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A NEW VENTILATION MODE " DECELERATED CONTROLLED EXPIRATION " (DCE): INFLUENCE ON RESPIRATORY MECHANICS.

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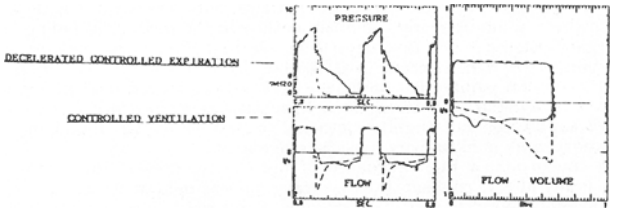
DCE is a new ventilation mode obtained on a CESAR ventilator (TAEMA-CFPO) by servo-controlling expiratory pressure from an initial value Pi to a final value Pf during a part of expiration Tf. This study evaluated the respiratory effects of DCE in order to optimize ventilation.12 sedated and curarized patients were ventilated for acute respiratory failure (6 ARDS,6 COPD) successively with controlled ventilation (CV) and optimum DCE. Optimum DCE was characterized by the squarest flow-volume curve during expiration (cf Figure).



After one hour of each mode, airway pressures (P, Ppeak, Pmean, PEEPi), flow, volume and arterial blood gas were mesured. When compared with CV, DCE allowed: 1)First, a complete control of the expiratory flow 2)Second, a significant increase of Pmean(11.3 vs 7.7cmsH2O) without significant increase of Ppeak(26 vs 25cmsH2O) and PEEPi(6.3 vs 4.8 cmsH2O). Consequently, PaO2 increased(113 vs 103cmsH2O) 3)Third, probably a prevention of the early collaps of bronchiols during expiration according to flow-volume curves. Consequently, the PaCO2 decreased(48.5 vs 54cms H2O).DCE should improve ventilation of patients by optimizing expiratory flow and minimizing barotraumatic effects.

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TREATEMENT OF PERSISTENT LOBAR ATELECTASIS WITH INTRAPULMONARY PERCUSSIVE VENTILATION DURING POSTOPERATIVE PERIOD

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Pulmonary atelectasis is frequently encountered in critically ill patients specially when they are smokers or during the postoperative period (thoracic surgery). Persitent atelectasis leading to hypoxemia and pulmonary infection needs particularly an aggressive therapeutic approach.

Nine patients (8 smokers) developed during the early postoperative period (bilobectomy 3, vascular surgery 1, thoracic reconstructive surgery 4) persistent lobar atelectasis resistant to current treatments including antibiotics, mucolytics, aerosols and physiotherapy (vibration, suction, postural drainage, ...) All these patients were nonintubated and developed clinical respiratory deterioration with diagnostic of a lobar atelectasis on chest roentgenogram. Repeated fiberoptic bronchoscopy was performed in all patients without definitive and complete treatment of the lobar atelectasis.

Intrapulmonary percussive ventilation (IPV) was applied by a face mask, every two hours, leading in all cases to the normalisation of the pulmonary clinical and radiological status, within 6 hours after the first application of the high frequency percussions (100 to 400 cycles/min). Tolerance was excellent in all patients.

This observation suggests that IPV can be a usefull therapeutic method to improve pulmonary drainage in postoperative nonintubated patients and to resolve persistent pulmonary atelectasis.

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Metabolism I

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EFFECT OF INTRAVENOUS GROWTH HORMONE ON FUEL METABOLISM AND ELECTROLYTE BALANCE IN CRITICALLY ILL PATIENTS.

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Growth hormone (GH) as adjunctive therapy during nutritional support has been shown to decrease nitrogen excretion in postoperative and burn patients. Its effects in critically ill patients are not known, especially when administered as a continuous intravenous infusion.

Twenty critically ill patients were studied within 24 hour after admission. Patients were studied for four days. All patients required mechanical ventilation and received standard parenteral nutrition. GH (Norditropin) 0.1 mg/Kg/day was administered on day 2,3 and 4. Fuel utilization was calculated from indirect calorimetry. Results: Nitrogen balance was negative upon entry, but became slightly positive on the third day in the GH group (p=0.03). Cumulative nitrogen balance was still negative but different: GH -13±32 g and controls -26±25 g. Carbohydrate and fat utilization were not different. Nonprotein RQ showed a tendency to decrease in the GH group, while it increased in controls.

Calcium, phosphate and potassium balance all increased during treatment in both groups (p < 0.005). Sodium balance decreased in both treatment and control group (p=0.001). The phosphate balance was higher in the treatment group, but of borderline significance (p=0.054).

Intravenous GH administration in critically ill patients is safe and results in a reduction of nitrogen loss and improves phosphate retention indicating preservation of muscle mass.

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EFFECT OF EXOGENOUS ADRENALINE ON AMINO ACID METABOLISM AS SHOWN IN ALANINE AND LEUCINE TURNOVER

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Introduction: Adrenaline is primarilly administered for hemodynamic therapy. But there are also clear metabolic effects on carbohydrate and fatty acid metabolism and despite lower evidence on amino acid metabolism too. Using a stable isotope tracer technique we investigated the effect of a 4 hr adrenaline infusion (0.1 µg/kg*min) on alanine and leucine turnover.

Methods: After approval by the ethic committee 7 young healthy volunteers participated in the study after informed written consent. After an overnight fast venous catheters were inserted on both arms for infusion and blood sampling respectively. At t=150 min priming doses of ^{15}N-alanine (0.63 mg/kg) and methyl-D_3-leucine (0.27 mg/kg) were administered followed by a continuous infusion of 0.007 mg/kg*min and 0.005 mg/kg*min respectively. Blood samples for determination of isotopic enrichment were drawn at t=220, 230 and 240 min. At t=241 min an infusion of adrenaline (0.1 µg/kg*min) was started. Further blood samples were drawn at t=260, 280, 300, 310, 320, 340, 360, 380, 390, 400 and 420, 440, 460, 470, 480 min. Isotopic enrichment was measured using gas chromatography/mass spectrometry (GC 5890 HP, MS 5970 HP). The n-propyl-N-acteyl-derivatives of both used amino acids were used for analysis. The statistical analysis of the data was performed by analysis of variance. $p<0.05$ was considered significant.

Results: The alanine flux showed a significant increase during adrenaline infusion whereas leucine flux decreased significantly ($p<0.05$). Values±SD are shown in Table 1.

	Ala-Flux*	Leu-Flux*
240	0,33±0,05	0,28±0,03
320	0,57±0,07	0,29±0,02
400	0,60±0,05	0,24±0,02
480	0,61±0,08	0,22±0,02

*Table 1: Values ±SD of alanine and leucine-flux (mg/kg*min) before (240 min) and during infusion of adrenaline (320, 400 and 480 min). (p<0,05).*

Conclusions: The administration of adrenaline induced an enhancement of alanine turnover as a sign of increased Cori cycle activity and simultaneous a decrease in leucine flux showing reduced proteolysis as an effect of adrenaline infusion. Similar results were described by other authors using AV-differences. These observations make it evident that adrenaline has beside its hemodynamic effect a clear impact on amino acid metabolism too.

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EFFECT OF ADRENALINE ON GLUCOSE AND UREA PRODUCTION AND LEUCINE AND ALANINE FLUX IN VOLUNTEERS

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Trauma, operation, sepsis and IPPV lead to catabolism and a decrease in the splanchnic perfusion. Sympathomimetics are often used in these patients and haemodynamics are in the centre of interest and monitoring. Metabolic effects of this treatment usually are not regarded. We investigated the effects of the prototype catecholamine adrenaline (ADR) on glucose (G) and urea (U) production and on leucine (LEU) and alanine (ALA) flux in volunteers using a stable isotope tracer technique.

After approval by the local ethics committee 7 volunteers were studied after an overnight fast. Priming doses of [$^{15}N_2$]-U, [6,6-D_2]-G,[methyl-D_3]-L-LEU and [$^{15}N_2$]-L-ALA were administered followed by continuous infusions. After isotopic steady state was reached blood samples were taken to determine the isotopic enrichment (APE). ADR 0.1 μg/kg min was then infused for 240 min. 80, 160 and 240 min after the start of the ADR infusion further blood samples were taken to determine the APE. The APE in the plasma and in the infusate (INF) were determined by GCMS (HP 5890, HP 5970B). The rate of appearance (Ra) was calculated as Ra=((APE(INF)/APE(plasma) - 1) * isotope infusion rate. Data are presented as means $\pm$ SD.

Ra of G increased from 14.1 μmol/kg min by 52 % after 80 min of ADR and decreased again without reaching baseline. The LEU flux decreased from 2.15 $\pm$ 0.21 μmol/kg min by 21 % and the ALA flux increased from 3.74 μmol/kg min by 84 % at t = 480 min. U production was 4.83 μmol/kg min and decreased by 10 % during adrenaline (all changes $p < 0.05$, anova).

The increase in G production represents hepatic gluconeogenesis (GNG) which is an oxygen consuming process. The increase in GNG may deteriorate the hepatic oxygen balance in patients during treatment with adrenoceptor agonists. ADR does not increase proteinolysis as shown by the decrease in LEU flux and U production. The increase in the ALA flux provides C_3 precursor from skeletal for hepatic gluconeogenesis. Since U production decreased the metabolic pathway of nitrogen from hepatic deamination of alanine remains to be investigated. Studies in patients are necessary to investigate the importance of these adrenergic metabolic effects.

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CRITICAL CARE HYPERCALCEMIA - A HYPERPARATHYROID STATE

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Hypocalcemia is a well known finding in critically ill patients. Later occurrence of mild hypercalcemia has also been reported.

In order to investigate the incidence and nature of critical care hypercalcemia serum calcium was measured in 83 critically ill ICU patients (TISS score ≥40) and related to the occurrence of acute renal failure (ARF) and severity of illness, evaluated by the APACHE-II and the multiple organ failure scoring systems.

Twenty-two percent of the patients developed hypercalcemia (serum calcium ≥2.60 mmol/l) during their ICU stay. These hypercalcemic episodes (mean maximal value 2.71 ± 0.12 mmol/l) were more common and occurred earlier in patients with co-existing ARF. However, multiple regression analysis showed the number of failing organ systems in the first days to be the best predictors for later occurrence of hypercalcemia ($p<0.0001$).

When serum parathyroid hormone (PTH) was measured in 6 of the patients without ARF during their hypercalcemic episodes PTH was not suppressed but slightly elevated, i.e. similar to that found in patients with mild primary hyperparathyroidism.

In conclusion, a high incidence of hypercalcemia was found in critically ill ICU patients. The hypercalcemia was mild and was more frequently found in patients with co-existing renal failure. The most powerful predictor to later occurrence of hypercalcemia was however the severity of the illness in itself. The raised levels of PTH found during the hypercalcemic episodes suggest ICU hypercalcemia to be caused by parathyroid overactivity.

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LACTIC ACIDOSIS IN THIAMINE DEFICIENCY

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Thiamine(Vitamin B1) is a co-factor of pyruvate-dehydrogenase. Thiamine deficiency may impair pyruvate utilisation and result in plasma lactate (LAC) accumulation. We report two cases of lactic acidosis, in whom no other causes of hyper-lactemia could be identified and in whom thiamine supplementation resulted in a rapid normalisation of LAC and acid base status.

Case 1 was a patient with multiple sclerosis and poor nutritional intake for more than 4 weeks who was admitted because of septicemia and lactic acidosis. Case 2 was a diabetic patient with acute on chronic renal failure, continuous hemofiltration therapy and impaired nutritional intake for several weeks. Values before/after supplementation of 600 mg thiamine:

Cases 1 and 2	before	after	before	after
LAC (mmol/l)	11.9	2.0	10.9	2.4
pH	7.24	7.46	7.11	7.29
Bic.(mmol/l)	16.0	29.0	8.60	21.2
Transketolase mU/g	1.35	1.98	0.54	1.10
Transketolase mU/g (thiamine-PP stimulation)	1.61	1.99	1.45	1.29
Stimulation (%)	20 %	0 %	170 %	17 %

Additionally, in both patients a right heart failure was confirmed by thermodilution catheter /echocardiography and this was rapidly reversible by vitamin supplementation.

We conclude that unexplained lactic acidosis may result from thiamine deficiency and that intensive care patients with infections and/or renal failure and limited nutritional intake are at risk of developing overt vitamin deficiency.

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PROGNOSTIC VALUE OF FREE THYROXINE (FT4) LEVEL IN EARLY SEPTIC SHOCK. T. Boulain, C. Osorio-Salazar*, P. Lecomte*, A. Legras, C. Valat**, D. Perrotin.

Critically ill patients often have abnormal circulating thyroïd hormones levels.Very low levels of free triiodothyronine (FT3) and FT4 are of poor prognosis. However this has not been investigated in the early hours of septic shock.

On admission FT3, FT4, reverse T3 (rT3), thyrotropin (TSH), thyroxin-binding protein, and free fatty acids levels were measured in fifteen patients (aged 64±17) with septic shock who had not any previous history of dysthyroïdism. Patients were not yet treated by intravenous catecholamines, corticosteroïds or anticonvulsants. Hormones dosages were performed by Radio-Immunology Assay.

RESULTS : Seven patients survived (S) and eight died (D). The mean Simplified Acute Physiology Score (SAPS) was 18.6 ± 5.5 (mean ±SD) . SAPS was different between S and D patients (S : 14 ± 2.4 vs D : 22. ± 4.3 ; $p<0.001$). TSH and FT3 were not different between S and D patients (TSH : 0.93 ± 0.6 mU/l vs 1.32 ± 1.8 and FT3 : 0.75 ± 0.3 pMol/l vs 0.66 ± 0.32 respectively). RT3 was higher in S patients (283 ±137 vs 645 ± 399 pMol/l ; $p<0.03$). FT4 was higher in S patients (6.2 ± 2.6 vs 15.4 ± 5.7 pMol/l ; $p < 0.002$). FT4 was correlated to SAPS ($r = 0.56$; $p < 0.04$). Both SAPS and FT4 were significantly associated with outcome ($p < 0.001$). Stepwise discriminant analysis confirmed these associations. When both FT4 and SAPS were taken into account, prediction of outcome was more powerful than when SAPS only was considered (SAPS only : partial R2= 0.63, F=20.7 ; FT4 + SAPS : partial R2=0.74, F=15.8).

CONCLUSION: High FT4 levels on admission of patients with septic shock, when determined before any treatment with drugs which can modify thyroïd hormones levels, are of good prognosis and complement the outcome prediction by SAPS.

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