# Noninvasive assessment by MR imaging of pulmonary congestion following a large myocardial infarction in rats

M. Obayashi[1], U. I. Tuor[2,3], T. Foniok[2], D. Kirk[3], B. Stuyvers[1], B. Tomanek[2,3], H. E. ter Keurs[1]

[1]Department of Medicine, University of Calgary, Calgary, Alberta, Canada, [2]MR Technology, Institute for Biodiagnostics (west), Calgary, Alberta, Canada, [3]Experimental Imaging Center, University of Calgary, Calgary, Alberta, Canada

**Introduction**

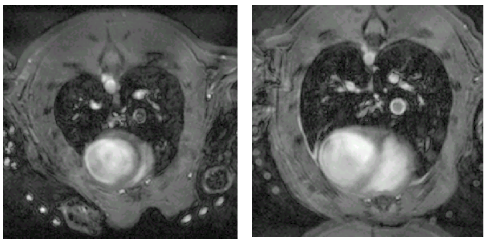
The diagnosis of congestive heart failure (CHF) is usually based on symptoms, clinical signs, and chest X-rays.CHF often occurs as a serious outcome of large myocardial infarction (MI).An increase in left ventricular (LV) end-diastolic pressure due to LV dilation after MI induces pulmonary congestion.Although post-infarction animal models have been studied extensively as experimental models of CHF, not all hearts with a large infarction subsequently undergo a transition to CHF.Therefore, when using these models of CHF, it would be important to be able to diagnose CHF noninvasively and these models may also help in developing new diagnostic methods of CHF.It has been reported that echocardiographic left atrium diameter is larger in failing post-MI heart, accompanying pulmonary congestion, than in compensated post-MI heart[1].Nevertheless it is difficult to estimate the extent of pulmonary congestion in animal models by echocardiography.In this study, we assessed the extent of in vivo pulmonary congestion following a large MI by MR imaging of the heart and lung and compared this to the extent of postmortem lung weight where the latter is usually used as an index of pulmonary congestion.

**Material and Methods**

Infarction model was induced in Male Lewis Brown-Norway rats of about 250-320 g body weight as described previously [2].Briefly, rats were anesthetized with injection 40 mg/kg of sodium pentobarbital into the peritoneal cavity and were artificially ventilated.After left thoracotomy, the main left coronary artery (LCA) was ligated using 6-0 nylon suture.Age-matched sham rats were prepared in the same fashion but the LCA was not ligated.The animals received food and water ad libitum during the development of CHF after the surgical procedure (3 months).3 months after the surgery, we estimated the extent of in vivo pulmonary congestion by MR imaging.Within 1 week after the MR imaging measurement, echocardiology and right ventricular (RV) cathertherization were performed, and postmortem heart weight and, lung wet and dry weights were measured.The MR imaging protocol used a SNAP_TOMO pulse program that provided multislice fast gradient-echo images [3].MR imaging parameters were: matrix size = 128x128 (reco 256x256), TR= 10 ms, TE=2.3 ms, $\alpha$= 30˚, SNAP Inter Scan Time= 530 ms, No.Avg.=40, Fov= 5 cm, Slices= 30x 1.5 mm, Slice Scheme= Sequential, Total Acquistion= 36m 12s.Lung volume was measured by measuring lung area in each slice by tracing the lung edges in each section and summing the area x 1.5 mm (slice thickness) for each slice containing lung.Lung intensity was measured by comparing lung and muscle intensity in the same slice.

**Results**

Rats were divided into 3 groups 3 months after MI: Sham; Failing MI (lung wet weight /body weight >6), and Nonfailing MI (lung wet weight /body weight <6).Peak RV pressure and RV weight in Failing MI significantly increased compared to Sham and Nonfailing MI (Peak RV pressure (mmHg) /RV weight (mg / body weight (g)) = 47.4 ±6.0 / 1.03 ±0.14 in Failing MI vs 21.9±3.2 /0.52 ±0.07 in Nonfailing MI by ANOVA).LV end-diastolic area (95.1±9.4 $mm^2$) and tricuspid valve diameter (5.6 ±0.4 mm) as an index of RV dilation in Failing MI also increased compared to Nonfailing MI (71.0 ±7.9, 4.3 ±0.2, respectively).Lung volume in Failing MI increased compared to Sham and Nonfailing MI, based on increases in each lung transverse area at upper, middle, and lower lung fields (lung volume = 26.4±0.5 $mm^3$/ body weight (g) in Failing MI vs 15.9±1.8 in Nonfailing MI, see Fig.2).There was a strong correlation ($r^2$=0.971) between lung wet weight and lung volume.Relative lung intensity compared to muscle did not increase in Failing MI.Lung wet/dry weight ratio, accompanied by an increase in lung dry weight in Failing MI, was not different from Sham and Nonfailing MI.



Nonfailing MI Failing MI

Fig 1. MR images at the same level through the heart showing the enlargement of the LV in Failing myocardial infarction (MI). Note that RV dilation occurs in Failing MI.

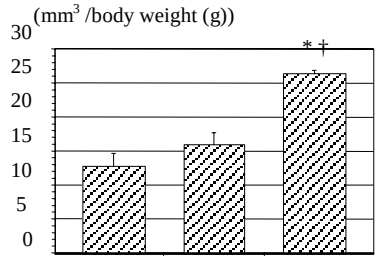



Fig 2. Lung volume calculated by MR imaging *p <0.05 vs Sham, † p< 0.05 vs Nonfailing MI by ANOVA

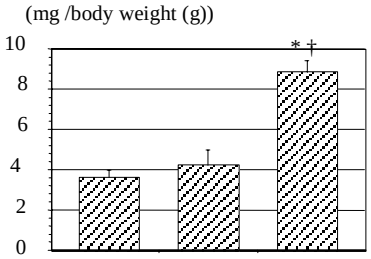



Fig 3. Postmortem lung wet weight

**Conclusions**

Lung wet weight, used usually as an index of pulmonary congestion, was neither correlated to a change in water content per dry weight of lung tissue nor MR tissue intensity. Instead wet weight correlated closely to lung volume measured using MR suggesting that MR determination of lung volume can be used to estimate the extent of in vivo pulmonary congestion noninvasively.

**References**

1. Sjaastad I., Sejersted O.M., Ilebekk A., Bjornerheim R.. J Appl Physiol 2000;89:1445-1454.

2. Davidoff A., Boyden P., Schwartz K., Michel J., Zhang Y., Obayashi M., Crabbe D., ter Keurs H.. Ann NY Acad Sci 1015:84-95,2004.

3. Haase A., Fraham J., Matthaei D., Haenicke W., Merboldt K.. J Mag Res 67:258-266, 1986.