Isometric muscle strength is an indicator of self-reported physical functional disability in patients with rheumatoid arthritis

Stucki, G ; Bruhlmann, P ; Stucki, S ; Michel, B A

Abstract: The objective of our study was to examine (1) whether isometric muscle strength contributes to the explanation of the physical functional disability of a rheumatoid arthritis (RA) patient population after accounting for other disease parameters and demographic variables and (2) whether change in isometric muscle strength is an indicator of change in physical functional disability. Sixty-five consecutive patients fulfilling the American Rheumatism Association 1987 revised criteria for RA were included in the study. Isometric muscle strength was measured with a validated Muscle Strength Index (MSI) calculated as the mean score of standardized isometric extension and flexion strength of the knee and elbow joints. Physical functional disability was measured with the physical dimension of the Health Assessment Questionnaire (HAQ). For 56 patients, we could obtain 1 yr follow-up data. Cross-sectionally, there was a significant correlation ($r = -0.51$, $P < 0.01$) between the MSI and the HAQ. Muscle strength remained a significant determinant of the HAQ in multivariate analysis accounting for disease and demographic variables. Longitudinally, change in MSI explained additional variance of change in HAQ after accounting for change in pain, the most important correlate of change in HAQ

DOI: https://doi.org/10.1093/rheumatology/37.6.643

British Journal of Rheumatology 1998;**37:**643–648

ISOMETRIC MUSCLE STRENGTH IS AN INDICATOR OF SELF-REPORTED PHYSICAL FUNCTIONAL DISABILITY IN PATIENTS WITH RHEUMATOID ARTHRITIS

G. STUCKI, P. BRÜHLMANN, S. STUCKI and B. A. MICHEL

Department of Rheumatology and Physical Medicine, University Hospital, Zurich, Switzerland

SUMMARY

The objective of our study was to examine (1) whether isometric muscle strength contributes to the explanation of the physical functional disability of a rheumatoid arthritis (RA) patient population after accounting for other disease parameters and demographic variables and (2) whether change in isometric muscle strength is an indicator of change in physical functional disability. Sixty-five consecutive patients fulfilling the American Rheumatism Association 1987 revised criteria for RA were included in the study. Isometric muscle strength was measured with a validated Muscle Strength Index (MSI) calculated as the mean score of standardized isometric extension and flexion strength of the knee and elbow joints. Physical functional disability was measured with the physical dimension of the Health Assessment Questionnaire (HAQ). For 56 patients, we could obtain 1 yr follow-up data. Cross-sectionally, there was a significant correlation ($r = -0.51$, $P < 0.01$) between the MSI and the HAQ. Muscle strength remained a significant determinant of the HAQ in multivariate analysis accounting for disease and demographic variables. Longitudinally, change in MSI explained additional variance of change in HAQ after accounting for change in pain, the most important correlate of change in HAQ.

K : Muscle weakness, Rheumatoid arthritis, Health status.

I understanding and reliable measurement of the determinants of physical, mental and social health are crucial for a rational choice of therapy and evaluation of treatment e ectiveness. The importance of muscle weakness to disability in patients with rheumatoid arthritis (RA) has long been recognized [1–4] and controlled exercise to maintain or increase muscle strength is considered a cornerstone of comprehensive treatment in RA [5–7]. However, there are little quantitative data on the relationship of muscle weakness and physical functional disability [6, 8, 9].

There are many methods for how best to assess muscle strength [7]. Although there are undoubtedly more sophisticated methods, we used simple isometric testing with a pull-gauge which is reliable, simple and relatively cheap, and which may thus prove helpful not only in research, but in clinical practice as well [10]. Physical functional disability in RA may be measured with a variety of psychometrically sound health status instruments. We chose to use the Health Assessment Questionnaire (HAQ) [11–13], a disease-specific instrument which comprehensively addresses physical function in patients with RA, and which has been widely used in clinical studies and clinical practice [12].

The objective of our study was to examine whether isometric muscle strength contributes to the explanation of self-reported physical functional disability as measured with the HAQ.

The specific aims were to examine (1) whether isometric muscle strength contributes to the explanation of the physical disability of a RA patient population after accounting for other disease parameters and demographic variables and (2) whether change in isometric muscle strength is an indicator of change in physical functional disability.

Submitted 4 July 1997; revised version accepted 12 December 1997.

Correspondence to: G. Stucki, Rheumaklinik und Institut für Physikalische Medizin, Universitätsspital, Gloriastrasse 25, 8091 Zurich, Switzerland.

METHODS

Patients

Consecutive patients fulfilling the American Rheumatism Association 1987 revised criteria for RA [14], attending the out-patient clinic from October to November 1992, were included in the study. After 1 yr, patients were re-examined. About half of our clinic patients are self-referred and about half are referred by general practitioners or specialists in private practice, and represent a wide spectrum with respect to physical functional disability. The distribution of the patients from our out-patient clinic by the new ACR functional class criteria [15, 16] and the median HAQ score is similar to the population of the validation study by the ACR subcommittee [15, 16].

Data collection procedures

At a regularly scheduled out-patient visit, patients were asked to complete the HAQ questionnaire [11, 13]. Afterwards, patients were evaluated clinically by a physician who was unaware of the HAQ score; laboratory tests were performed and radiographs taken. The clinical evaluation was performed by eight fellows in rheumatology with regular training and experience in standardized data collection. A trained single observer, unaware of both the HAQ score and results from the clinical evaluation, then performed the muscle strength measurements with a hand-held pull-gauge according to a standardized protocol [10].

Measures

Physical functional disability was measured with use of the physical dimension of the HAQ, a validated RA

health status questionnaire [11, 13]. The HAQ asks questions about function in dressing, arising, walking, hygiene, reaching, gripping and in other activities. The questionnaire is self-administered and takes ~5 min to complete [12].

Pain was measured on a numerical rating scale (NRS) ranging from 0 to 10 (with anchors: no pain; extreme pain) and morning sti ness was recorded in minutes.

Isometric muscle strength of flexion and extension of elbow and knee joints of both sides was recorded with a hand-held pull-gauge which has been shown to be a reproducible method (interobserver and intra-observer reliability 0.94) [10]. For each measurement, the patient was positioned in such a way as to exclude the e ect of gravity. A non-elastic band was connected to a pull-gauge with a continuous scale (range 0–50 kp; Model DPPH, Chatillon Inc., Greensboro, NC, USA). The observer kept the pull-gauge in a stable position against a bar, the examination table, or his body. The patient was instructed to increase muscle strength gradually to his/her limit. An overall Muscle Strength Index (MSI) was then calculated as the mean of the individual standardized scores [(strength observed/maximum sample strength) × 100] [10].

Disease-specific clinical findings recorded were swollen joint count and tender joint count. Laboratory assessment included erythrocyte sedimentation rate (ESR; Westergren), C-reactive protein (CRP), haemoglobin (HB), rheumatoid factor (Singer-Plotz titre) and creatine kinase (*N*-acetyl-cysteine, 37°C; upper normal values are 150 U/l for women and 270 U/l for men). Radiological assessment included radiographs of the hands and feet; radiological damage was graded according to the Larsen radiological scoring system [17].

Overall disease activity was measured with the Disease Activity Score, a validated index using the following algorithm: Disease Activity Score = 0.064565 (number of swollen joints) + sqrt (Ritchie score) + 0.33 ln (ESR) [18].

Cross-sectional analyses

In univariate analyses using Pearson's correlation coe cient, we examined the association of the MSI with physical functional disability as measured with the HAQ, and the association with demographic and disease parameters.

In linear regression analysis, we examined the importance of muscle strength as compared to other disease parameters associated with physical functional disability in RA. Covariates were selected based on known and hypothesized relationships with the MSI or the HAQ. Demographic variables included in the model were age, gender and body height. Disease-specific variables included the Disease Activity Score, morning sti ness and pain to control for disease activity, pathological anatomical destruction (Larsen score [17]), haemoglobin and disease duration to control for disease severity, and prednisone dose to control for a possible metabolic myopathy.

Longitudinal analyses

In univariate analyses using Pearson's correlation coe cient, we examined the association of change in the MSI with change in the HAQ, and the association with changes in demographic and disease parameters.

In linear regression analysis, we examined the importance of change in MSI as compared to change in other disease parameters associated with change in HAQ and/or change in MSI in univariate analysis.

Because the HAQ is an ordinal rather than an interval scale, linear regression analysis of change scores may not be appropriate [19, 20]. We therefore also performed a logistic regression analysis using the direction of change in HAQ score (improved or unchanged HAQ score *vs* worsened HAQ score) as the dependent and change (either absolute change or dichotomous) in MSI and disease parameters as independent variables.

RESULTS

Subjects

Sixty-five patients were included in the study, for 56 patients we could obtain 1 yr follow-up data. All patients were Caucasian. Demographic and clinical characteristics at baseline of the 65 patients with RA are shown in Tables I and II.

The mean HAQ score was 1.1 (0.8) which is comparable to values of other reported studies of patients with RA [11, 15]. The mean swollen joint count was 7.3 (7.2), whereas the mean Ritchie articular count

TABLE I
Baseline characteristics of the 65 patients with rheumatoid arthritis

	%
Gender	
Male	35.4
Female	64.6
Age group (yr)	
⩽50	27.7
51–60	15.4
61–70	30.8
>70	26.2
College or professional training	63
ACR functional class	
I	15.4
II	29.2
III	52.3
IV	3.1
Rheumatoid factor (Singer Plotz, $n = 64$)	
Negative	48.6
Positive	51.6
DMARD ($n = 59$)	
None	13
Methotrexate	27
Gold, i.m.	25
Azathioprine	9
Salazopyrin	9
-penicillamine	7
Others	10
Prednisone	
None	55
<7.5 mg	28
⩾7.5 mg	17
NSAID treatment	94

TABLE II
Correlation of disease-specific and demographic variables with the Health Assessment Questionnaire (HAQ) and the Muscle Strength Index (MSI)

Variable	Mean (. .)	Correlation with the HAQ†	Correlation with the MSI†
Muscle Strength Index (%)	41 (17.0)	−0.51**	na
Disease Activity Score (points)	3.72 (1.74)	0.57**	−0.29*
Morning sti ness (min)	37.9 (56.6)	0.52**	−0.09
Pain (NRS)	4.2 (2.4)	0.55**	−0.31*
Haemoglobin	12.9 (1.67)	−0.19	0.43**
Larsen score (0–40)	12.8 (8.6)	0.40**	−0.44**
Disease duration (yr)	8.1 (9.1)	0.22	− 0.27*
Height (cm)	165 (9.4)	−0.12	0.33
Age (yr)	59.3 (15.2)	−0.06	− 0.12
Prednisone (mg)	4.12 (6.15)	0.13	0.07

na, not applicable.
*$P < 0.05$; **$P < 0.01$.
†Pearson's correlation coe cient.

was 24.8 (22.0). The mean ESR was 23.1 (19.4). The mean Disease Activity Score was 3.7 (1.7), which is slightly higher than the 3.3 (1.2) observed in the RA population of a Dutch clinic from which the Disease Activity Score was derived [18]. The creatine kinase of all patients was within normal limits.

Cross-sectional analyses

The univariate relationship between the HAQ and the MSI, and the association with demographic and disease parameters, are shown in Table II. Age, gender and body height and haemoglobin, cortisone use and/or cortisone dosage were not significant correlates of the HAQ either in univariate or in multivariate analyses. Instead, the MSI, pain, morning sti ness, the Larsen score [17] and the components of the Disease Activity Score—swollen and tender joint count and ESR—were significant univariate correlates of the HAQ.

In the multivariate analysis of the determinants of the HAQ, the Disease Activity Score and the MSI were significant at $P < 0.01$, and explained 31 and 12% of the variance of the HAQ in a forward stepwise selection process. Morning sti ness explained an additional 10%. Pain, Larsen radiological score [17], disease duration, haemoglobin, age, gender and height did not explain significant additional variance. The model explained 61% of the total variance in HAQ scores (Table III).

The regression coe cient of a simple regression with HAQ as dependent variable and muscle strength as independent variable was 0.024. With inclusion of disease activity, morning sti ness and Larsen radiological score in the model, the parameter estimate changed in clinically meaningful steps to 0.012. About half of the variation in HAQ due to variation in muscle strength may thus be attributed to these disease variables.

Based on the parameter estimates of our model, the predicted di erence in the HAQ score between a patient having 90% of observed MSI *vs* a patient with 10% is 0.96 HAQ points, which is considered a large di erence [21]. The smallest clinically important di erence in HAQ scores may be defined as 0.17 HAQ points [21]. Hence, HAQ scores needed to di er by 0.17 units for patients on average to stop rating themselves as the same and start rating themselves as somewhat better than other patients in one-on-one conversations. This di erence corresponds to a di erence in muscle strength of 14 percentage units. For example, a patient with a mean population strength of 41% and a patient with a strength of 54% would di er meaningfully in their physical functional disability based on this model. This is after controlling for all other potentially confounding variables in the model whose values are assumed to be equal for the purpose of this comparison.

Longitudinal analyses

Longitudinally, the univariate relationships between change in HAQ, change in MSI and change in disease parameters are shown in Table IV.

TABLE III
Parameter estimates of the regression model. The dependent variable is physical disability measured with the Health Assessment Score (HAQ)

Variable	Parameter estimate	95% Confidence interval	*P* value	Partial r^2†
Intercept	1.55	−2.71; 5.81	0.47	–
Disease Activity Score (points)	0.143	0.031; 0.255	0.01	0.31
Muscle Strength Index (%)	−0.012	−0.022; −0.002	0.03	0.12
Morning sti ness (min)	0.004	0.001; 0.007	0.02	0.10

†Partial r^2 values from the forward selection process. Model $r^2 = 0.61$; *F*-test for the model: <0.01.
Pain, haemoglobin, Larsen radiological score, disease duration, age, female gender, height and prednisone dose were not significant and each contributed <1% to the explanation of HAQ and are not shown in the table.

TABLE IV
Correlation of change in disease-specific and demographic variables with change in the Health Assessment Questionnaire (HAQ) and change in the Muscle Strength Index (MSI) over a 1 yr period

Variable	Correlation with the HAQ†	Correlation with the MSI†
Muscle Strength Index (%)	−0.36**	na
Modified Disease Activity Score (points)	−0.01	−0.15
Morning sti ness (min)	0.19	−0.37**
Pain (0–10)	0.41**	−0.15
Larsen score (0–40)	−0.10	0.06
Haemoglobin (mg/dl)	−0.17	0.31*

na, not applicable.
*$P < 0.05$; **$P < 0.01$.
†Pearson's correlation coe cient.

In the linear regression model with change in HAQ score as dependent variable, change in MSI as independent variable, and change in haemoglobin, morning sti ness and pain as covariates, pain was a significant covariate ($P < 0.05$), whereas change in MSI did not reach statistical significance ($P = 0.08$). However, in logistic regression analysis with change in HAQ as dichotomous dependent variable, change in MSI (either in terms of absolute change or as dichotomous variable) remained significant after accounting for pain ($P < 0.05$), haemoglobin and morning sti ness (which were not significant). In dichotomous analysis, the odds ratio for pain was 7.4 (CI 2.0; 29.1) and 5.3 (CI 1.5; 19.8) for the MSI. In other words, patients who after 1 yr had worse isometric muscle strength as measured with the MSI were 5.6 times more likely to report worse physical functional status compared to patients with unchanged or improved MSI.

The parameter estimate of the linear model was −0.009, which is slightly smaller than in the cross-sectional analysis (0.012). Based on this estimate, the smallest clinically important di erence in HAQ scores (which has been defined as 0.17 HAQ points [21]) corresponds to a di erence in muscle strength of 19 percentage units, which is slightly higher than the estimate from cross-sectional analysis (14 percentage units).

DISCUSSION

Because of the financial constraints of our health care systems, and growing awareness and commitment of the medical society to continuous quality improvement, documentation of treatment e ectiveness either for health care programmes or individual patients is becoming increasingly important. Patient-derived measures of outcome, such as physical, mental and psychological disability, may be considered gold standards for the evaluation of treatment e ectiveness in RA [11]. From a practical viewpoint, it is, however, insu cient to rely solely on patient outcomes. Patient outcomes usually have many causes and only with clinical measures may one identify potentially treatable mechanisms and estimate their importance to patient outcomes. When monitoring treatment e ectiveness in individual patients, change may first occur in clinical parameters, whereas translation of improved physical and psychological capacities into patient-relevant improvement in health status often involves complex behavioural changes, experimentation and training, which may take considerable time. When monitoring in-patient rehabilitation, health status measures which ask about patients' perception of disability at home are not useful. Under these circumstances, simple-to-measure and reliable clinical indicators of change in self-perceived disability may be of considerable value. Based on the results of our study, isometric muscle strength, as measured with the MSI [10], may serve as an indicator of change in self-perceived physical functional disability. Isometric muscle strength may also be useful for a more refined documentation and stratification of patient subgroups for clinical, health services and epidemiological studies.

While we found a relationship between muscle strength and physical functional disability both in cross-sectional and longitudinal analyses, and being aware of the exercise study in which grip strength was associated with a Dutch version of the HAQ at baseline [22], improvement in isokinetic knee extensor and flexor strength did not translate into improvement of the Dutch HAQ in the exercise study [23]. Possible reasons are the relative insensitivity of the HAQ to change [22], particularly in patients with mild disabling disease [24]. Since in our study about half of our patients had worse HAQ scores, while the other half had unchanged or improved HAQ scores, the ceiling e ect may have played a lesser role. Also, it seems less likely that improved strength translates into physical functional gains than for a loss of strength to translate into worsening physical function. It was also argued that the benefit of exercise will be shown only in the long term, e.g. over a period of a year, as in our study. To examine change in strength in relation to physical functional ability during a flare situation could elucidate the importance of time and/or worsening of the condition as opposed to improvement.

Whereas isometric muscle strength as measured with the MSI is an indicator of physical functional disability, we can only hypothesize about the mechanism which leads to muscle weakness and/or physical functional disability. Cross-sectionally, disease activity seems to play an important role. In other words, patients di er from each other in their muscle strength and physical

functional disability according to disease activity. Possible mechanisms include reflex inhibition due to synovitis [25]. In our population, steroid use, which may be the most important cause of metabolic myopathy in RA [26–28], was not significantly related to muscle strength or physical disability. We could not meaningfully study the e ect of potentially myopathy-inducing slow-acting anti-inflammatory drugs because of the limited sample size. However, only few patients were treated with known myopathic disease-modifying anti-rheumatic drugs (DMARDs) (four patients did take -penicillamine) and no patient was treated with possibly myopathic non-steroidal anti-inflammatory drugs (NSAIDs) (phenylbutazone, aspirin or niflumin acid) [29]. Also, we found no clinical signs or laboratory findings indicating myositis, which has been rarely described in patients with RA [30, 31]. Peripheral neuropathies caused by amyloidosis or vasculitis have been described in RA [32]. However, they are rare and are not likely to be of importance. Entrapment neuropathies due to synovial tissue and/or joint deformity are seen in RA, particularly in the ankle and wrist joint, but only occasionally in the elbow or knee where we measured muscle strength. We did not directly account for altered biomechanics from subluxed joints. However, we did examine radiological destruction, which was a univariate correlate of both the MSI and HAQ.

Because these disease-related mechanisms explained only half of the relationship between muscle strength and HAQ in cross-sectional analysis and, with the exception of pain, none in longitudinal analysis, other pathogenetic mechanisms must be of importance. Most likely, variation in disuse [6] due to di erent behaviour is important. Variation in muscle strength due to behavioural factors such as physical training has been shown to be of importance [9, 8], and these are potentially amenable to therapy. A limitation of our study is that we did not measure attitudes and behaviour. However, it is interesting to note that a small increase in muscle strength of 14–19 percentage units, which is a realistic goal for an exercise therapy, would translate into 0.17 HAQ points, which patients considered the minimal clinically meaningful di erence comparing their own disability to that of others [21].

In conclusion, isometric muscle strength measured with a pull-gauge and integration of measurements into an index with cardinal properties is a cross-sectional and longitudinal correlate of self-perceived physical functional status. It may thus be used as an indicator to document the e ectiveness of rehabilitative interventions in RA in clinical and health services research or clinical practice.

A

We thank Bea Banks for help with the data collection and Leanne Pobjoy for help in preparing the manuscript, and the physicians from the out-patient clinic, Department of Rheumatology, University Hospital, Zurich, for their help with the data collection.

R

1. Tiselius P. Studies on joint temperature, joint sti ness and muscle weakness in rheumatoid arthritis: an experimental and clinical investigation. Acta Rheumatol Scand 1969;suppl. 14:70–98.
2. Ekblom B, Lövgren O, Alderin M, Fridström M, Sätterström G. Physical performance in patients with rheumatoid arthritis. Scand J Rheumatol 1974;3:121–5.
3. Nordesjö LO, Nordgren B, Wigren A, Kolstad K. Isometric strength and endurance in patients with severe rheumatoid arthritis or osteoarthritis in knee joints. Scand J Rheumatol 1983;12:152–6.
4. Ekdahl C, Andersson SI, Svensson B. Muscle function of the lower extremities in rheumatoid arthritis and osteoarthritis: A descriptive study of patients in a primary health care district. J Clin Epidemiol 1989;42:947–54.
5. Hicks JE. Exercise in patients with inflammatory arthritis and connective tissue disease. Rheum Dis Clin North Am 1990;16:845–70.
6. Jokl P. Prevention of disuse muscle atrophy in chronic arthritis. Rheum Dis Clin North Am 1990;16:837–44.
7. Herbison GJ, Ditunno JF, Mazher Jaweed M. Muscle atrophy in rheumatoid arthritis. J Rheumatol 1987;suppl. 15 14:78–81.
8. Nordemar R. Physical training in rheumatoid arthritis: A controlled long-term study. Scand J Rheumatol 1981;10:25–30.
9. Stenstrom CH. Home exercise in rheumatoid arthritis functional class II: Goal setting versus pain attention. J Rheumatol 1994;21:627–34.
10. Stucki G, Schönbächler J, Brühlmann P, Mariacher S, Stoll T, Michel BA. Does a muscle strength index provide complementary information to traditional disease activity variables in patients with rheumatoid arthritis? J Rheumatol 1994;21:2200–5.
11. Fries JF, Spitz PW, Kraines RG, Holman HR. Measurement of patient outcome in arthritis. Arthritis Rheum 1980;23:137–45.
12. Ramey DR, Raynauld JP, Fries JF. The Health Assessment Questionnaire 1992. Status and review. Arthritis Care Res 1992;5:119–29.
13. Brühlmann P, Stucki G, Michel BA. Evaluation of a German version of the physical dimension of the health assessment questionnaire in patients with rheumatoid arthritis. J Rheumatol 1994;21:1245–9.
14. Arnett FC, Edworthy SM, Bloch DA *et al.* The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. Arthritis Rheum 1988;31:315–24.
15. Hochberg MC, Chang RW, Dwosh I, Lindsey S, Pincus T, Wolfe F. The American College of Rheumatology 1991 revised criteria for the classification of global functional status in rheumatoid arthritis. Arthritis Rheum 1992;35:498–502.
16. Stucki G, Stoll T, Brühlmann P, Michel BA. Construct validation of the ACR 1991 revised criteria for global functional status in rheumatoid arthritis. Clin Exp Rheumatol 1995;13:349–52.
17. Larsen A, Dale K, Eek M. Radiographic evaluation of rheumatoid arthritis and related conditions by standard reference films. Acta Radiol Diagn 1977;18:481–91.
18. van der Heijde DMFM, van't Hof MA, van Riel PLCM, van Leeuwen MA, van Rijswijk MH, van de Putte LBA. Validity of single variables and composite indices for measuring disease activity in rheumatoid arthritis. Ann Rheum Dis 1992;51:177–81.

19. Silverstein B, Fisher WP, Kilgore KM, Harley JP, Harvey RF. Applying psychometric criteria to functional assessment in medical rehabilitation: II. Defining interval measures. Arch Phys Med Rehabil 1992;73:507–18.
20. Merbitz C, Morris J, Grip JC. Ordinal scales and foundations of misinference. Arch Phys Med Rehabil 1989;70:308–12.
21. Redelmeier DA, Lorig K. Assessing the clinical importance of symptomatic improvements. An illustration in rheumatology. Arch Intern Med 1993;153:1337–42.
22. van den Ende CHM, Breedveld FC, Dijkmans BAC, Hazes JMW. The limited value of the Health Assessment Questionnaire as an outcome measure in short term exercise trials. J Rheumatol 1997;24:1972–7.
23. van den Ende CHM, Hazes JMW, le Cessie S, Mulder WJ, Belfor DG, Breedveld FC *et al.* Comparison of high and low intensity training in well controlled rheumatoid arthritis. Results of a randomised clinical trial. Ann Rheum Dis 1996;55:798–805.
24. Stucki G, Stucki S, Brühlmann P, Michel BA. Ceiling e ects of the Health Assessment Questionnaire and its modified version in some ambulatory rheumatoid arthritis patients. Ann Rheum Dis 1995;54:461–5.
25. Stokes M, Young A. The contribution of reflex inhibition to arthrogenous muscle weakness. Clin Sci 1984;67:7–14.
26. Edström L, Nordemar R. Di erential changes in type I and type II muscle fibres in rheumatoid arthritis. Scand J Rheumatol 1974;3:55–60.
27. Wróbleski R, Nordemar R. Ultrastructural and histochemical studies of muscle in rheumatoid arthritis. Scand J Rheumatol 1975;4:197–204.
28. Moritz U. Electromyographic studies in adult rheumatoid arthritis. Acta Rheumatol Scand 1963;suppl. 6:1–123.
29. Zuckner J. Drug-induced myopathies. Semin Arthritis Rheum 1990;19:259–68.
30. Halla JT, Koopman WJ, Fallahi S, Oh SJ, Gay RE, Schrohenloher RE. Rheumatoid myositis. Clinical and histologic features and possible pathogenesis. Arthritis Rheum 1984;27:737–43.
31. Wortmann RL. Inflammatory diseases of muscle. In: Kelley WN, ed. Textbook of rheumatology, 4th edn. Philadelphia: WB Saunders Co., 1993:1159–88.
32. Chang DJ, Paget SA. Neurologic complications of rheumatoid arthritis. Rheum Dis Clin North Am 1993;19:955–73.