ling the causes, achieving prevention, and managing many arthritic disorders require a rigorous biomechanical approach. Man is not entirely a series of immunoglobulins, but possesses more than 300

beautifully engineered joints. When they go wrong the engineer, in collaboration with the clinician, may well be able to lend a helping hand.

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Pulmonary involvement in SLE

Sir,

Grennan *et al.*¹ recently reported in 22 patients with systemic lupus erythematosus (5 with respiratory symptoms), a restrictive ventilatory defect or reduction in diffusing capacity for CO.

Their paper is interesting but we should like to make a few comments: (a) Only 3 tests of lung function were employed: VC, FEV₁%/FVC, and transfer factor. (b) Most patients were receiving corticosteroids at the time of assessment. (c) Transfer factor was considered to be reduced if it was less than 80% of the mean predicted. One patient (case 12) with a reduced diffusing capacity of 78% had a transfer factor of 7.1 mmol/min/KPa under the predicted normal level (7.4–10.8 mmol/min/KPa) and 2 patients (cases 9 and 10) with a similar mean predicted reduction of diffusing capacity (79%) had normal transfer factor values. In this sense patients 6 and 8 were considered affected, although their transfer factor levels fit into predicted normal values. (d) Statistical methodology should have been described in order to show that there were no significant differences between patients with and without reduced transfer factor with regard to mean DNA binding capacity, mean C3 and C4 levels, or a positive test for rheumatoid factor.

At the XIVth International Congress on Internal Medicine (Rome, October 1978) we communicated a study of pulmonary function in 22 lupus patients without respiratory symptoms compared to 30 normal persons. Our analysis included assessment of static and dynamic lung volumes, lung mechanics, and CO diffusing capacity

(see Table 1). The most important alterations found in our patients were: (1) A restrictive pattern with hyperdistention expressed by decreased VC and increased RV with regard to controls ($P<0.01$). (2) Loss of elastic component expressed by decreased compliance ($P<0.01$). (3) A block in the alveolar gaseous transference capacity with highly significant alterations in CO diffusion ($P<0.01$), and in CO₂ and O₂ arterio-alveolar gradients pressures ($P<0.01$). (4) No correlation between pulmonary dysfunction with either activity or duration of the disease.²

These conclusions are in keeping with those recorded by Olsen and Lever,³ Edmonds *et al.*,⁴ and recently Grennan *et al.*¹

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Table 1 Pulmonary function data in normal controls and in systemic lupus erythematosus patients

	Controls Mean $\pm$ SD		Patients Mean $\pm$ SD		P
Vital capacity (VC) ml	3458	691	2795	864	<0.01
Mean predicted VC (%)	100	0	79	18	<0.01
FEV ₁ /VC $\times$ 100	75	4	77	10	NS
Residual volume (RV) ml	1096	252	1602	753	<0.01
Total lung capacity (TLC) ml	4556	865	4398	962	NS
RV/TLC $\times$ 100	24	4	39	18	<0.01
Compliance (ml/cm H ₂ O)	0.18	0.12	0.08	0.17	<0.01
Airways resistance (raw) (cm H ₂ O/l/s)	1.50	0.30	3.10	0.50	<0.01
Critical flow (l/s/l) [†]	2.70	0.40	3.0	0.90	<0.05
Peak flow (l/s)	6.0	3.0	5.9	2.1	NS
Specific conductance (SGaw) (cm H ₂ O/l/s/l)	0.245	0.115	0.161	0.146	NS
Functional alveolar ventilation (ml/min)	3886	366	5777	2807	<0.01
PaO ₂ (mmHg)	82	10	77	10	NS
PaCO ₂ (mmHg)	40	5	36	2	<0.01
Alveolar—arterial gradient pressure for O ₂ (A-a) DO ₂ (mmHg)	10	5	20	6	<0.01
Arterio—alveolar gradient pressure for CO ₂ (a-A) DCO ₂ (mmHg)	3	1.5	12	4.5	<0.01
CO diffusion (D-CO) (ml/min/mmHg)	18.7	1.6	13.7	4.6	<0.01
D-CO/functional alveolar ventilation ratio (ml/min/mmHg/l)	4.8	0.3	2.6	0.9	<0.01

*FEV₁ = Forced expiratory volume in the first second.

[†]Critical flow = forced maximal flow (FMF) 25%–75%.