an important point. There is considerable data to suggest that physicians do not titrate statin doses upward after the initial prescription, a likely contributor to the "treatment gap" between cholesterol-lowering guidelines and actual practice.

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RIGHT HEART FAILURE AND HYPERTHYROIDISM: A NEGLECTED PRESENTATION

To the Editor:

Heart failure is a rare manifestation of thyrotoxicosis in previously healthy patients (1). Predominantly right-sided heart failure in hyperthyroidism is even more uncommon. We describe 2 such patients and discuss the mechanism.

The first patient was a previously healthy 47-year-old woman who presented with shortness of breath and leg swelling. She was tachypneic and mildly tremulous, with a temperature of 38.5°C, an irregular heart rate of 136 beats per minute, and blood pressure of 127/70 mm Hg. Jugular venous distension, moderate ascites, and 4+ pedal edema were prominent. Thyroid gland was diffusely enlarged, without lid lag or exophthalmos. Cardiac examination revealed an apical 2/6 systolic murmur, no third heart sounds, and bibasilar rales. Electrocardiography showed atrial fibrillation. A chest radiograph showed mild pulmonary congestion and cardiomegaly. Laboratory results included a thyrotropin level <0.005 μIU/mL, a free thyroxine level of 2.5 ng/mL, and a total triiodothyronine level of 432 ng/dL. Uptake of I_{131} was 42% (2 hours) and 62% (24 hours). Workup for pulmonary emboli or infectious diseases was negative, pulmonary function tests were normal, and autoantibodies were not found. Echocardiography revealed normal left ventricular (LV) function, mild mitral regurgitation, and pulmonary hypertension (45 mm Hg) with tricuspid regurgitation. Symptoms resolved rapidly with furosemide, propranolol, and propylthiouracil, and the patient was discharged. However, noncompliance with the medications resulted in relapse of thyrotoxicosis and right heart failure, which resolved with reinstitution of therapy. The patient remains well.

The second patient was a 42-year-old woman admitted for anasarca that developed over several weeks. Ten years ago, she was diagnosed with Graves' disease and treated with propylthiouracil and propranolol. However, she remained in atrial fibrillation and dependent on both drugs. Radioactive iodine was refused. Two months before admission, she stopped taking her medications. Shortly afterward she had progressive swelling of the legs and abdomen, and weight gain. She was afebrile, with a blood pressure of 170/100 mm Hg and an irregular heart rate of 150

Table. Characteristics of Patients with Graves' Disease and Right-Sided Heart Failure

Patient Age/Sex	Clinical Presentation	Pretreatment PAP* (PVR†)	Outcome	Post-treatment PAP* (PVR†)	Reference
47/F	Severe right heart failure	45 mm Hg	Rapid resolution	Not done	Current report
42/F	Anasarca	60 mm Hg	Complete resolution	22 mm Hg	Current report
47/M	Anasarca	45/18 mm Hg (78 dyne · sec · cm^{-5})	Rapid resolution	Not done	3
62/F	Anasarca	Not done	Tricuspid regurgitation resolved	Not done	4
59/F	Anasarca	50 mm Hg	Complete resolution of right heart failure	Not done	4
32/M	Edema, severe tricuspid regurgitation	27 mm Hg	Edema and tricuspid regurgitation resolved	Not done	5
54/M	Ankle edema, distended neck veins	56 mm Hg	Complete resolution	35 mm Hg	6
46/F	Peripheral edema	53 mm Hg (256 dyne · sec · cm^{-5})	Complete resolution	15 mm Hg (80 dyne · sec · cm^{-5})	7

* Systolic PAP obtained by transthoracic echocardiogram except in 2 patients (references 3 and 7) in whom systolic and diastolic pulmonary pressures were obtained by cardiac catheterization.
† PVR obtained by cardiac catheterization.
F = female; M = male; PAP = pulmonary artery pressure; PVR = pulmonary vascular resistance.

beats per minute. She had marked jugular venous distension, a diffusely enlarged thyroid gland, and no lid lag or exophthalmos. Lung bases were dull to percussion with decreased breath sounds. A 2/6 holosystolic murmur was heard at the apex with no third heart sounds. The abdomen was distended with massive ascites that yielded a transudative fluid. Laboratory studies revealed thyrotoxicosis (serum thyrotropin <0.01 μIU/mL).

Electrocardiography showed atrial fibrillation. Chest radiographs showed cardiomegaly and bilateral moderate pleural effusions. Ventilation-perfusion lung scanning was normal. Chest computed tomography and echocardiography showed normal pericardium and severe pulmonary hypertension (60 mm Hg), with marked dilatation of the right atrium and ventricle, and moderate mitral and tricuspid regurgitation. There was paradoxical motion of the septum and normal LV function. Cardiac catheterization showed normal coronary arteries and confirmed the echocardiographic findings (wedge pressure, 23.2 mm Hg; right ventricular pressure, 56/18 mm Hg). Rapid volume expansion by normal saline infusion ruled out constriction. After treatment (furosemide, propranolol, propylthiouracil), she had a rapid diuresis and the anasarca resolved. She remains euthyroid and well 4 years after presentation.

Because of the positive effects of triiodothyronine on cardiac function (2), overt heart failure in thyrotoxicosis is unexpected (1). Occasionally, patients develop primarily systolic heart failure because of the toxic effects of thyroid hormones on the myocardium or chronic tachycardia (1). In contrast, predominantly right-sided heart failure in thyrotoxic patients with preserved left ventricular function is rare (Table). It can be due to myocardial dysfunction of the right ventricle or to increased afterload. Review of the literature supports the second alternative.

Pulmonary artery pressure in these patients (including our patients) decreased following resolution of the hyperthyroid state (Table). The normalization of pulmonary artery pressure and resolution of all signs of heart failure with treatment of the thyrotoxicosis support a causal association. Hyperthyroid rats have been observed to develop ventricular hypertrophy affecting predominantly the right ventricle (8). A study of 4 thyrotoxic patients revealed that all patients had clinically "silent" pulmonary hypertension that decreased about 35% when they were euthyroid (6). Another study reported mild pulmonary hypertension (29 ± 6 mm Hg) in 35% of patients with recently diagnosed hyperthyroidism (9). Pulmonary artery pressure in these patients correlated with serum thyrotropin and free thyroxine levels ($r = 0.8$; $P < 0.001$), but was normal in the euthyroid control group. The mechanism, however, remains unclear (10). Nonetheless, hyperthyroidism should be considered in the differential diagnosis of right-sided heart failure with pulmonary hypertension.

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PHIALOPHORA RICHARDSIAE BURSITIS TREATED MEDICALLY

To the Editor:

Although *Staphylococcus aureus* is responsible for most cases of septic bursitis, fungi are rarely isolated (1). *Phialophora richardsiae*, a dematiaceous fungi, was first reported as a cause of human infection in 1968 (2). There has been one case of septic bursitis caused by *P. richardsiae* (3). The dearth of information on antifungal susceptibility for *P. richardsiae,* and the favorable response to excision, have made surgery the treatment of choice (4). We report a case of septic bursitis with contiguous cellulitis caused by *P. richardsiae* that was treated successfully with systemic antifungal agents and irrigation of the bursa.

A healthy 77-year-old man presented with chronic swelling below the right knee. On examination, the infrapatellar bursa was markedly enlarged without warmth or erythema. Microscopic examination of fluid aspirated from the bursa revealed abundant neutrophils. Gram's stain and bacterial culture were negative. A presumptive diagnosis of "Carpenter's knee" was made. Methylprednisolone was instilled into the bursa to reduce inflammation.

The infrapatellar bursa subsequently diminished in size; however, 2 months later the patient presented