essential. In this case, needle biopsy was unsuccessful and an open thoracotomy was performed. An alternative approach used in many centers would have been a thoracoscopic excision, which might have resulted in a shorter hospitalization.

The precise etiology of the cystic lesion in this case is unknown. We speculate that hemorrhage and introduction of a small amount of air occurred on removal of the chest drain. The local collection of blood and air, combined with later organization of the blood clot, resulted in the formation of a cystic lesion. The reported incidence of hemorrhage occurring as a complication of the clinical use of heparin varies between 1% to 32%.^{3,4} Spontaneous hemorrhage at a number of sites, including intrathoracic hemorrhage, has been reported,⁵ and this was a likely contributory factor in this case.

Although the cystic lesion proved to be benign in this case, the normal features that aid differentiation of a "pseudotumor" from an actual tumor were absent. There was no pleural effusion elsewhere, the lesion was cystic, and there were no other medical conditions predisposing toward pleural fluid collection, such as cardiac failure or pleurisy. In such cases when intravenous heparin has been used, particularly in the presence of chest drains a hepa-

rin-associated intrathoracic pseudotumor should be considered as part of the differential diagnosis. Nevertheless, a histologic diagnosis is essential to exclude malignancy, and an open thoracotomy or thoracoscopic resection is necessary if less invasive means are unsuccessful.

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SINGLE LEUKOCYTE FILTER (PALL BC1B) FAILS IN MULTIDOSE COLD BLOOD CARDIOPLEGIA

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Leukocyte depletion and its effect on reperfusion injury has been thoroughly investigated over the past several years. It has been shown to prevent myocardial edema, to decrease the incidence of ventricular arrhythmias, and to reduce free radical-mediated lung injury and cardiac reperfusion injury in animal models. Neutrophil granulocytes may cause myocardial stunning as a result of production of oxygen-derived free radicals, which may damage cell membranes, sarcoplasmic reticulum, and different enzymes. Furthermore, granulocytes can plug capillaries

during ischemia; after their degranulation, cell membranes may be damaged by different cell enzymes. Several reports describe leukocyte depletion in human beings. However, most results are not conclusive, especially with regard to filter efficiency.¹⁻³ This study was undertaken to test the efficiency of the Pall BC1B leukocyte filter (Pall Biomedical Products Corp., East Hills, N.Y.) during multidose cold blood cardioplegia.

In a future study we intend to apply leukocyte-depleted blood during blood cardioplegic perfusion and during the first 5 minutes of reperfusion in selected patients with reduced left ventricular function (ejection fraction < 35%) using a double-blind study protocol. Before doing this study, we wanted to test the efficiency of the Pall BC1B leukocyte filter for blood cardioplegia in 16 patients.

Methods. Intermittent cold blood cardioplegic solution was used in all patients. Before aortic declamping the "hot shot" was infused during 4 minutes (250 ml · min⁻¹). All patients received a combination of antegrade (50%) and retrograde (50%) cardioplegic infusion. A single or a double leukocyte filter BC1B for blood cardioplegia (Pall Biomedical) was placed in the cardioplegia line before blood was mixed with cardioplegic solution.

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Table I. Filtration rate (%) of blood cells with single and double Pall BC1B leukocyte filter for blood cardioplegia ($n = 8$)

	Time	SF	DF	p Value
Leukocytes (μ l)	I	97.6 $\pm$ 0.89	99.7 $\pm$ 0.16	0.04
	II	86.5 $\pm$ 2.12	99.2 $\pm$ 0.35	0.002*
	III	69.6 $\pm$ 3.34	98.6 $\pm$ 0.40	0.002*
	IV	55.7 $\pm$ 3.72	95.6 $\pm$ 1.11	0.002*
Neutrophils (μ l)	I	97.6 $\pm$ 1.09	99.9 $\pm$ 0.07	0.1
	II	86 $\pm$ 2.98	99 $\pm$ 0.91	0.002*
	III	72.6 $\pm$ 2.63	98 $\pm$ 0.36	0.002*
	IV	53.3 $\pm$ 4.30	95.2 $\pm$ 0.70	0.002*
Lymphocytes (μ l)	I	97 $\pm$ 0.73	99.5 $\pm$ 0.15	0.02
	II	88.8 $\pm$ 2.27	99.5 $\pm$ 0.13	0.002*
	III	53.4 $\pm$ 4.82	99.8 $\pm$ 0.12	0.002*
	IV	65.6 $\pm$ 5.00	93.3 $\pm$ 0.75	0.002*
Platelets ($10^3/\mu$ l)	I	7.1 $\pm$ 1.78	13.9 $\pm$ 1.15	0.02
	II	6.8 $\pm$ 0.88	18.1 $\pm$ 1.79	0.002*
	III	2.7 $\pm$ 0.48	5 $\pm$ 0.95	0.08
	IV	0.7 $\pm$ 0.42	5.5 $\pm$ 1.05	0.002*

Values for filtration rate are given as mean plus or minus standard error of the mean. n , Number of patients in each group; SF, single Pall BC1B leukocyte filter; DF, double Pall BC1B leukocyte filter; time I, initial blood cardioplegia; time II, second blood cardioplegia; time III, third blood cardioplegia; time IV, terminal blood cardioplegia (hot shot).

* $p < 0.01$, significant difference from single filter rate versus double filter rate.

Blood samples for blood cell counts were collected proximal and distal to the leukocyte filter. Blood cell counts were immediately determined on samples containing ethylenediaminetetraacetic acid with the use of a counter (Sysmex K 1000, Sysmex Corporation of America, Long Grove, Ill.) and differential cell counting in Naegotte chambers.

The results are presented as the mean $\pm$ standard error of the mean. Statistical analysis was performed by means of the Mann-Whitney U test for difference in medians, after an analysis of variance. A p value < 0.01 was considered to be statistically significant.

Results. Filtration rates of blood cells are depicted in Table I. After the second period of cardioplegia, the filtration rate of leukocytes in patients with only one filter was significantly lower than that of patients with two filters. Similar results were obtained from neutrophil and lymphocyte counts. Platelets were barely filtered in either group. However, the filtration rate for platelets was higher in the double filter group.

Discussion. Ishihara and coworkers³ demonstrated that elimination of neutrophils by a filter down-regulated neutrophil elastase and lipid peroxidation, resulting in significantly lower values of creatine kinase MB. They

summarized that leukocyte removal filters may decrease the myocardial damage caused by neutrophils during cold blood cardioplegia in cardiac operations. Sawa and associates¹ found that use of terminal leukocyte-depleted blood cardioplegia reduced the release of oxygen-derived free radicals and the amount of catecholamine support. However, Mihaljevic and colleagues² could not demonstrate a favorable effect after use of a leukocyte filter.

Heggie and coworkers⁴ reported a leukocyte filtration rate of 75% for up to 5.3 liters of whole blood when a Pall BC1B filter was used; the filtration rates for platelets and erythrocytes were 15% and 2.5%, respectively. In our study, the leukocyte filtration rates when a single Pall BC1B filter was used were 97.6% and 86.5% during the first two cardioplegic perfusions. However, during the following perfusions, the filtration rate in the single filter group was significantly lower than in the double filter group. Thus we could demonstrate that the filtration rate of one single filter is insufficient after just two perfusions of cardioplegic solution. Inasmuch as leukocyte depletion increases left ventricular recovery, especially in the initial reperfusion period, a sufficient leukocyte filtration rate would be essential during the terminal perfusion with blood cardioplegia.

We conclude that leukocyte filtration with a single Pall BC1B filter for blood cardioplegia is merely effective up to 800 ml of whole blood. When additional perfusions are necessary, a second filter is required to attempt effective leukocyte filtration.

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