intake but no effect when bone mineral density was assessed.

COMMENT BY ALLAN J. WILKE, MD

The thiazide-hip fracture connection has been around for better than a decade now,[1,2] and the association of increased bone mineral density and thiazides goes back even further.[3] We even think we know how it happens: thiazides reduce urinary calcium excretion,[4] and they may have a direct action on osteoclasts.[5] So, do we have a lock on prescribing thiazides in hopes of preventing fractures? No! None of these studies (including the present one) are randomized controlled trials (RCT). They are observational; they did not randomly assign patients to either receive the drug or a placebo. The patients were on thiazides presumably to treat hypertension or congestive heart failure (CHF). There could be something about hypertensives or CHFs that protects them from hip fractures (not likely, but theoretically possible). In fairness, there is a study that randomized hypertensive women to chlorthalidone that demonstrated decreased bone loss,[6] but this is a secondary end point, not the gold standard of fracture prevention. The current study went to great lengths to control for confounding factors, but in the end the best spin you can put on the data is that if you are older than age 55 and taking a thiazide for at least 1 year, your risk of having a hip fracture is about half of what it would be if you weren't. If you stop, your risk goes back to baseline in 4 months.

One of the things that I consider when I'm sizing up articles for review is whether the results will change the way I care for patients. This article does not meet that standard, because, just as JNC-7 recommends,[7] I almost always prescribe a thiazide diuretic when I treat hypertension. That said, I will feel more assured (sort of) that at the same time I am treating hypertension, I may be preventing hip fractures.

Currently, the Food and Drug Administration has approved alendronate (Fosamax®), calcitonin (Miacalcin®), calcium, estrogens, raloxifine (Evista®), risendronate (Actonel®), and teriparatide (Forteo®) for the prevention of osteoporosis. Should the thiazides be added? After all, they are dirt-cheap and have reasonable adverse side effect profiles. However, until there are well done double-blinded, placebo-controlled RCTs that enroll normotensive individuals and that use fracture prevention as the primary end point, we should stifle our desire to do something for our patients just because "it sounds like a good thing." ■

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Left Ventricular Hypertrophy Regression—Losartan vs Atenolol

ABSTRACT & COMMENTARY

Synopsis: *Losartan therapy of hypertensive patients with LVH resulted in greater LVH regression as compared to atenolol independent of blood pressure control and baseline ECG findings.*

Source: Okin PM, et al. *Circulation*. 2003;108:684-690.

Since left ventricular hypertrophy (LVH) has been associated with preclinical atherosclerotic vascular disease and predicts cardiovascular morbidity and mortality, LVH regression has been the Holy Grail of hypertension therapy. Unfortunately, many previous studies have failed to provide clear information on how best to accomplish LVH regression. Meta analyses of available trials have suggested that agents that antagonize the rennin-angiotensin system may be best. Thus, the investigators in the LIFE trial undertook a sophisticated analysis of ECG-LVH regression in this large trial. Over 9000 hypertensive patients were randomized to losartan or atenolol therapy, treated to target blood pressure levels, and followed for up to 5 years. ECGs were done at baseline, 6 months, and then yearly for the duration of the study. Two criteria for ECG-LVH were used: Cornell method—amplitude of the R wave in lead AVL plus the S wave in lead V_3 times the QRS duration; and Sokolow-Lyon method—the S wave amplitude in V_1 plus the R wave in V_5 or V_6. Losartan reduced the Cornell LVH score by -200 vs -69 mm × ms on atenolol ($P < .001$) and the Sokolow-Lyon by -2.5 vs 0.7 mm ($P < .001$). These changes persisted over the follow-up period. There were no subgroup differences; age, gender, ethnicity, and diabetics all showed similar results. Also, the ECG results were adjusted for baseline ECG findings, blood pressure, and diuretic use. In addition, the prevalence of LVH by the Cornell method decreased 21% on losartan vs 13% on atenolol ($P < .001$) and by Sokolow-Lyon by 12 vs 8.5% ($P < .001$). Okin and colleagues concluded that losartan therapy of hypertensive patients with LVH resulted in greater LVH regression as compared to atenolol independent of blood pressure control and baseline ECG findings.

COMMENT BY MICHAEL H. CRAWFORD, MD

The strength of this study is that it is large enough to detect a difference in LVH despite the use of 2 effective antihypertensive agents. It would seem to settle the issue between renin-angiotensin system blockers and beta-blockers, at least in a largely white population with LVH. Also, unlike previous studies, the results were adjusted to baseline ECG findings and blood pressure, as well as diuretic use. These results are consistent with the HOPE trial, where it was found that ramipril as compared to placebo lowered the likelihood of developing LVH or its persistence and raised the likelihood of prevention or regression of LVH, even when adjusted for the larger change in blood pressure on ramipril. Also, these results are consistent with animal data showing that angiotensin II has a potent myocardial hypertrophy effect. What is not known is how losartan would compare to calcium channel blockers. Some studies have shown equivalent efficacy of calcium blockers in LVH regression and more potent antihypertensive effects.

There are some limitations to the study. The results are only applicable to those with moderate-to-severe hypertension and ECG evidence of LVH. Whether similar results would be seen in those with mild hypertension is doubtful. Also, the use of the ECG to diagnose LVH is problematic. In this study, patients were eligible if they met either the Cornell or the Sokolow-Lyon criteria, but the total number of patients who met LVH criteria by each method does not add up to the total number of patients in the study. Okin et al comment that after enrollment some patients no longer met criteria for LVH but were not excluded at that point. This raises the issue of whether some of the LVH regression observed was due to regression to the mean. In addition, the 2 ECG methods are quite different. The Sokolow-Lyon is very sensitive and the Cornell is much more specific. Combining them represents an interesting mix of patients. It would have been interesting to have echocardiograms in a subgroup. Despite these issues, this large trial strongly suggests that the greater reduction in cardiovascular end points on losartan as compared to atenolol observed in LIFE are probably partly due to the superior effect on LVH regression. ■

Dr. Crawford is Professor of Medicine, Associate Chief of Cardiology for Clinical Programs, University of California, San Francisco, Calif.

When Can People With Epilepsy Drive?

ABSTRACT & COMMENTARY

Synopsis: *The rate of seizure-related crashes did not significantly increase in the state of Arizona after the seizure-free interval was reduced from 12 to 3 months.*

Source: Drazkowski JF, et al. *Mayo Clin Proc*. 2003;78: 819-825.

When is it safe to sit behind the wheel if you've just had a seizure? Specific seizure-free intervals vary from state to state. In this time-trend study, an analysis was undertaken to determine if lowering the interval from 12 months to 3, as was done in Arizona, negatively impacted on motor vehicle accident (MVA) frequency. Seizure-related MVAs were defined as such if the patient self-reported a seizure at the time of the accident, was witnessed to have a seizure or was confused at the scene in the absence of head trauma, or had low anti-epileptic drug levels as reported by hospital personnel. Non seizure-related MVAs included cardiac-related crashes due to arrhythmia or myocardial infarction and diabetes-related MVAs as documented by hypoglycemia, altered consciousness, or response to intravenous glucose at the scene. Psychiatric conditions, medication effects, stroke, visual impairment, dementia, and migraine comprised other medically related MVAs. Crash incidence rates were obtained by dividing the number of MVAs by the number of miles driven, as estimated from the Arizona State Motor Vehicle Department annual report.

Approximately 614,000 MVAs were reported during the 6-year study period, 859 of which were medically related. Comparing 1991-1993, when a 12-month seizure-free interval was required, to 1994-1996, when the interval was lowered to 3 months, the number of seizure-related MVAs increased slightly from 125 to 136. Based on incidence rate, however, seizure-related MVAs dropped by 2%, not a significant difference, despite an 8% increase in MVAs overall. Fatality incidence dropped by 64%, but injuries associated with seizure-related MVAs increased 31%. No significant change was evident in mean age of driver (37 vs 38 years of age), percentage of urban accidents, or percentage of single- vs multiple-car MVAs. Arizona's excellent weather patterns and road conditions precluded inclement weather or road surface from playing a role in any crash. MVAs related to other medical conditions similarly showed no significant change. Common sense must always prevail, but limiting driving restriction to 3 months following a seizure appears safe and reasonable.

COMMENT BY MICHAEL RUBIN, MD

This report supports the consensus developed at a symposium held in Quebec City in November 1998, regarding epilepsy and driving.[1] Attendees included an invited international group of neurologists from Canada, the United States, and Europe; Canadian licensing representatives; and delegates from the Canadian Council of Motor Transport, the Canadian Medical Protective Association, and the Canadian Medical Association. Among the medical experts, abolition of mandatory reporting of epilepsy to the Motor Vehicle Department (as is the law in Canada) was felt to be desirable. Most patients, it was felt, could return to driving private automobiles within 6-12 months of seizure freedom. First and foremost, observance of local laws remains paramount until changes are appropriately legislated. ■

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

Reference

1. Remillard GM, et al. *Can J Neurol Sci*. 2002;29: 315-325.

Oral Contraceptive Mortality

ABSTRACT & COMMENTARY

Synopsis: *There was no harmful effect of oral contraceptive use on overall mortality. By contrast, death from all causes was more than twice as high in smokers of 15 or more cigarettes a day as in nonsmokers.*

Source: Vessey M, Painter R, Yeates D. *Lancet*. 2003;362: 185-191.

Vessey, painter, and yeates from the university of Oxford used the prospective cohort of women enrolled in the Oxford Family Planning Association Study to assess mortality in users and nonusers of oral contraceptives. By the end of the year 2000, 889 deaths had occurred in 17,032 women in England and Scotland enrolled in the study. There was no increase in breast cancer associated with oral contraceptives in either smokers or nonsmokers. However, death from cervical cancer was increased with oral contraceptive use (although the confidence intervals were very wide

Table

Death from Ischemic Heart Disease, Stroke, and VTE

Users compared with nonusers	RR, 1.4 (CI, 0.8-2.5)
Heavy smokers for 48 mos	2.4 (0.4-16.3)
For 49-96 mos	4.8 (1.3-26.2)
97 mos or more	2.8 (0.8-15.8)
Hemorrhagic stroke, heavy smoker	5.8 (2.2-16.5)
Venous thromboembolism	no increase

because of small numbers). Mortality from endometrial cancer and ovarian cancer were reduced in the oral contraceptive users, a result consistent with many previous reports. Comparing never users and users, there was an 80% overall reduction in endometrial cancer deaths and a 60% reduction in ovarian cancer deaths (see Table). The risk of deaths from all causes was significantly increased only in smokers (especially with 15 or more cigarettes daily), and the risk increased with increasing age.

COMMENT BY LEON SPEROFF, MD

The conclusions of this prospective cohort study are limited by the small numbers of deaths in the various categories, as indicated by the wide confidence intervals. However, the results are consistent with a very large literature and further strengthened because they are derived from a single cohort of women. It is important to point out that the data are derived largely from the use of products containing 50 mg ethinyl estradiol, a dose that is now considered to be high. For this reason, the overall safety of oral contraceptives in this report is reassuring, and we would expect even better results with modern, low-dose formulations.

This report confirms previous reports (especially the publications from the World Health Organization and the Nurses' Health Study) that the risk of cardiovascular mortality associated with oral contraceptives is confined to smokers. It is very likely that this risk is present only in current users, an observation that could not be documented in the Oxford study because of the design.

The accumulated literature over many years has consistently established an increased risk of venous thrombosis associated with oral contraceptives. The Oxford report identified no deaths from venous thromboembolism that could be attributed to oral contraceptive use. Most deaths from this condition are linked to trauma, surgery, or a major illness.

The experience with oral contraceptives, in my view, emphasizes the importance of good patient screening. The occurrence of arterial thrombosis is essentially limited to older women who smoke or have other cardiovascular risk factors, especially hypertension. Avoiding the use of oral contraceptives in older smokers and hypertensive women requires effective interaction between the patient and a clinician (not necessarily a physician). Providing oral contraceptives over the counter would bypass this vital interaction, and undoubtedly there would be deaths that could have been avoided. ■

Dr. Speroff is Professor of Obstetrics and Gynecology, Oregon Health Sciences University, Portland, Ore.

Pharmacology Update

Rosuvastatin Calcium (Crestor)

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

THE FDA HAS APPROVED ANOTHER HMG-COA REDUCTase inhibitor (statin) for the treatment of elevated cholesterol. Rosuvastatin is a potent new statin that is not extensively metabolized and has low potential for drug interaction. It is licensed from Shionogi in Japan and marketed by AstraZeneca as Crestor.

Indications

Rosuvastatin is indicated as an adjunct to diet to reduce elevated total cholesterol, LDL-cholesterol, ApoB, nonHDL-cholesterol and triglycerides, and to increase HDL-cholesterol in patients with primary hypercholesterolemia (heterozygous familial and nonfamilial) and mixed dyslipidemia (Fredrickson Type IIa and IIb). It is also indicated for patients with elevated triglycerides (Fredrickson Type IV) and patients with homozygous familial hypercholesterolemia as an adjunct to other lipid-lowering treatments to lower LDL-cholesterol, total-cholesterol, and ApoB.[1]

Dosage

The dose range for rosuvastatin is 5-40 mg depending on goal of therapy, concomitant drugs, and patient response. The usual starting dose is 10 mg daily and 20 mg daily for patients with homozygous familial hypercholesterolemia. It may be taken at any time of day and without regard to meals, and no dosage adjustment is needed for patients with mild-to-moderate renal impairment. In patients with severe renal impairment not on hemodialysis, the starting dose is 5 mg daily, and in those taking

cyclosporine, the dose should be limited to 5 mg daily.[1]

Rosuvastatin is available as 5-mg, 10-mg, 20-mg, and 40-mg tablets.

Potential Advantages

Rosuvastatin is not extensively metabolized by the cytochrome P450 enzyme system, and, as a result, drug interactions associated with other statins are not associated with rosuvastatin.[1] Rosuvastatin appears to be the most potent statin on a milligram basis. In a 12-week, randomized, double-blind, placebo-controlled, comparative study, rosuvastatin 5 mg and 10 mg produced a greater reduction in LDL-C than atorvastatin 10 mg.[2] LDL-C reductions were 40% and 43% for rosuvastatin 5 mg (n = 128) and 10 mg (n = 129), respectively, compared to 35% for atorvastatin 10 mg (n = 127). These were significant at $P < 0.01$ and $P < 0.001$, respectively. At those doses, 42% and 47% of patients on rosuvastatin met NCEP ATP III goal compared to 19% for atorvastatin.

Potential Disadvantages

Higher doses of rosuvastatin (ie, 40 mg and 80 mg) appeared to be associated with a higher frequency of proteinuria of 2+ or higher compared to the same dose of atorvastatin.[5] The frequencies were 2.8% and 11%, respectively, compared to 0.4% and 0.3% for atorvastatin 40 mg and 80 mg. There is limited clinical experience with rosuvastatin. While it appears to be well tolerated, its safety profile needs to be confirmed with further clinical studies and postmarketing surveillance.

Comments

Rosuvastatin is a potent, hydrophilic HMG-CoA reductase inhibitor (statin) with a low potential for drug interactions and a long elimination half-life (about 19 hours). In patients with hypercholesterolemia (heterozygous familial and nonfamilial) and mixed dyslipidemia (Fredrickson Type IIa and IIb), rosuvastatin 10 mg reduced LDL-C by about 50%, total cholesterol by about 36%, and triglycerides by about 10% at 6 weeks.[1-3] In comparative studies, rosuvastatin is more potent than atorvastatin and simvastatin on a mg-for-mg basis.[1,2,4] The drug appears to be well tolerated and the overall incidence of adverse events were similar to other statins with the possible difference of proteinuria.[4] The average wholesale cost of rosuvastatin is $2.63 per tablets for all strengths. This is 12% less than the average weighted cost for atorvastatin.[6]

Clinical Implications

Statins are the firstline pharmacological management of hypercholesterolemia. Rosuvastatin offers a potent new agent in this class with a low risk for drug interactions. Due to rosuvastatin's potency, a higher percent of patient may achieve NCEP LDL-C goals compared to currently available statins. ■

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CME Questions

16. Thiazide diuretics:

a. increase bone mass by enhancing osteoblast activity.
b. cause increased urinary excretion of calcium.
c. are associated with a reduction in hip fractures that persists for at least a year after discontinuation.
d. lose their ability to prevent hip fractures after 120 days of use.
e. None of the above

17. As compared to atenolol, losartan for hypertension:

a. reduces cardiovascular events.
b. regresses LVH.
c. improves blood pressure control.
d. a and b

18. The following statements are true of oral contraceptives *except*:

a. The cardiovascular side effects associated with oral contraceptives are related by the dose of the estrogen component.
b. The side effect rate is affected by the number of cigarettes smoked daily.
c. The major serious side effect is venous thrombosis.
d. Good patient screening can virtually eliminate serious side effects associated with oral contraceptives.

Answers: 16 (e); 17 (d); 18 (c)

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By Louis Kuritzky, MD

Ultralow-Dose Estrogen and Bone in Older Women

Hormone replacement therapy (HRT), has been found to have favorable effects upon osteoporosis and fracture risk in postmenopausal women. Unfortunately, this bone benefit is at the expense of increased risk for breast cancer, heart disease, stroke, and DVT. The increased risks are generally acknowledged to outweigh benefits for most women, though the jury is still out on estrogen replacement (ERT) without progesterone (in hysterectomized women).

In an effort to reduce risk, decrements in estrogen dose have been evaluated, usually in combination with supplements of calcium and vitamin D. This randomized, double-blind placebo controlled trial evaluated the provision of 0.25 mg/d of 17-beta estradiol (17-ERT), which is one-fourth to one-half the "conventional" dose previously used, daily for 4 years. End points included bone mineral density (BMD) and markers of bone turnover. Women were all menopausal, and in those women who had not undergone hysterectomy, 100 mg/d micronized progesterone was given for 2 weeks every 6 months. All study subjects were given supplemental vitamin D (1000 IU/d) and calcium (1300 mg/d).

In participants who received this low-dose estrogen, BMD by DEXA scanning showed favorable effects at the femoral neck, hip, spine, and total body. Bone turnover markers were also favorably affected.

At this low dose, the adverse effect profile was essentially indistinguishable from placebo, including breast tenderness. It is encouraging to note that ultralow-dose estrogen has favorable bone effects. Ultimately, it will be essential to ascertain whether the BMD changes found will be reflected in fracture risk reduction. Additionally, though lower estrogen dose might be anticipated to reduce risk of serious adverse events, this remains to be determined. ■

Prestwood KM, et al. JAMA. 2003;290:1042-1048.

Effect of Intensity of Oral Anticoagulation in Atrial Fibrillation

The value of warfarin anticoagulation (WAC) in atrial fibrillation (AF) to prevent ischemic stroke is well established. Despite therapeutic levels of WAC, however, some AF patients still suffer ischemic stroke. In persons who do suffer stroke while on WAC, it is unclear whether their stroke severity is related to degree of anticoagulation. To clarify that question, this investigation studied acute ischemic stroke (n = 596) among persons with nonvalvular AF who were being treated at the time of stroke with WAC (32%), aspirin (27%), or were on no prophylactic treatment. In patients on WAC, stroke severity was assessed in relation to INR, comparing those with an INR > 2 to patients having an INR < 2.

For the end point of mortality or discharge with severe stroke, there was a dramatic disadvantage demonstrated for stroke patients with an INR < 2 (15% vs 5%). The relative hazard for death within 30 days for patients with an INR < 2 was increased over 3-fold.

Stroke that occurs while on WAC in AF is less severe, and has more favorable mortality outcome, when the INR is maintained at a level of > 2. Since increased risk of intracranial hemorrhage was not seen until INR levels rose to > 3.9, maintenance of the traditionally accepted INR 2-3 range appears to maximize benefit, and minimize risk. ■

Hylek EM, et al. N Engl J Med. 2003;349:1019-1026.

Patient Knowledge and Awareness of Hypertension

Despite a diversity of excellent pharmacotherapeutic tools for treating hypertension (HTN), national population surveys continue to indicate that there is much room for improvement in HTN detection, awareness, and control. Of course, if patients are unaware of BP goals, or their own BP and its adequacy of control, there is substantially less likelihood that they will achieve all the potential benefits of antihypertensive treatment.

Based upon a recent survey of hypertensive patients in the Northern California Kaiser Permanente Medical Care system (n = 2500), there remains a great deal of room for improvement in patients' knowledge about blood pressure. Among this population, almost 80% of persons with BP > 140/90 did not recognize their BP as "high," although 38.5% identified this level of BP as "borderline high;" a similar number of individuals were not able to recall their BP levels taken at the most recent clinic visit. Perhaps most distressing is that the majority of patients neither knew a goal for their BP treatment, nor was able to appropriately identify whether systolic or diastolic BP levels were a greater risk factor.

Encouragingly, more than 85% of patients recognized that HTN increased risk for stroke and MI, but only half as many individuals knew that HTN might increase risk of kidney disease. The message that patients need to know their BP, BP goals, and the greater relative risk of elevated systolic than diastolic blood pressure will have to be given greater attention by clinicians and other patient educators. ■

Alexander M, et al. J Clin Hypertens. 2003;5:254-260.

Typical LBBB? LVH? Acute MI?

By Ken Grauer, MD

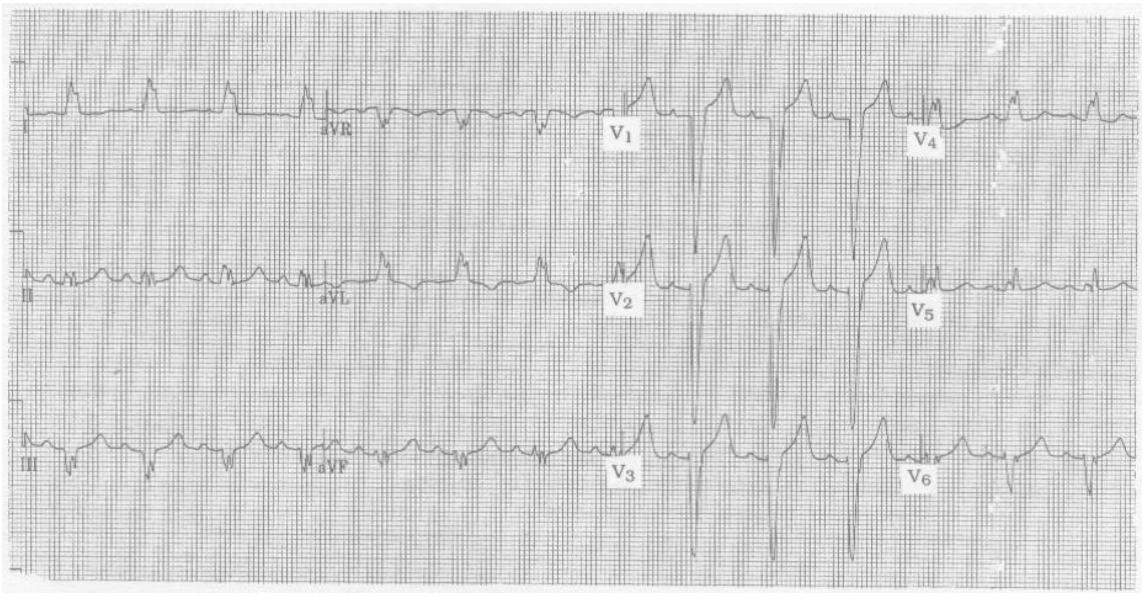

Figure. 12-lead ECG obtained from a 63-year-old woman with a history of hypertension, heart failure, and atypical chest pain.

Clinical Scenario: The ECG in the Figure was obtained from a 63-year-old woman with a history of hypertension, heart failure, and atypical chest pain. The ECG shows normal sinus rhythm at a rate of 85 beats/minute. The QRS complex is widened. Would you say there is typical LBBB (left bundle branch block)? Would you interpret this tracing as suggestive of LVH (left ventricular hypertrophy)? of acute MI (myocardial infarction)?

Interpretation: The first point to make about this 12-lead ECG relates to the bizarre progression of QRS morphology in the precordial leads. It makes no anatomic (or physiologic) sense for the QRS complex to alternate from near total negativity (in leads V_1, V_2, V_3)—to total positivity (in leads V_4, V_5)—and then abruptly back to near total negativity in lead V_6. Instead, we strongly suspect misplacement of several precordial leads. Most likely the QRS complex seen in lead V_6 should really appear in lead V_4 — and the complex in lead V_4 should appear in lead V_6. Were this the case, then this patient would manifest the typical pattern of complete LBBB (predominantly negative QRS in lead V_1; monophasic R wave with or without a notch in leads I and V_6). A repeat ECG is of course needed to verify our suspicion.

The diagnosis of LVH cannot be made by the usual criteria in the presence of complete LBBB. This is because the conduction defect dramatically alters the usual sequence (and therefore QRS morphology) of ventricular activation. However, several relevant points relating to LBBB can still be made. First, most patients with complete LBBB have underlying heart disease. Simply the presence of LBBB identifies a high prevalence group of individuals who are statistically likely to have heart disease predisposing to ventricular hypertrophy (note the history of the 63-year-old woman in this case). In the presence of underlying heart disease and complete LBBB, the ECG finding of very deep S waves (of more than 25-30 mm) in leads V_1, V_2, V_3 makes it highly likely that the patient also has LVH. On the other hand, nothing can be said about the presence or absence of myocardial infarction (old or acute) from interpretation of the typical LBBB pattern seen here. ■

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