

(figure). Capillary loops were not thickened and no trichrome positive deposits were seen. The tubules, interstitium, and blood vessels were all normal. Immunofluorescence studies were negative for all immunoglobulins, fibrin, and  $\beta$ -1C. Electron microscopy showed diffuse foot process fusion of the podocytes (see figure) with no electron dense deposits. The basement membrane was normal.

He received corticosteroids, and his oedema decreased dramatically in two weeks. He again defaulted follow up.

### Comment

Our patient clearly had mild decompression sickness with substernal pain, dizziness, headache—usual symptoms of the decompression syndrome<sup>1</sup>—and minimal change glomerulonephritis. Any association between the two conditions is difficult to ascertain. Rapid ascent to the surface after diving causes decompression sickness because inert gases, mainly nitrogen, which are dissolved in the blood and tissues, come out of solution and form bubbles that produce embolism. The protean manifestations of decompression sickness cannot be completely explained by the physical effects of the bubbles. Interface of blood and bubbles causes protein denaturation, increases platelet adhesiveness, produces red cell sludging in the microcirculation, and promotes the formation of lipid emboli.<sup>1</sup> Localised oedema occurs in 9% of patients with acute decompression sickness,<sup>2</sup> although we know of no reports of generalised oedema. Minimal change glomerulonephritis might be the result of rapid change in environmental pressure, especially if the glomerular basement membrane is regarded as a thixotropic gel.<sup>3</sup> The occurrence of the nephrotic syndrome with decompression sickness might have been coincidental. The change in environmental pressure on rapid ascent after diving, therefore, probably accelerates and aggravates oedema, perhaps the reverse of what occurs in up to the neck immersion in water.<sup>4</sup>

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## Heart failure and breast enlargement suggesting cancer

Unilateral or asymmetric pleural effusions are a well recognised clinical finding in moderate cardiac failure.<sup>1</sup> We should like to add breast enlargement mimicking malignancy to the unilateral manifestations of congestive cardiac failure.

### Case reports

#### CASE 1

An 81 year old woman presented with a three week history of general malaise with swelling of the right arm and right breast and increasing abdominal girth. Nine months previously she had been admitted with episodic dyspnoea. Clinically she had had right basal consolidation and mild congestive cardiac failure. Chest radiography had suggested pulmonary infarction. She was not given anticoagulants but responded to antibiotics and diuretics. At outpatient follow up at eight months she was well.

At her second presentation she was short of breath at rest and had massive pitting oedema to the sacrum, over the right flank, and over the whole of the right arm. The right breast was enlarged and also showed pitting oedema (figure). There was tender hepatomegaly and bilateral pleural effusions. Clinical findings suggested congestive cardiac failure and an underlying malignancy of the right breast or chest wall. Chest radiography showed cardiomegaly and bilateral pleural effusions, the right greater than the left. Pleural aspiration yielded yellow transudate (protein concentration 21 g/l) containing scanty inflammatory cells and no malignant cells. Mammography and xerography showed no evidence of

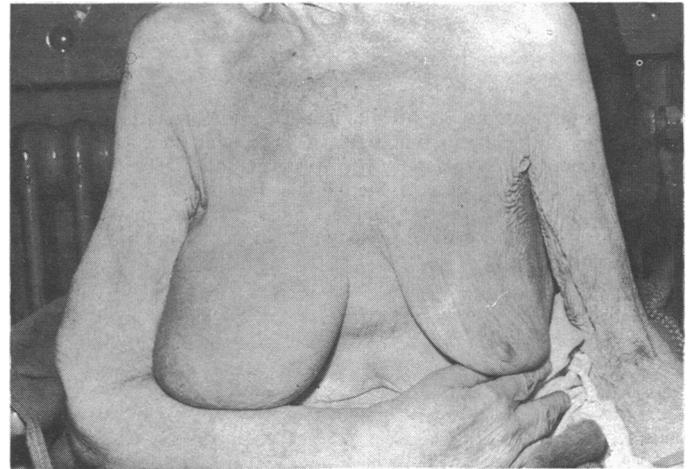
malignancy. Phlebography of the right arm showed patency of all veins including the superior vena cava.

Treatment with fluid restriction, frusemide, and ethacrynic acid produced satisfactory diuresis with resolution of all symptoms and 6 kg weight loss.

#### CASE 2

A 77 year old woman was admitted with a recurrence of heart failure. She complained of exertional dyspnoea, ankle swelling, and heaviness of the right breast. She had a 13 year history of hypertension. In the preceding six months she had been treated as an inpatient for congestive cardiac failure. She was taking captopril 25 mg thrice daily.

On examination she had the signs of severe right ventricular failure. The right breast was enlarged, the nipple was inverted, and the skin around the nipple had a peau d'orange appearance with pitting oedema. No definite mass was felt.



Enlarged right breast due to cardiac failure.

Carcinoma of the breast and congestive cardiac failure were provisionally diagnosed. Chest radiography showed cardiomegaly with bilateral pleural effusions.

Treatment with frusemide and captopril improved all her symptoms, with 7 kg weight loss. At six months' follow up the breast remained normal.

### Comment

We suggest that unilateral breast enlargement mimicking breast cancer is a feature of congestive cardiac failure and should be considered in the differential diagnosis of breast disease in patients with a history of cardiac disease. Both patients presented with breast enlargement as a major symptom and had a history of uncontrolled congestive cardiac failure. Both had other clinical features to support a preliminary diagnosis of breast malignancy—namely, unilateral swelling of the arm, nipple retraction, or peau d'orange. Both responded to standard management of cardiac failure with complete resolution of the breast changes.

Unilateral breast enlargement might be due to the patient's tendency to lie on one side, which could lead to dependent oedema. The atrophic breast of elderly women may be more susceptible than younger tissue. Hoeffken and Lanyi described bilateral breast oedema in severe anasarca.<sup>2</sup> Muller and Koehler reported a case of the nephrotic syndrome with unilateral breast enlargement associated with generalised oedema of the arms and legs and a case in which a mass in an enlarged breast resolved with treatment of cardiac failure.<sup>3</sup>

We thank Dr G Beynon for encouraging us to report these cases.

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2 Hoeffken W, Lanyi M. *Mammography*. Stuttgart: Thieme, 1977:300-5.

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