100% responded. Only a small minority (2/20) disagreed with the diagnosis and most (14/20) felt that the diagnosis was of benefit in the management of the patient. Since receiving instruction in breathing exercises from a physiotherapist only 1 of 20 patients required referral elsewhere with similar problems. This data supports the use of physiotherapy from an experienced professional in the treatment of hyperventilation.

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Is spirometry feasible for institutionalised mentally handicapped?

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In recent years both therapeutic and diagnostic procedures for asthma and COPD patients have been improved. However, these improvements did not reach mentally handicapped. Stuart (BMJ 1990; 300: 720–1) showed increased morbidity and mortality due to asthma in mentally handicapped. Diagnosis of asthma/COPD is based on history, examination and lung function assessment. In mentally handicapped often only examination is used.
In this study we aimed to assess if lung function assessment is possible in institutionalised mentally handicapped. In 172 mentally handicapped with a mild-severe handicap lung function was assessed using a Multi-Spiro SX (MultiSPiro Inc, Tempe, USA). A special incentive (birthday cake) was used to make the test more attractive. Spirometry results were scored as yes/no technically acceptable. Results of spirometry were compared with chart review.
In 95 mentally handicapped the test was not acceptable. In 77 it was acceptable. 50 mentally handicapped showed an FEV_1 ≥80% predicted; 27 showed an FEV_1 <80% predicted. The ATS reproducibility criterion was met in 44 patients only. (24 FEV_1 ≥80%) Less severe mentally handicapped produced more technically well tests, and were more easily testable. The reproducibility criteria are less usable in this population. Chart review showed 12 mentally handicapped with a COPD, 1 with an asthma and 2 with an CNSLD diagnosis.
Conclusion: Also in mentally handicapped spirometry is a valuable tool to diagnose asthma/COPD. For severe mentally handicapped subjects other diagnostic tools are necessary.

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"Pneumotox": A Drug-induced lung diseases page on the web

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There is a continuing increase in the number of drugs that may damage the respiratory system (more than 220 at this time), and the clinical pattern of involvement from drugs is varied. Thus, they cannot be easily memorized. The GEPPI (Groupe d'Etudes de la Pathologie Pulmonaire Iatrogène) group was founded in France in 1995 under the auspices of the SPLF (Société de Pneumologie de Langue Française) and the AFCP (Association Française des Centres de Pharmacovigilance), mainly to provide readily available and uptodate information regarding drug-induced lung disease and collect case reports.
For this purpose, the GEPPI created *Pneumotox* in November 1996. *Pneumotox* is a Web site that provides easy-access to a continuously upgraded information about drug-induced lung diseases over the Internet network. So far, it is the only available site on this topic.
The database can be browsed either by generic drug name or by clinical-radiographic pattern of involvement (e.g. interstitial lung disease, pulmonary oedema, pulmonary haemorrhage, airways disease, pleural changes, vascular changes, mediastinal changes, major airways involvement, muscle and nerves, constitutional/systemic symptoms, and variegated effects). Both modes lead to a specific page for each of the 226 drugs quoted to date. Each page contains data concerning the drug of interest as well as typical radiographic and pathology pictures whenever available along with a selected literature.
Some areas such as drug-induced effects on respiratory muscles or drugs that may cause bronchospasm or asthma are still under construction.
Literature on any drug-induced pulmonary effect can be extracted from a database of 2780 papers (to date) through a simple form.
Pneumotox is a non-commercial, non-sponsored and free access contribution to medical information on adverse effects of drugs. The URL (Uniform Resource Locator) of *Pneumotox* is *http://www.pneumotox.com.*

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The relationship between lung function and lethal outcome in patients with severe kyphoscoliosis

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The aim of the study was to observe the lung function and survival in 28 patients with severe degree of kyphoscoliosis originating in the childhood. The group of 17 patients, who died during 8 year follow up period, was submitted to detailed analysis. The average age of these patients was 54 ± 12.01 years initially, ranging from 29 to 71 years. At the beginning of the study they had severe degree of predominantly restrictive ventilatory impairment with mean spirometric values of vital capacity (VC) 1.16 ± 0.44 litres (48.2 ± 11.77% of predicted), forced expiratory volume in one second (FEV_1) 0.70 ± 0.23 litres (37.2 ± 10.37% of predicted) and the ratio $100 \cdot FEV_1/VC$ 62 ± 9.6. At the same time the patients were in the stable state of the respiratory insufficiency with following arterial partial pressures of oxygen and carbon dioxide: PaO_2 7.57 ± 0.79 kPa and $PaCO_2$ 7.2 ± 1.06 kPa, respectively. All of them had cor pulmonale. Dyspnea occurred in the history 2–15 years before death (average 7.53 ± 4.68 years). First episode of heart failure appeared 1–6 years before death (average 3.17 ± 2.07 years). There was a significant decrease in PaO_2 (in average from 7.57 ± 0.79 to 6.26 ± 0.94 kPa; $p < 0.005$) during the follow up period. At the same time no significant changes were found in $PaCO_2$ as well as in spirometric measurements of VC, FEV_1 and the ratio $100 \cdot FEV_1/VC$. It is concluded that, among the observed parameters of lung function, PaO_2 is the best indicator of deterioration to the lethal outcome in patients with severe kyphoscoliosis.

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Reduction of inspiratory muscle performance during steroidtherapy

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A marked reduction of inspiratory muscle performance under therapy with high-dose corticosteroids for a short period is already known. Aim of our study was to check whether a low dose steroidtherapy for 6 months could also influence inspiratory muscle contractility in patients with chronic obstructive pulmonary disease (COPD) or asthma (A).
In 51 patients (A, n = 23, COPD, n = 28) we performed bodyplethysmography and measured mouth occlusion pressures twice within an period of 202 ± 30 days. Mean daily steroid dose during this period was calculated. Data analysis was done with exel 5.0, calculation of significance with student-t-test.
Over all the maximum static inspiratory pressure (pimax) decreased from 6.90 ± 2.43 to 6.34 ± 2.17 kPa ($p = 0.01$) whereas residual volume (RV) and FEV1 did not change. Divided in subgroups the decrease of pimax was significant in patients with AB, in patients with mean daily steroiddose <10 mg prednisolone (n = 25) and in patients with lower daily steroiddose than before (n = 18). In three other subgroups-patients with COPD, with daily steroiddose >10 mg prednisolone (n = 26) and patients with higher daily steroiddose than before (n = 24) – the reduction of pimax was not significant. See table

	Tot.	A.	COPD	<10 mg	>10 mg	Dose¬	Dose√
pimax1	6.9 ± 2.4	8.0 ± 2.6	6.0 ± 1.9	7.2 ± 2.0	6.6 ± 2.8	8.1 ± 2.5	6.8 ± 1.5
pimax2	6.3 ± 2.2	7.0 ± 2.5	5.8 ± 1.7	6.7 ± 1.9	6.0 ± 2.4	7.1 ± 2.5	6.3 ± 1.7
p-value	0.01	0.01	>0.05	0.03	>0.05	0.01	>0.05

The function of the ventilatory pump is negatively influenced by longtime low dose steroidtherapy and this even in doses <10 mg prednisolone. In patients who already show low pimax values a further significant decrease of pimax values is not detectable. We also could show this in patients who required a higher daily dose steroidtherapy due to a worsened airway obstruction.

Interstitial lung disease

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Bronchiolitis obliterans organising pneumonia (BOOP)

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BOOP is a specific clinical-pathological entity of unknown etiology (then preferably named cryptogenic organising pneumonia) or possibly associated with some other conditions. It generally has a good prognosis except for the uncommon, rapidly progressive form.
This study reports on 11 cases (6 males/5 females) of clinical-pathological BOOP-syndrome (mean age 58 yrs, range 17–73 yrs), with an unexpectedly high mortality rate of 36% (4 cases). The disease was idiopathic in 7, and was associated with intake of amiodarone (in 1), with past mycoplasma pneumonia (in 1) and with connective tissue disease (in 2). There was a history of flu-like syndrome, cough and dyspnea of a mean duration of 4 months (range 1 week to 8 months). Lung function was mostly restrictive or/and obstructive with a diffusing capacity ranging between 47 and 95%; there was hypoxia in about half of the patients. Chest X-ray and CT-scan showed a patchy consolidation with linear opacities (unilateral in 4, bilateral in 5) and/or possibly a ground glass pattern (in 4), and a focal pseudo-tumoral lesion (in 1). Bronchoalveolar lavage showed a variable pattern of mixed, or eosinophilic or neutrophilic alveolitis. Histologic diagnosis was based on open lung biopsy (in 3), on thoracoscopic biopsy (in 2), on transbronchial biopsy (in 2), on wedge resection of the nodular lesion (in 1) and on postmortem lung biopsy (in 3): it showed proliferative obliterative bronchiolitis and organising pneumonia. One patient recovered spontaneously, 1 remained cured after resection of the focal lesion, 7 were treated with 16–125 mg methylprednisolone (of whom