Table 1 Cardiovascular responses to 10° Trendelenburg and 60° passive leg raising. Data are means ± SD (*LVEDD* left ventricular end-diastolic dimension, *LVESD* left ventricular end-systolic dimension, *LVEDV* left ventricular end-diastolic volume, *LVESV* left ventricular end-systolic volume, *SV* stroke volume, *HR* heart rate, *CO* cardiac output)

	Supine (baseline)	10° Trendelenburg		Supine (2nd baseline)	60° Passive leg raising	
		1 min	10 min		1 min	10 min
LVEDD (mm)	51.6 ± 0.3	54.3 ± 0.3*	52.5 ± 0.3	52.1 ± 0.3	54.4 ± 0.3*	52.9 ± 0.3
LVESD (mm)	32.0 ± 0.2	32.0 ± 0.2	31.5 ± 0.2	32.1 ± 0.2	33.0 ± 0.2	32.6 ± 0.3
LVEDV (ml)	127.6 ± 14.9	143.4 ± 16.9*	132.9 ± 15.8	130.4 ± 14.2	144.1 ± 18.4*	134.9 ± 17.8
LVESV (ml)	41.1 ± 6.6	41.3 ± 6.8	39.5 ± 5.1	41.3 ± 7.5	44.3 ± 6.7	43.4 ± 9.2
SV (ml/beat)	86.5 ± 12.6	102.1 ± 14.7*	93.4 ± 14.0*	89.1 ± 12.9	99.9 ± 14.5*	91.5 ± 12.7
HR (beats/min)	64 ± 12	61 ± 14*	61 ± 14*	60 ± 13	59 ± 14	61 ± 14
CO (l/min)	5.4 ± 0.3	6.1 ± 0.5*	5.6 ± 0.6	5.2 ± 0.3	5.7 ± 0.4*	5.5 ± 0.6

* Significant difference from respective baseline values ($p < 0.05$)

angle for 10 min. Hemodynamic measurements were made at 1 and 10 min during each maneuver. Differences within the maneuvers were analyzed with Scheffé's test after a one-way analysis of variance. A comparison between the Trendelenburg position and PLR was performed by a two-way ANOVA for repeated measurements.

The results are summarized in Table 1. There were small but significant decreases in HR, while LVEDV, SV, and CO increased at 1 min in the 10° Trendelenburg position. The increase in LVEDV was associated with the increase in SV and, thus, the increases in CO. These changes, however, were transient and returned toward baseline levels 10 min following tilting. 60° PLR also caused a transient increase in LVEDV, SV, and CO. The relationship between LVEDV and CO was the same as that seen with Trendelenburg. There were no significant differences between the increase in CO by the Trendelenburg position and the PLR (12.3 ± 9.0% vs 10.7 ± 5.8%).

Our study demonstrates that the 10° Trendelenburg position produced the autotransfusion effect to the same degree as does the 60° PLR in normovolemic individuals. However, hypovolemic patients may have a smaller lower extremity blood volume available for central translocation than normovolemic subjects. Thus, the approximately 10% increases in CO seen with these two maneuvers in normovolemic people may not be of clinical significance. However, Taylor and Weil [3] did document an increase in CO using the 10° Trendelenburg in patients with decreased plasma volume. Therefore, in hypovolemia both the Trendelenburg and the PLR may be of benefit, although their effects are transient. Due to previously described adverse effects of the Trendelenburg [4, 5], the PLR may be the treatment of choice when either position is being considered in patients with hypovolemic shock.

References

1. Sahn DJ, DeMaria A, Kisslo J, Weyman A, The Committee on M-mode Standardization of the American Society of Echocardiography (1978) Recommendations regarding quantitation in M-mode echocardiography: results of a survey of echocardiographic measurements. Circulation 58:1072–1083
2. Teichholz LE, Kreulen T, Herman MV, Gorlin R (1976) Problems in echocardiographic–angiographic correlations in the presence or absence of asynergy. Am J Cardiol 37:7–11
3. Taylor J, Weil MH (1967) Failure of the Trendelenburg position to improve circulation during clinical shock. J Am Coll Surg 124:1005–1010
4. Reed JH Jr, Wood EH (1970) Effect of body position on vertical distribution of pulmonary blood flow. J Appl Physiol 28:303–311
5. Shenkin HA, Scheuerman WG, Spitz EB, Groff RA (1949) Effect of change of position upon the cerebral circulation of man. J Appl Physiol 2:317–326

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Intra-alveolar hemorrhage following bipedal lymphography

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Sir: We describe the first documented case of symptomatic intra-alveolar hemorrhage (IAH) following bipedal lymphography.

A 50-year-old man was hospitalized for acute respiratory failure. He had no history of cardiovascular or respiratory disease. A Hodgkin's disease grade II Aa was diagnosed on December 1993. The treatment has been three cycles of chemotherapy with chlormethine, vincristine, procarbazine and methylprednisolone, followed by seven sessions of radiotherapy (month field irradiation). A staging workup performed on January 1995 (abdominal ultrasound and thoracoabdominal scan, bipedal lymphography) was normal. Four days after lymphography, he was admitted to the hospital complaining of two days of increasing weakness, fatigue, progressive dyspnea, a nonproductive cough , and fever (39 °C). On admission his temperature was 37 °C, and he was in moderate respiratory distress. The heart rate was 85/min, blood pressure 120/70 mmHg, and respiratory rate 26/min. Chest examination revealed diffuse crackles without wheezes. Laboratory studies showed the following: white blood

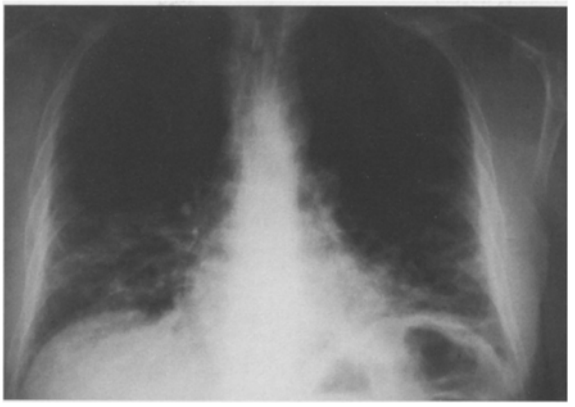

Fig. 1 Chest X-ray showing bibasal pulmonary infiltrates

cells 3.9 10^9/l with 70% segmented forms, platelets 49 10^9/l, hemoglobin 8.5 g/dl, prothrombin time 80%, thrombin time 100%, fibrinogen 5.95 g/l. Serum cratinine was 85 µmol/l. Arterial blood gases while breathing 10 l/min supplemental oxygen were: pH 7.46, PO_2 63 mmHg, and PCO_2 36 mmHg. Chest X-ray (Fig. 1) showed diffuse patchy infiltrates with a predominance on the two bases of the lungs. Bronchoscopy revealed no evidence of gross hemorrhage. Bloody fluid was aspirated from bronchoalveolar lavage (BAL). The cytologic study showed 370000 cells/ml with 48% macrophages (with numerous intracellular red blood cells and siderocytes), 43% granulocytes (with no morphological abnormalities), and 2% lymphocytes. Bacteriologic study (BAL culture) and tests for pathogenic agent (Pneumocystis carinii, fungus, acid-fast bacilli, viral inclusions) and tumor cells were negative. The diagnosis was IAH secondary to lymphography. Dyspnea resolved spontaneously within 2 days with improvement in blood gases (PO_2 93 mmHg on room air) and a slow improvement shown on chest X-ray.

Lymphography is still used for the extension staging and survey of Hodgkin's disease. Pulmonary complications [1] of lymphography, especially deposition of lipid droplets from the ethiodized oil throughout the pulmonary capillary bed [2], has been well decribed, most often without clinical significance. However, Silvestri et al. have reported two cases of ARDS following injection of ethiodized oil probably related to toxic fatty acid pulmonary emboli inducing extensive alveolar and interstitial inflammation, edema and hemorrhage, as suggested by an animal model [2]. One case of pulmonary hemorrhage that resolved without BAL cytologic examination has been described but was not well documented [3]. Whether IAH takes part of this syndrome is not clearly demonstrated. The differential diagnosis for dyspnea, fever, and pulmonary infiltrates in immunocompromised patients is broad. IAH following lymphography must be kept in mind as reversibility is complete.

References

1. Jackson RSA (1966) Complications of lymphography. B M J 1:1203–1205
2. Silvestri RC, Huseby JS, Rughani I, Thorning D, Culver BH (1980) Respiratory distress syndrome from lymphangiography contrast medium. Am J Respir Crit Care Med 122:543–549
3. Tapper DP, Taylor JR (1989) Diffuse pulmonary infiltrate following lymphography. Chest 96:915–916

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Letter to the editor

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Sir: It is to the great credit of Bone and colleagues to have brought about a transformation in our understanding of sepsis by emphasizing the pathogenetic role of the host's inflammatory reaction. The ACCP/SCCM Consensus Conference in 1992 attempted to develop new nomenclature suitable to this view of sepsis [1]. Nonetheless, the recommended terminology has not received general acceptance within the medical and scientific community and has not gone unchallenged [2]. The greatest weakness of the consensus recommendation is that the definitions of the new disease entities are only in part supported by pathophysiological or epidemiological data and are based on criteria, only a small part of which to date can be seen as characterized with regard to specificity and sensitivity.

The new nosological definitions derive from two assumptions. Firstly, that there is a continuum of the inflammatory host response from minimal to hyperinflammatory; secondly, that a catalogue of *categorical* criteria could permit the definition of homogeneous patient populations with gradually increasing inflammatory activity and corresponding prognoses. The hope of the conference was that the assumption of a continuous course from one level of inflammation to the next would make it possible, with the help of weaker biological responses, to determine in advance the population at risk for the most serious inflammatory syndromes.

So far only one systematic prospective study of the hypotheses of the consensus conference has been conducted. The results have been published in this journal [3] and elsewhere [4]. We would like to comment on the results and their interpretation.

The continuum and clear progression from SIRS to sepsis to severe sepsis and septic shock, which is claimed by Rangel-Frausto et al., cannot be demonstrated by the study design employed. The method used to describe crude mortalities is not adequate for demonstrating the actual SIRS/sepsis-determined contribution to mortality. The actual value of interest would be the attributable mortality rather than the crude mortality, since one can only expect that inflammation-modulating strategies of treatment will be of therapeutic value within the actual mortality contribution of pathological inflammatory responses. We question, for example, whether there is an attributable mortality for a syndrome defined by increased body temperature and white blood cell count, as is assumed by the consensus conference. The correlation of different criteria combinations with an attributable mortality would be demonstrable, for example, by means of case–control studies, as has been shown several times for the classic definition of sepsis as sepsis-induced excess mortality [5].