Response to: 'Disease activity and left ventricular systolic function in rheumatoid arthritis' by Giollo *et al*

We thank Giollo *et al*[1] for their interest and comments to our paper.[2] In this paper, we demonstrated that rheumatoid arthritis (RA) disease activity was associated with lower left ventricular (LV) myocardial systolic function when assessed by stress-corrected midwall shortening (scMWS) or global longitudinal strain (GLS).[2] Further, we found that this association was independent of the presence of a normal ejection fraction and traditional cardiovascular risk factors like hypertension and smoking. In a letter to the editor, Giollo *et al*[1] present data from a series of 235 patients with RA.[3] In contrast to our findings, Giollo *et al*[1] did not find an association between RA disease activity and LV systolic myocardial function when assessed by scMWS or tissue Doppler mitral annular systolic velocity (S´), despite a much higher median RA disease activity in their population.[3] The higher RA disease activity probably also explains the much higher prevalence of abnormal LV geometry in the study by Giollo *et al*,[1] since prevalences of hypertension, diabetes and obesity, all factors associated with LV hypertrophy, did not differ between the study populations.[2 3] Of note, the higher RA disease activity was found despite a higher use of immunomodulation therapy in their study. Giollo *et al* suggest that the association between RA disease activity and LV systolic function should have been evaluated in relation to LV geometry, and the results are shown in table 1 below. As demonstrated, LV systolic function was lower among patients with active RA (Simplified Disease Activity Index ≥3.3) compared with patients with RA in remission when LV geometry was normal. Also when LV geometry was abnormal, patients with active RA had numerically lower LV systolic function although this was not statistically significant, due to the low number of patients with RA in remission who actually had abnormal LV geometry in our study (table 1).

The link between RA disease activity and cardiovascular disease has been well established over the recent years.[4] Treatment with immunomodulation therapy has been shown to improve GLS in patients with RA,[5] and disease activity has been correlated to myocardial fibrosis and impaired strain on cardiac MRI.[6] Our finding of lower LV systolic function in patients with active RA is in line with these previous publications. It is likely that the higher RA disease activity in the Giollo study contributed to the differential findings between the studies. Furthermore, GLS may capture longitudinal function better than the simpler S′ used by Giollo *et al*. We, therefore, do not agree that the reported association between RA disease activity and lower LV systolic function solely reflects changes in LV geometry.

Table 1 Systolic left ventricular function in relation to left ventricular geometry and disease activity in patients with RA

	Normal geometry			Abnormal geometry		
	Active RA, SDAI >3.3 (n=55)	Remission RA, SDAI ≤3.3 (n=34)	p Value	Active RA, SDAI > 3.3 (n=23)	Remission RA, SDAI ≤3.3 (n=7)	p Value
GLS (%)	−19.2±2.9	−20.7±3.0	0.02	−18.1±3.4	−20.2±5.7	0.24
scMWS (%)	103±13	109±15	0.05	77±16	89±15	0.11

GLS, global longitudinal strain; RA, rheumatoid arthritis, scMWS, stress-corrected midwall shortening; SDAI, Simplified Disease Activity Index.

Helga Midtbø,[1,2] Anne Grete Semb,[3] Knut Matre,[2] Tore K Kvien,[3] Eva Gerdts[2]

[1]Department of Heart Disease, Haukeland University Hospital, Bergen, Norway
[2]Department of Clinical Science, University of Bergen, Bergen, Norway
[3]Preventive Cardio-Rheuma Clinic, Department of Rheumatology, Diakonhjemmet Hospital, Oslo, Norway

Correspondence to Dr Helga Midtbø, Department of Heart Disease, Haukeland University Hospital, Jonas Lies vei 65, 5021 Bergen, Norway; helgamit@online.no

Competing interests None.

Provenance and peer review Commissioned; internally peer reviewed.

To cite Midtbø H, Semb AG, Matre K, *et al*. *Ann Rheum Dis* Published Online First: [*please include* Day Month Year] doi:10.1136/annrheumdis-2016-210516

Received 22 September 2016
Accepted 26 September 2016

Ann Rheum Dis 2016;**0**:1. doi:10.1136/annrheumdis-2016-210516

REFERENCES

1 Giollo A, Cioffi G, Ognibeni F, *et al*. Disease activity and left ventricular systolic function in rheumatoid arthritis. *Ann Rheum Dis* 2016; Published Online First: 28 Sept 2016. doi:10.1136/annrheumdis-2016-210482
2 Midtbø H, Semb AG, Matre K, *et al*. Disease activity is associated with reduced left ventricular systolic myocardial function in patients with rheumatoid arthritis. *Ann Rheum Dis*; Published Online First 7 June 2016.
3 Cioffi G, Viapiana O, Ognibeni F, *et al*. Prevalence and factors related to inappropriately high left ventricular mass in patients with rheumatoid arthritis without overt cardiac disease. *J Hypertens* 2015;33:2141–9.
4 Solomon DH, Reed GW, Kremer JM, *et al*. Disease activity in rheumatoid arthritis and the risk of cardiovascular events. *Arthritis Rheumatol* 2015;67:1449–55.
5 Ikonomidis I, Tzortzis S, Andreadou I, *et al*. Increased benefit of interleukin-1 inhibition on vascular function, myocardial deformation, and twisting in patients with coronary artery disease and coexisting rheumatoid arthritis. *Circ Cardiovasc Imaging* 2014;7:619–28.
6 Ntusi NA, Piechnik SK, Francis JM, *et al*. Diffuse myocardial fibrosis and inflammation in rheumatoid arthritis: insights from CMR T1 mapping. *JACC Cardiovasc Imaging* 2015;8:526–36.