Table 1. Age- and sex-adjusted baseline characteristics according to serum CRP levels (n = 775)

	CRP tertiles, mg/l			CRP 1.01–9.92 mg/l	p for trend[a]
	0.05–0.28	0.28–0.51	0.51–1.00		
Participants	181	182	182	230	–
Age, years[b]	75.51 (74.82–76.2)	76.19 (75.51–76.88)	75.96 (75.28–76.65)	75.68 (75.06–76.29)	0.36
Males, %	38.1	44.0	45.1	44.4	0.44
BMI[b]	22.05 (21.60–22.50)	23.33 (22.88–23.77)	24.37 (23.93–24.82)	24.86 (24.46–25.26)	<0.0001
Albumin, g/dl[b]	4.33 (4.29–4.37)	4.36 (4.32–4.39)	4.36 (4.32–4.39)	4.32 (4.29–4.36)	0.31
Hypercholesterolemia, %	28.2	33.0	37.4	43.5	<0.001
Low HDL cholesterol (≤ 40 mg/dl), %	7.7	7.7	13.2	14.8	0.02
History of CHD, %	6.6	10.4	11.0	10.4	0.44
Hypertension, %	54.7	68.1	72.0	75.2	<0.001
Diabetes, %	3.9	8.2	11.0	12.2	0.01
History of cancer, %	6.6	7.7	2.8	7.4	0.68
Depressive symptoms (GDS ≥ 11), %	35.9	25.3	30.2	28.7	0.58
Cognitively impaired (MMSE < 26), %	18.2	13.2	18.7	15.2	0.77
Smoker					
Current smoker, %	12.2	9.9	11.5	17.4	0.03
Ex-smoker, %	20.4	33.0	33.5	32.6	0.17
Nonsmoker, %	67.4	57.1	55.0	50.0	<0.001
Drinking					
Current drinker, %	44.3	45.6	42.3	41.4	0.26
Ex-drinker, %	8.6	13.0	12.0	14.1	0.21
Nondrinker, %	47.1	41.4	45.7	44.5	0.70
Use of NSAIDs, %	9.9	13.2	13.7	11.7	0.90
Use of statin drugs, %	13.8	12.6	17.0	14.8	0.69
Use of aspirin drugs, %	4.4	9.9	6.6	11.3	0.04
Use of ACE inhibitors, %	5.0	6.0	7.7	8.3	0.22
n-3 PUFA intake, g/day × 2,000 kcal	3.42 (3.27–3.58)	3.38 (3.23–3.53)	3.52 (3.36–3.67)	3.26 (3.13–3.40)	0.41

ACE = Angiotensin-converting enzyme.

[a] Analysis of covariance (age, albumin and BMI) or multiple logistic regression adjusted for age and sex where appropriate.

[b] Adjusted least squares mean (95% CI).

ates. When we calculated log-transformed CRP, 1.0 was added [CRP value (mg/l) + 1] before transformation. A significant difference was defined as $p < 0.05$. All statistical analyses were performed using Statistical Analysis System 9.1 edition for Windows (SAS Institute Inc., Cary, N.C., USA).

Results

In this study, the subjects whose serum CRP concentrations were higher than 10.0 mg/l were excluded. The median value (interquartile range) of CRP was 0.55 (0.29–1.20) mg/l.

Age- and sex-adjusted baseline characteristics according to the tertiles of a serum CRP concentration <1.0 mg/l or >1.0 mg/l are presented in table 1. Mean BMI was significantly higher across CRP levels (p for trend <0.0001). The prevalence of hypercholesterolemia, low HDL cholesterol, hypertension, diabetes and the use of aspirin drugs were significantly larger in the higher CRP levels (p for trend ≤0.04). The proportion of nonsmokers was significantly lower in the higher CRP levels (p for trend <0.001). In contrast, the proportion of current smokers was significantly higher in the higher CRP levels (p for trend = 0.03). Otherwise, no significant difference was observed among CRP levels (p for trend ≥0.17).

Table 2 shows the adjusted association between CRP level and physical performance. After adjustment for potential confounding factors, the significant inverse relation of the CRP level with leg power was observed in all subjects. Similarly, although not statistically significant, increasing CRP levels tended to relate inversely to 10-meter walk at maximum speed (p for trend = 0.10). For subjects who had CRP <1.0 mg/l, the CRP levels showed a significant inverse relationship with 10-meter walk at maximum speed (p for trend = 0.03) and leg power (p for

Table 2. Adjusted C-reactive protein levels in relation to physical activity

	CRP tertiles, mg/l			CRP 1.01–9.92 mg/l	p for trend	p for trend[b]
	0.05–0.28	0.28–0.51	0.51–1.00			
Participants	181	182	182	230	–	–
Ten-meter walk at max. speed, m/s						
Age-, sex-adjusted	1.80 (1.76–1.85)	1.75 (1.71–1.79)	1.73 (1.68–1.77)	1.69 (1.65–1.72)	<0.01	<0.01
Multiple adjusted[a]	1.44 (1.23–1.66)	1.40 (1.19–1.61)	1.39 (1.18–1.60)	1.36 (1.15–1.57)	0.10	0.03
Leg power, W/kg						
Age-, sex-adjusted	11.49 (11.03–11.95)	11.07 (10.61–11.52)	10.35 (9.90–10.81)	10.32 (9.91–10.73)	<0.001	<0.001
Multiple adjusted[a]	9.23 (6.92–11.53)	9.05 (6.76–11.34)	8.57 (6.32–10.81)	8.64 (6.37–10.91)	0.04	0.02
Timed up and go, s						
Age-, sex-adjusted	9.29 (8.98–9.59)	9.22 (8.92–9.52)	9.10 (8.8–9.41)	9.56 (9.29–9.83)	0.41	0.42
Multiple adjusted[a]	11.92 (10.37–13.47)	11.80 (10.26–13.35)	11.57 (10.05–13.08)	11.99 (10.46–13.52)	0.11	0.16

Variables are presented as least squares means (95% CI).
[a] Adjusted for age, sex, body mass index, serum albumin concentration, hypercholesterolemia (nonstatin drugs), low high-density lipoprotein-cholesterol (≤40 mg/dl), history of coronary heart diseases, history of cancer, hypertension, diabetes, depressive symptoms, impaired cognitive function, smoking habits/history, PA, use of nonsteroidal anti-inflammatory drugs, statin drugs, aspirin, angiotensin-converting enzyme inhibitors and tertile of n-3 polyunsaturated fatty acids intake.
[b] Only for subjects who had a C-reactive protein <1.0 mg/l.

trend = 0.02). No relation was found between the tertiles of serum CRP concentration <1.0 mg/l, >1.0 mg/l and timed up and go test in all models (p for trend ≥0.11). Furthermore, because the test for interaction between CRP levels and sex was statistically significant (p for interaction for 10-meter walk at maximum speed: 0.02; p for interaction for leg power: 0.04), we conducted stratified analysis for sex. Although not statistically significant, increasing CRP levels had a stronger relationship with 10-meter walk at maximum speed (p for trend = 0.23) and leg power (p for trend = 0.16) in men as compared to women (p for trend = 0.60 and 0.35, respectively).

The multiple regression model analysis also showed an inverse and significant relationship between log-transformed CRP and leg power (standard regression coefficient = –0.07, p = 0.03) after adjustment for covariates in table 2 in subjects who had CRP <1.0 mg/l. Although not statistically significant, log-transformed CRP was inversely related to 10-meter walk at maximum speed (standard regression coefficient = –0.06, p = 0.09). In contrast, no relation was found between log-transformed CRP and TUGT (standard regression coefficient = –0.05, p = 0.19).

Discussion

In this cross-sectional study, we examined the relationship between serum CRP concentrations and physical performance in an elderly Japanese population. We also examined the relationship between CRP and physical performance in subjects with a serum CRP concentration <1.0 mg/l. These results suggested that a lower serum CRP concentration is favorably associated with physical performance even within a very low range.

The comparisons of the various inflammatory markers, including soluble adhesion molecules (e.g. E-selectin, P-selectin, intracellular adhesion molecule-1, vascular cell adhesion molecule-1), cytokines (e.g. interleukin-1β, -6, -8, and -10 and tumor necrosis factor-α), acute phase reactants (e.g. fibrinogen, serum amyloid A protein and hs-CRP) and WBC count, favor CRP from the clinical chemistry perspective [27]. Although the detection of elevated levels of CRP in the serum is not specific for any particular disease, it is a useful indicator of inflammatory processes [42]. High-sensitivity CRP is the term applied to a test that detects serum CRP concentration at lower levels than previous generations of laboratory tests. The lower limit of detection is substantially lower and can therefore detect lower levels of inflammation. In Western countries, the concentrations of CRP are proposed to be <1.0 mg/l as low risk, 1.0–3.0 mg/l as intermediate risk, and >3.0 mg/l as high risk for CHD [27]. Nonetheless, the current results suggested that high CRP also is independently related to poorer physical performance in elderly populations who have a serum CRP concentration <1.0 mg/l.

Several epidemiological studies assessed the relationship between CRP and physical performance [9, 16–19].

Most of these studies reported an inverse relation between CRP and physical performance or disability. However, the level of serum CRP was remarkably higher in these studies compared to the present study. In three of these studies, hsCRP was not used [9, 16, 17]. Only two studies used hsCRP to assess the relationship between CRP and physical performance [18, 19]. McDermott et al. [18] reported that higher CRP levels were associated with lower physical performance among subjects with PAD but not among those without non-PAD. Another cross-sectional study used hsCRP to assess that the relationship between CRP and physical performance in a community-based elderly population aged ≥60 years, but in their study, several confounding factors such as use of NSAIDs [43], statin drugs [38], and PAD [18, 35] associated with CRP were not considered [19]. Moreover, although multiple linear regression was conducted to assess the natural-log-transformed CRP and physical performance in their study, it was not shown whether the CRP also is associated with physical performance even within a very low rang (CRP <1.0 mg/l). Compared with these studies, hsCRP was used in this study, and the median CRP value of 0.55 mg/l of our participants was lower. This population with low CRP and hsCRP measure gave us an opportunity to examine the relationship between a very low range of CRP and physical performance. In the current study, we found that the serum CRP concentration is inversely associated with physical performance, even within a very low range (CRP <1.0 mg/l). Therefore, maintaining lower CRP levels may be important in clinical or subclinical practice. However, whether reducing inflammatory status by use of a drug will lead to a reduced risk of dependency in old age remains to be an important research question.

Since the concentrations of CRP are proposed to be <1.0 mg/l as low risk, 1.0–3.0 mg/l as intermediate risk, and >3.0 mg/l as high risk for CHD [27], the current results have also shown that levels associated with risk of coronary artery disease may be less sensitive than muscular performance for those participants with CRP <1.0 mg/l.

Although the association of CRP and disability or mortality has been proven by a large number of studies [9, 16–19], a direct mechanistic impact of CRP on mortality or disability has to be regarded as still doubtful. The underlying processes may have certainly more impact on risk factors and functionality than CRP. For example, the BMI is known to relate to the length of life with disability before death or severity of disability [44]. Because the BMI has been strongly associated with the serum CRP levels [45], the present study suggests that one possible pathway by which the control of BMI may reduce these risks is through decreasing serum CRP levels. Further study is required to explore the mechanisms that are involved in the associations.

We did not find significant associations between CRP and TUGT. Since TUGT [31] was not a test concerning peak physical performance as compared to 10-meter maximum walk test and leg extension power, CRP may be sensitively associated with peak physical performance within a very low range.

This study had several limitations. First, since all assessment was carried out in a public facility, participants were sufficiently active and healthy. Therefore, our results may not represent an elderly general population. Second, since this study was a cross-sectional study, we were not able to infer causality from our results. Still, several prospective studies suggested that CRP was associated with a decline in physical performance [9, 16, 17]. Therefore, the current results may be reliable. Further, although compared to those without fractures, individuals with a hip, arm, or clinical spinal fracture have shown similar global declines in physical performance [46], the results were not adjusted for fracture status because of a lack of information.

In summary, a lower serum CRP concentration is favorably related to physical performance, even within a very low range. The findings suggest that maintaining CRP levels as low as possible may potentially maintain better physical performance.

Acknowledgments

References

1 Runge M, Rittweger J, Russo CR, Schiessl H, Felsenberg D: Is muscle power output a key factor in the age-related decline in physical performance? A comparison of muscle cross section, chair-rising test and jumping power. Clin Physiol Funct Imaging 2004;24:335–340.

2 Goodpaster BH, Park SW, Harris TB, Kritchevsky SB, Nevitt M, Schwartz AV, Simonsick EM, Tylavsky FA, Visser M, Newman AB: The loss of skeletal muscle strength, mass, and quality in older adults: the health, aging and body composition study. J Gerontol A Biol Sci Med Sci 2006;61:1059–1064.

3 Doherty TJ: The influence of aging and sex on skeletal muscle mass and strength. Curr Opin Clin Nutr Metab Care 2001;4:503–508.

4 Navarro A, Lopez-Cepero JM, Sanchez del Pino MJ: Skeletal muscle and aging. Front Biosci 2001;6:D26–D44.

5 Larsson L, Ramamurthy B: Aging-related changes in skeletal muscle. Mechanisms and interventions. Drugs Aging 2000;17:303–316.

6 Rosenberg IH, Miller JW: Nutritional factors in physical and cognitive functions of elderly people. Am J Clin Nutr 1992;55:1237S–1243S.

7 Visser M, Pahor M, Taaffe DR, Goodpaster BH, Simonsick EM, Newman AB, Nevitt M, Harris TB: Relationship of interleukin-6 and tumor necrosis factor-alpha with muscle mass and muscle strength in elderly men and women: the Health ABC Study. J Gerontol A Biol Sci Med Sci 2002;57:M326–M332.

8 Rantanen T, Guralnik JM, Foley D, Masaki K, Leveille S, Curb JD, White L: Midlife hand grip strength as a predictor of old age disability. JAMA 1999;281:558–560.

9 Taaffe DR, Harris TB, Ferrucci L, Rowe J, Seeman TE: Cross-sectional and prospective relationships of interleukin-6 and C-reactive protein with physical performance in elderly persons: MacArthur studies of successful aging. J Gerontol A Biol Sci Med Sci 2000;55:M709–M715.

10 Ferrucci L, Harris TB, Guralnik JM, Tracy RP, Corti MC, Cohen HJ, Penninx B, Pahor M, Wallace R, Havlik RJ: Serum IL-6 level and the development of disability in older persons. J Am Geriatr Soc 1999;47:639–646.

11 Spate U, Schulze PC: Proinflammatory cytokines and skeletal muscle. Curr Opin Clin Nutr Metab Care 2004;7:265–269.

12 Kuller LH: Serum levels of IL-6 and development of disability in older persons. J Am Geriatr Soc 1999;47:755–756.

13 Volanakis JE: Human C-reactive protein: expression, structure, and function. Mol Immunol 2001;38:189–197.

14 Claus DR, Osmand AP, Gewurz H: Radioimmunoassay of human C-reactive protein and levels in normal sera. J Lab Clin Med 1976;87:120–128.

15 Burnett JR, Watts GF, Vasikaran SD: C-reactive protein: a new cardiovascular risk factor? Med J Aust 2000;173:117–118.

16 Schaap LA, Pluijm SM, Deeg DJ, Visser M: Inflammatory markers and loss of muscle mass (sarcopenia) and strength. Am J Med 2006;119:526.e9–526.e517.

17 Cesari M, Penninx BW, Pahor M, Lauretani F, Corsi AM, Rhys Williams G, Guralnik JM, Ferrucci L: Inflammatory markers and physical performance in older persons: the InCHIANTI study. J Gerontol A Biol Sci Med Sci 2004;59:242–248.

18 McDermott MM, Greenland P, Green D, Guralnik JM, Criqui MH, Liu K, Chan C, Pearce WH, Taylor L, Ridker PM, Schneider JR, Martin G, Rifai N, Quann M, Fornage M: D-dimer, inflammatory markers, and lower extremity functioning in patients with and without peripheral arterial disease. Circulation 2003;107:3191–3198.

19 Kuo HK, Bean JF, Yen CJ, Leveille SG: Linking C-reactive protein to late-life disability in the National Health and Nutrition Examination Survey (NHANES) 1999–2002. J Gerontol A Biol Sci Med Sci 2006;61:380–387.

20 Woloshin S, Schwartz LM: Distribution of C-reactive protein values in the United States. N Engl J Med 2005;352:1611–1613.

21 Sekikawa A, Ueshima H, Zaky WR, Kadowaki T, Edmundowicz D, Okamura T, Sutton-Tyrrell K, Nakamura Y, Egawa K, Kanda H, Kashiwagi A, Kita Y, Maegawa H, Mitsunami K, Murata K, Nishio Y, Tamaki S, Ueno Y, Kuller LH: Much lower prevalence of coronary calcium detected by electron-beam computed tomography among men aged 40–49 in Japan than in the US, despite a less favorable profile of major risk factors. Int J Epidemiol 2005;34:173–179.

22 Saijo Y, Kiyota N, Kawasaki Y, Miyazaki Y, Kashimura J, Fukuda M, Kishi R: Relationship between C-reactive protein and visceral adipose tissue in healthy Japanese subjects. Diabetes Obes Metab 2004;6:249–258.

23 Yamada S, Gotoh T, Nakashima Y, Kayaba K, Ishikawa S, Nago N, Nakamura Y, Itoh Y, Kajii E: Distribution of serum C-reactive protein and its association with atherosclerotic risk factors in a Japanese population: Jichi Medical School Cohort Study. Am J Epidemiol 2001;153:1183–1190.

24 Khera A, McGuire DK, Murphy SA, Stanek HG, Das SR, Vongpatanasin W, Wians FH Jr, Grundy SM, de Lemos JA: Race and gender differences in C-reactive protein levels. J Am Coll Cardiol 2005;46:464–469.

25 Anand SS, Razak F, Yi Q, Davis B, Jacobs R, Vuksan V, Lonn E, Teo K, McQueen M, Yusuf S: C-reactive protein as a screening test for cardiovascular risk in a multiethnic population. Arterioscler Thromb Vasc Biol 2004;24:1509–1515.

26 Niu K, Hozawa A, Kuriyama S, Ohmori-Matsuda K, Shimazu T, Nakaya N, Fujita K, Tsuji I, Nagatomi R: Dietary long-chain n-3 fatty acids of marine origin and serum C-reactive protein concentrations are associated in a population with a diet rich in marine products. Am J Clin Nutr 2006;84:223–229.

27 Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO 3rd, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL, Rifai N, Smith SC Jr, Taubert K, Tracy RP, Vinicor F: Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 2003;107:499–511.

28 Pepys MB, Hirschfield GM: C-reactive protein: a critical update. J Clin Invest 2003;111:1805–1812.

29 Ledue TB, Weiner DL, Sipe JD, Poulin SE, Collins MF, Rifai N: Analytical evaluation of particle-enhanced immunonephelometric assays for C-reactive protein, serum amyloid A and mannose-binding protein in human serum. Ann Clin Biochem 1998;35:745–753.

30 Harada ND, Chiu V, Stewart AL: Mobility-related function in older adults: assessment with a 6-minute walk test. Arch Phys Med Rehabil 1999;80:837–841.

31 Mathias S, Nayak US, Isaacs B: Balance in elderly patients: the 'get-up and go' test. Arch Phys Med Rehabil 1986;67:387–389.

32 Yamashina A, Tomiyama H, Takeda K, Tsuda H, Arai T, Hirose K, Koji Y, Hori S, Yamamoto Y: Validity, reproducibility, and clinical significance of noninvasive brachial-ankle pulse wave velocity measurement. Hypertens Res 2002;25:359–364.

33 Niino N, Imaizumi T, Kawakami N: A Japanese translation of the Geriatric Depression Scale. Clin Gerontol 1991;10:85–87.

34 Folstein MF, Folstein SE, McHugh PR: 'Mini-mental state'. A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975;12:189–198.

35 Hozawa A, Ohmori K, Kuriyama S, Shimazu T, Niu K, Watando A, Ebihara S, Matsui T, Ichiki M, Nagatomi R, Sasaki H, Tsuji I: C-reactive protein and peripheral artery disease among Japanese elderly: the Tsurugaya Project. Hypertens Res 2004;27:955–961.

36 King DE, Carek P, Mainous AG 3rd, Pearson WS: Inflammatory markers and exercise: differences related to exercise type. Med Sci Sports Exerc 2003;35:575–581.

37 Chobanian A, Bakris G, Black H: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report. JAMA 2003;289:2560–2571.

38 Ridker PM, Rifai N, Lowenthal SP: Rapid reduction in C-reactive protein with cerivastatin among 785 patients with primary hypercholesterolemia. Circulation 2001;103: 1191–1193.

39 Niu K, Hozawa A, Fujita K, Ohmori K, Okutsu M, Kuriyama S, Tsuji I, Nagatomi R: Influence of leisure-time physical activity on the relationship between C-reactive protein and hypertension in a community-based elderly population of Japan: the Tsurugaya project. Hypertens Res 2005;28:747–754.

40 Brink T, Yesavage J, Lum O, Heersema P, Adey M, Rose T: Screening tests for geriatric depression. Clin Gerontol 1982;1:37–44.

41 Siu AL: Screening for dementia and investigating its causes. Ann Intern Med 1991;115: 122–132.

42 Morley JJ, Kushner I: Serum C-reactive protein levels in disease. Ann N Y Acad Sci 1982; 389:406–418.

43 Chenevard R, Hurlimann D, Bechir M, Enseleit F, Spieker L, Hermann M, Riesen W, Gay S, Gay RE, Neidhart M, Michel B, Luscher TF, Noll G, Ruschitzka F: Selective COX-2 inhibition improves endothelial function in coronary artery disease. Circulation 2003;107:405–409.

44 Ohmori K, Kuriyama S, Hozawa A, Ohkubo T, Tsubono Y, Tsuji I: Modifiable factors for the length of life with disability before death: mortality retrospective study in Japan. Gerontology 2005;51:186–191.

45 Pieroni L, Bastard JP, Piton A, Khalil L, Hainque B, Jardel C: Interpretation of circulating C-reactive protein levels in adults: body mass index and gender are a must. Diabetes Metab 2003;29:133–138.

46 Greendale GA, DeAmicis TA, Bucur A, Bretsky P, Rowe JW, Reuben DB, Seeman T: A prospective study of the effect of fracture on measured physical performance: results from the MacArthur Study–MAC. J Am Geriatr Soc 2000;48:546–549.

6. Paauw A, Verhoef J, Fluit AC, et al. Failure to control an out-break of qnrA1-positive multidrug-resistant Enterobacter cloacae infection despite adequate implementation of recommended infection control measures. J Clin Microbiol 2007;45:1420–1425.

7. Jarlier V, Nicolas MH, Fournier G, Philippon A. Extended-broad-spectrum β-lactamases conferring transferable resistance to newer β-lactam agents in Enterobacteriaceae: hospital prevalence and susceptibility patterns. Rev Infect Dis 1988;10:867–878.

8. Touati A, Benallaoua S, Djoudi F, Madoux J, Brasme L, De Champs C. Characterization of CTX-M-15 producing Klebsiella pneumoniae and E. coli strains isolated from hospital environment in Algeria. Microb Drug Resist 2007;13:85–89.

A. Touati[a],*
L. Brasme[b]
S. Benallaoua[a]
J. Madoux[b]
A. Gharout[a]
C. de Champs[b]
[a]Laboratoire de microbiologie appliquée, FSNV, Université A/MIRA de Béjaia, Algérie
[b]Laboratoire de Bactériologie-Virologie-Hygiène Hospitalière, CHU Reims, Hôpital Robert Debre, Reims, France
E-mail address: ziz1999@yahoo.fr

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* Corresponding author. Address: Laboratoire de microbiologie appliquée, FSNV, Université A/MIRA de Béjaia, 06000, Algérie. Tel./fax: +213 34 21 47 62.

Infection and its control in group homes for the elderly in Japan

Madam,

Following the implementation in Japan of a new, long-term care insurance system in April 2000, the number of small-scale facilities known as group homes has risen rapidly to more than 6000. These homes, which provide an alternative to traditional, larger-scale long-term care facilities for elderly demented people, are regulated by the municipality. The municipality is responsible for assigning, supervising and instructing all group home employers. Compared with care at traditional, larger-scale facilities, care at small-scale group homes is believed to treat patients better primarily in terms of dementia symptom management and minimizing functional decline.

Recently, mass outbreaks of influenza and norovirus in senior care facilities have been reported in many regions.[1–3] Small-scale facilities have paid less attention to infection control than large-scale ones and were unequipped with manuals outlining policies for the prevention of infectious diseases. Therefore, we examined the actual conditions of infection and the systems of infection control in small-scale care facilities in Japan.

Questionnaires were sent to 1899 care facilities registered with the National Association of Dementia Group Homes throughout Japan to investigate infection control measures at each facility. Discussions were held with community-based service representatives, including municipal supervisors and instructors as well as infection control specialists; and inspections of small-scale multifunctional group homes, dementia group homes, and group homes for fewer than 29 people needing heavy care were conducted to examine the current situation from multiple perspectives.

In all, 684 facilities (36%) completed the questionnaires. As shown in Figure 1, 26% of facilities had residents who had been infected with influenza, 14.5% with scabies, 12% with norovirus and 8.2% with meticillin-resistant Staphylococcus aureus (MRSA). These four communicable diseases were frequently found in residents. The mass outbreaks
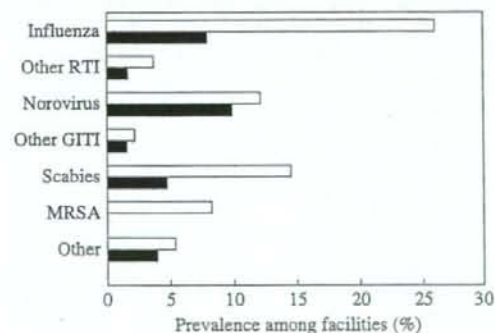

Figure 1   Prevalence of infections and outbreaks among the facilities. Open bars show the prevalence of facilities which had residents who suffered from the infection indicated. Closed bars show the prevalence of facilities which experienced mass outbreaks of infection indicated. 'Other RTI' denotes respiratory tract infections other than influenza and meticillin-resistant Staphylococcus aureus (MRSA). 'Other GITI' denotes gastrointestinal tract infections other than norovirus and MRSA. 'Other' denotes infection in organs other than the respiratory tract and the gastrointestinal tract.

reported in these facilities were norovirus (9.8%), influenza (7.9%) and scabies (4.7%) (Figure 1). Influenza vaccination was provided to all employees and residents upon request, in most facilities. Although facilities did not experience any mass outbreaks of MRSA, the procedures to cope with MRSA differed among facilities; 10.1% failed to address MRSA, 3.9% isolated infected/colonised individuals in a room while 3.5% used gowns and pre-prepared disinfectant. Regarding the response following norovirus infection, 90.1% of facilities used gloves but only 60.1% used masks when disposing of vomit. In all, 26.9% of facilities kept pets, 11.6% kept dogs, 4.7% kept cats and 0.3% kept reptiles that are known to be carriers of *Salmonella* spp. Although most facilities reported policies addressing the collection of bodily fluids, blood and faeces for disposal, no standardized policies outlining final disposal methods were reported; 60% of facilities disposed of them as general refuse. Oral care, which is considered to have an effect in preventing pneumonia, was done regularly by dentists or hygienists in 22.4% of facilities. It was also revealed that even though many facilities implemented response measures to stop the spread of influenza, they experienced mass outbreaks of norovirus, influenza and scabies. Improvement is needed especially in the disposal of infectious waste since many facilities did not use masks when disposing of norovirus vomit.

We conclude that improvement in the management of infectious disease in small-scale facilities for elderly people in Japan is needed. The problems highlighted in this research show the need for developing standardized infectious disease control strategies and for creating a manual that outlines detailed measures designed to specifically meet the needs of small-scale group homes in Japan.

Conflict of interest statement
None declared.

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References

1. Bradley SF. Prevention of influenza in long-term-care facilities. *Infect Control Hosp Epidemiol* 1999;20:629—637.
2. Dedman D, Laurichesse H, Caul EO, Wall PG. Surveillance of small round structured virus (SRSV) infection in England and Wales, 1990—5. *Epidemiol Infect* 1998;121: 131—149.
3. Goller JL, Dimitriadis A, Tan A, Kelly H, Marshall JA. Long-term features of norovirus gastroenteritis in the elderly. *J Hosp Infect* 2004;58:286—291.

S. Ebihara[a,b,*]
J. Aida[a]
S. Freeman[b]
K. Osaka[a]
[a]*Department of International Oral Health,
Tohoku University School of Dentistry,
Sendai, Japan*
[b]*Department of Geriatrics and Gerontology,
Tohoku University School of Medicine,
Sendai, Japan*
E-mail address: s_ebihara@geriat.med.
tohoku.ac.jp

* Corresponding author. Address: Department of Geriatrics and Gerontology, Tohoku University School of Medicine, Seiryo-machi 1-1, Aoba-ku, Sendai 980-8574, Japan. Tel: +81 22 717 7182; fax: +81 22 717 7186.

Meticillin-resistant *Staphylococcus aureus* in the community: homeless are also at risk

Madam,

Thomas *et al.* recently identified the district nurse population as a significant reservoir for meticillin-resistant *Staphylococcus aureus* (MRSA) in the community, with 21.1% [confidence interval (CI): 11.6—30.4] of the study population found to be MRSA positive.[1] Other population groups known to be at risk of community MRSA colonisation or infection include military recruits, sports teams players, men who have sex with men, people in jail, injecting drug users (IDUs) and the homeless. Current or past IDUs and a history of skin abscess is associated with a higher prevalence of meticillin resistance in those who are *S. aureus*-colonised.[2–5] Studies from the USA have shown that the homeless are at a significantly increased risk (odds ratio: 3.35; 95% confidence interval: 1.22—9.22) of community-acquired MRSA skin and soft tissue infections compared with the non-homeless.[3,4] To our knowledge, we present the first assessment of skin and soft tissue infections due to MRSA in people who are homeless or at risk of homelessness in the UK.

We identified all wound swabs routinely submitted to the Health Protection Agency Regional Microbiology Laboratory in Cambridge over a period of four years (3 August 2003 to 3 August 2007) from

Original Article

Home Blood Pressure Is Associated with Depressive Symptoms in an Elderly Population Aged 70 Years and Over: A Population-Based, Cross-Sectional Analysis

Kaijun NIU[1], Atsushi HOZAWA[2],[3], Shuichi AWATA[4], Hui GUO[1],
Shinichi KURIYAMA[2], Toru SEKI[4], Kaori OHMORI-MATSUDA[2], Naoki NAKAYA[2],
Satoru EBIHARA[5], Yun WANG[1], Ichiro TSUJI[2], and Ryoichi NAGATOMI[1]

Although several epidemiologic studies have assessed the relationship between low blood pressure and depressive symptoms in geriatric populations, the results have been inconsistent. Because the white-coat phenomenon is observed frequently in patients with depressive symptoms, we have considered that blood pressure measured in nonmedical settings is important in assessing the relationship between blood pressure and depressive symptoms among the geriatric population. The aim of this study was to investigate the relationships between home blood pressure and depressive symptoms in a community-based elderly population aged 70 years and over. We analyzed a cross-sectional survey comprised of 888 community-dwelling Japanese aged 70 years and older. Blood pressure was self-measured at home, and depressive symptoms were evaluated using the 30-item Geriatric Depression Scale (GDS 30) with a cutoff point of 11. The prevalence of depressive symptoms was 34.8%. For all subjects, after adjustments for potentially confounding factors, the odds ratios of having depressive symptoms by increasing quartiles of systolic blood pressure of subjects not taking antihypertensive drugs to subjects taking them were 1.00, 0.97, 0.88, 0.59, and 0.70. Statistically significant inverse relationships were observed in subjects not taking antihypertensive drugs. No apparent association between diastolic blood pressure and depressive symptoms was observed in any subjects or in a stratified analysis of antihypertensive drug use. In this study, a higher home systolic blood pressure was independently and continuously related to a lower prevalence of depressive symptoms in participants not using antihypertensive medication. Further study is required to clarify the causality of this relationship. (*Hypertens Res* 2008; 31: 409–416)

Key Words: home blood pressure, depressive symptoms, Japanese, community-dwelling population

Introduction

Depression in late life is a recognized public health problem. Population-based estimates of the prevalence of mild and severe depression (using cutoffs of 5/6 on the Geriatric Depression Scale [GDS]-15) among older women in Japan range from 34.2% to 37.0% and among older men range from 29.7% to 35.4% (*1*).

The global burden of hypertension is a leading risk factor for cardiovascular and kidney disease and for mortality (*2–4*). It has overshadowed possible health problems associated with chronic low blood pressure (BP). Hypotension also is common in the general population and is associated with a distinct body habitus (*5*). Low BP was associated with the development of idiopathic chronic fatigue in women (*6*) and with neurasthenic symptoms such as weakness, fatigue, crying, psychological dysfunction, dizziness, and headache (*7–9*). Recent studies also have indicated that low BP reduced cortical activity (*10*), cerebral perfusion, and maladaption of blood flow to cognitive demands (*11*). Since these chronic psychological dysfunctions and/or body symptoms may be associated with depressive symptoms (*9*), it was hypothesized that low BP may be a potential risk factor for depressive symptoms (*12*).

However, in epidemiological studies, observations of the relationship between BP and depression have been inconsistent (*12–20*). Because the white-coat phenomenon has been observed frequently in all psychological types but especially in types having depressive symptoms (*21, 22*), we have considered that BP measured in nonmedical settings is important in assessing the relationship between BP and depressive symptoms among the geriatric population. BP measurements at home (home blood pressure, HBP) are known to avoid the white-coat phenomenon (*23–25*). However, to our knowledge, no previous study used HBP to examine the association between BP and depressive symptoms among a geriatric population.

Thus, we used HBP to investigate the relationship between BP (including systolic BP [SBP] and diastolic BP [DBP]) and depressive symptoms among community-dwelling elderly aged 70 years and over.

Methods

Study Participants

Our study population was comprised of subjects aged 70 years and older who were living in the Tsurugaya area of Sendai, one of the major cities in the Tohoku area of Japan. At the time of the study in 2002, there were 2,730 individuals aged 70 years and older living in Tsurugaya. All of these individuals were invited to participate in a comprehensive geriatric assessment, which included medical status, physical function, cognitive function, and dental status. Among them, 1,178 did

so, giving their informed consent for data analysis. The protocol of this study was approved by the Institutional Review Board of the Tohoku University Graduate School of Medicine.

In this study, the depressive symptoms were assessed with the aid of the GDS (*26*). Out of the 1,178 subjects, 1,169 completed the GDS. Since BP was assessed using data from self-measured BP at home, subjects who did not measure HBP data for more than 3 days during the 4-week study period were excluded (*n*=184; GDS≥11: 41.0%). This criterion was based on a previous observation that average BP values for the first 3 days did not differ significantly from those obtained during the entire study period (*27, 28*). We also excluded all potential subjects with notable comorbidities, those who were using drugs that could influence the degree of depressive symptoms, those who reported a history of cancer (*29*) (*n*=68), those who were using anti-depressants (*n*=16), and 13 subjects with cognitive dysfunction (Mini Mental State Examination, MMSE, Score (*30*) <18). As a result of these exclusions, the final study population was comprised of 888 subjects (mean±SD age 75.7±4.6 yeras, men: 44.0%).

Assessment of Depressive Symptoms

Depressive symptoms were assessed according to the Japanese version (*31*) of the 30-item GDS using a cutoff point (GDS score ≥11) indicating relatively mild to severe depressive symptoms (*26*).

Assessment of HBP

HBP was self-measured with an HEM747IC device (Omron Life Science, Tokyo, Japan), which uses the cuff oscillometric method to generate a digital display of SBP and DBP. This device has been validated previously, and satisfies the criteria of the Association for the Advancement of Medical Instrumentation (*32*). The following procedure was used to ascertain the accuracy of the HBP measurement. First, physicians informed the subjects about HBP recording and taught them how to measure their own BP. The daily measurement was made within 1 h after waking up and before breakfast, with the subject seated and having rested for at least 2 min. Subjects taking antihypertensive drugs measured their HBP before taking the drugs. An individual's HBP was defined as the mean of all measurements obtained for that person. The mean (±SD) number of HBP measurements was 15.7±10.5 (range, 3–49).

Assessment of Other Variables

Anthropometrics (height, body weight) were recorded by a standardized protocol. Body mass index (BMI) was calculated as weight (kg)/height^2 (m^2).

Sociodemographic variables including gender, age, educational level, and perceived social supports were also assessed.

Table 1. Baseline Characteristics According to Blood Pressure Status

	Subjects non-taking antihypertensive drugs (quartile)				Subjects taking antihypertensive drugs
	SBP				SBP
	81.4–123.6 mmHg	123.7–134.7 mmHg	134.9–147.0 mmHg	147.2–203.3 mmHg	105.7–224.7 mmHg
No. of participants	128	127	129	128	376
Age (years)	74.2 (73.5–75.0)	75.4 (74.6–76.2)	76.3 (75.5–77.1)	76.4 (75.6–77.2)	75.9 (75.5–76.4)
Sex (female)	43.8	63.0	55.8	58.6	56.9
BMI	22.3 (21.8–22.9)	23.3 (22.8–23.9)	23.8 (23.2–24.3)	24.6 (24.0–25.2)	24.4 (24.1–24.8)
Total number of physical illness					
≥4	12.5	15.0	17.1	21.9	39.1
2–3	42.2	43.3	46.5	38.3	43.4
Lack of perceived social support (total score=0)	18.8	22.8	20.2	19.5	18.7
Cognitive ability					
Normal (28≤MMSE≤30)	63.3	63.8	55.0	60.9	59.3
Slightly impaired (24≤MMSE≤27)	31.3	32.3	39.5	33.6	33.8
Smoking status					
Current smoker	14.8	11.8	16.3	11.7	10.9
Ex-smoker	31.3	23.6	24.0	33.6	32.5
Driking status					
Current drinker	38.3	38.6	45.0	37.5	41.2
Ex-drinker	16.4	11.8	7.8	7.8	13.0
PA (≥ level 4)	39.8	33.1	30.2	28.1	31.9
Educational level (≤12 years)	33.6	45.7	45.0	39.1	44.4
Pain	26.6	15.0	24.0	20.3	23.9
Impaired IADL	17.2	18.9	20.2	13.3	20.5

	Subjects non-taking antihypertensive drugs (quartile)				Subjects taking antihypertensive drugs
	DBP				DBP
	49.6–69.0 mmHg	69.3–74.9 mmHg	75.0–82.1 mmHg	82.1–105.4 mmHg	49.3–123.8 mmHg
No. of participants	128	128	128	128	376
Age (years)	75.8 (75.0–76.6)	75.8 (75.0–76.6)	75.4 (74.6–76.2)	75.3 (74.5–76.1)	75.9 (75.5–76.4)
Sex (female)	54.7	53.9	57.8	54.7	56.9
BMI	22.4 (21.8–22.9)	23.7 (23.1–24.2)	23.8 (23.2–24.3)	24.2 (23.6–24.8)	24.4 (24.1–24.8)
Total number of physical illness					
≥4	14.1	17.2	15.6	19.5	39.1
2–3	49.2	41.4	42.2	37.5	43.4
Lack of perceived social support (total score=0)	22.7	23.4	20.3	14.8	22.3
Cognitive ability					
Normal (28≤MMSE≤30)	63.3	56.3	57.8	65.6	59.3
Slightly impaired (24≤MMSE≤27)	30.5	38.3	37.5	30.5	33.8
Smoking status					
Current smoker	14.8	10.2	14.8	14.8	10.9
Ex-smoker	25.0	31.3	25.0	31.3	32.5
Driking status					
Current drinker	36.7	35.2	43.0	44.5	41.2
Ex-drinker	14.8	14.1	7.8	7.0	13.0
PA (≥ level 4)	41.4	35.2	28.9	25.8	31.9
Educational level (≤12 years)	39.1	46.1	43.0	35.2	44.4
Pain	22.7	21.1	24.2	18.0	23.9
Impaired IADL	20.3	20.3	17.2	11.7	20.5

Variables are presented as mean (95% CI) or %. CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; MMSE, Mini Mental State Examination; PA, physical activity; IADL, instrumental activities of daily living.

Educational level was assessed by determining age at completion of schooling and was divided into two categories: ≤12 or >12 years. Perceived social support (PSS) was evaluated on the basis of responses (yes or no) to five questions: "Do you have someone to talk to when you are in trouble?" (PSS 1); "Do you have someone to talk to when you're not feeling well?" (PSS 2); "Do you have someone who can help you with daily housework?" (PSS 3); "Do you have someone who can take you to the hospital when you don't feel well?" (PSS 4); and "Do you have someone who can take care of you when you are ill in bed?" (PSS 5). These questions were extracted from a previous study regarding social support and elderly depression in a rural community (33). A strong association between negative answers to these items and depression has been confirmed in two Japanese community studies of elderly populations (33, 34). A single summed score was calculated based on the PSS 1–5. No PSS was defined as a score of 0.

Health-related variables assessed included history of physical illness, pain, cognitive function, instrumental activities of daily living (IADL), and current use of medication. History of physical illness was evaluated on the basis of responses (yes or no) to questions concerning history of a stroke, ischemic heart disease, diabetes mellitus, hyperuricemia, hyperlipidemia, renal disease, liver disease, cholelithiasis or cholecystitis, a gastric or duodenal ulcer, tuberculosis, pneumonia, asthma, a hearing disturbance, cataracts, glaucoma, arthritis, and osteoporosis. Subjects were classified into three categories according to the total number of these conditions in the subject's history: 0–1, 2–3, or ≥4. Pain within the previous 4 weeks was assessed by the question, "Have you had any pain recently? If so, how intensely do you feel such pain?" Possible answers were "no pain," "very mild pain," "mild pain," "moderate pain," and "severe pain." A subject who reported mild to severe pain was considered to have pain. Cognitive function was assessed on the basis of the MMSE and was classified into three categories: 18–23, 24–27, and 28–30. IADLs were assessed using the Rouken-Shiki scale (35), and a cutoff point of 10/11 was used to determine impairment in IADL. Information about antihypertensive drugs was confirmed by a well-trained pharmacist.

Information on smoking status and drinking status was obtained from the questionnaire survey. Physical activity (PA) was assessed first by a self-reported single-item question on whether or not the participant obtained any PA in the past year. If yes, questions were asked about the frequency and duration of walking, brisk walking, and sports. PA was then classified into three categories, based on frequency and duration in the participant: 1) High: at least 3–4 times per week for at least 30 min each time; 2) Low: reporting some activity in the past year, but not enough to meet high levels; and 3) None: no PA. Furthermore, PA was classified into six levels based on these three categories and each PA such as walking, brisk walking, and sports: 1) Level 1: no walking, no brisk walking, no sports; 2) Level 2: low walking, no brisk

walking, no sports; 3) Level 3: high walking, no brisk walking, no sports; 4) Level 4: any walking, low brisk walking, no sports; 5) Level 5: any walking, high brisk walking, no sports; 6) Level 6: any walking, any brisk walking, low or high sports. Detailed information was provided in previous reports (36). Finally, subjects were divided into two categories: ≤level 3 or >level 3.

Statistical Analysis

Descriptive data are presented as means (95% confidence interval [CI]) or percentages. Depressive symptoms (GDS scores ≥11) were used as the dependent variables and the HBP level as the independent variable. Multiple logistic regression analysis was used to examine the relationship between HBP and depressive symptoms after adjustment for age, sex, BMI, self-reported medical conditions, lack of PSS, smoking and drinking habits/history, educational level, impaired physical functioning, cognitive status, pain, PA, and the number of HBP measurements. p values for linear trends were calculated using the median (mmHg) of HBP groups. The odds ratios (ORs) and 95% CI of depressive symptoms for increasing HBP levels, with the lowest level as the reference, were also calculated using multiple logistic regression analysis. A significant difference was defined as $p < 0.05$. All statistical analyses were performed using the Statistical Analysis System 9.1 edition for Windows (SAS Institute, Cary, USA).

Results

In this study, 34.8% (407/1,169) of the subjects were classified as having depressive symptoms. Among the 888 subjects who were available for analysis, 285 (32.1%) were classified as having depressive symptoms.

Baseline characteristics according to BP status are presented in Table 1.

Table 2 shows the adjusted association between the HBP quartile of subjects not taking antihypertensive drugs, subjects taking antihypertensive drugs, and depressive symptoms. Although not statistically significant, ORs adjusted for potentially confounding factors for depressive symptoms were lowest in the highest tertiles of subjects not taking antihypertensive drugs (OR: 0.59; 95% CI: 0.32–1.08) in SBP. Similarly, we observed lower ORs of having depression in participants taking antihypertensive medication compared with the lowest SBP quartile of participants not taking antihypertensive medication. In contrast, there was no apparent association between DBP and depressive symptoms.

Table 3 shows separately the adjusted association between HBP quartiles and depressive symptoms in subjects not taking antihypertensive drugs and those taking antihypertensive drugs. In subjects not taking antihypertensive drugs, the ORs of depressive symptoms decreased across the levels of SBP. The age- and sex-adjusted ORs for depressive symptoms

Table 2. Adjusted Relationships of Blood Pressure to Depressive Symptoms

	Subjects non-taking antihypertensive drugs (quartile)				Subjects taking antihypertensive drugs
	SBP				SBP
	81.4–123.6 mmHg	123.7–134.7 mmHg	134.9–147.0 mmHg	147.2–203.3 mmHg	105.7–224.7 mmHg
No. of participants	128	127	129	128	376
No. of depressive symptoms	43	44	43	32	123
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	0.92 (0.54–1.56)	0.87 (0.51–1.47)	0.57 (0.32–0.98)	0.85 (0.55–1.32)
Multiple adjusted*	1.00	0.97 (0.55–1.69)	0.88 (0.50–1.55)	0.59 (0.32–1.08)	0.70 (0.43–1.15)

	Subjects non-taking antihypertensive drugs (quartile)				Subjects taking antihypertensive drugs
	DBP				DBP
	49.6–69.0 mmHg	69.3–74.9 mmHg	75.0–82.1 mmHg	82.1–105.4 mmHg	49.3–123.8 mmHg
No. of participants	128	128	128	128	376
No. of depressive symptoms	38	49	42	33	123
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	1.48 (0.88–2.52)	1.16 (0.68–1.98)	0.83 (0.48–1.45)	1.14 (0.74–1.78)
Multiple adjusted*	1.00	1.51 (0.87–2.64)	1.20 (0.68–2.12)	0.94 (0.52–1.69)	0.96 (0.59–1.57)

*Adjusted for age, sex, BMI, self-reported medical conditions, lack of perceived social support, respectively, smoking and drinking habits/history, educational level, cognitive status, pain, physical activity, impaired instrumental activities of daily living and the number of HBP measurements. CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; HBP, home blood pressure.

across SBP were 1.00, 0.86, 0.82, and 0.53 (p for trend=0.03), and ORs across DBP were 1.00, 1.50, 1.16, and 0.83 (p for trend=0.31), respectively. These results were unchanged when adjusted for multiple confounding factors. In contrast, no apparent association was observed in subjects taking antihypertensive drugs.

Discussion

In this cross-sectional study, we examined the relationship between HBP and depressive symptoms in a community-dwelling elderly population aged 70 years and over. Our results suggested that lower SBP, but not lower DBP, was related to depressive symptoms independently of potential confounding factors in subjects not taking antihypertensive drugs.

Several studies used office BP to investigate the relationship between BP and depressive symptoms, but the results were inconsistent. Four cross-sectional (*12–15*) studies and a prospective study (*16*) showed inverse associations between BP and depressive symptoms. Another prospective study found no association (*17*). In contrast, three cross-sectional studies found a positive association between hypertension

and depression (*18–20*). Only one previous study (*37*) using HBP reported no significant relationship between BP levels, and psychological dysfunction/fatigue was demonstrated among younger subjects (mean age±SD: 48.7±16 years). However, they did not assess the depressive symptoms. Because some studies reported that the white-coat phenomenon was frequently observed in depressive symptoms (*21*, *22*), it might be interesting to investigate the relationship between BP measured in nonmedical settings and depressive symptoms. Therefore, HBP was used to assess the relationships between BP and depressive symptoms in this study. The present results suggested that SBP, but not DBP, is inversely related to depressive symptoms among an elderly population not taking hypertensive drugs.

An important finding in the current study was the inverse relationship between HBP and depressive symptoms in subjects who were not taking antihypertensive drugs. Moreover, the ORs of having depression were lower, although not significantly, in subjects taking antihypertensive drugs than among the lowest SBP quartile of subjects not taking antihypertensive drugs. This result can be interpreted in several ways. First, because antihypertensive medication lowered BP, weaken BP's influence on depressive symptoms. Second, one

Table 3. Adjusted Relationships of Blood Pressure (Quartile) to Depressive Symptoms

a. Subjects non-taking antihypertensive drugs (*n*=512)

	Systolic blood pressure (mmHg)				*p* for trend
	81.4–123.6	123.7–134.7	134.9–147.0	147.2–203.3	
No. of participants	128	127	129	128	—
No. of depressive symptoms	43	44	43	32	—
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	0.86 (0.50–1.47)	0.82 (0.48–1.40)	0.53 (0.30–0.92)	0.03
Multiple adjusted*	1.00	0.81 (0.46–1.47)	0.72 (0.39–1.33)	0.46 (0.23–0.88)	0.02
	Diastolic blood pressure (mmHg)				*p* for trend
	49.6–69.0	69.3–74.9	75.0–82.1	82.1–105.4	
No. of participants	128	128	128	128	—
No. of depressive symptoms	38	49	42	33	—
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	1.50 (0.88–2.55)	1.16 (0.67–1.98)	0.83 (0.48–1.45)	0.31
Multiple adjusted*	1.00	1.47 (0.82–2.64)	1.15 (0.64–2.09)	0.90 (0.48–1.69)	0.52

b. Subjects taking antihypertensive drugs (*n*=376)

	Systolic blood pressure (mmHg)				*p* for trend
	105.7–134.3	134.5–144.3	144.7–156.4	156.5–224.7	
No. of participants	94	94	94	94	—
No. of depressive symptoms	33	29	30	31	—
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	0.82 (0.44–1.51)	0.86 (0.46–1.58)	0.84 (0.45–1.57)	0.64
Multiple adjusted*	1.00	0.84 (0.44–1.62)	1.03 (0.53–1.99)	0.80 (0.41–1.59)	0.64
	Diastolic blood pressure (mmHg)				*p* for trend
	49.3–72.0	72.0–77.9	77.9–84.0	84.0–123.8	
No. of participants	94	94	94	94	—
No. of depressive symptoms	32	38	23	30	—
Odds ratio (95% CI)					
Age- and sex-adjusted	1.00	1.34 (0.74–2.43)	0.64 (0.34–1.21)	0.93 (0.51–1.72)	0.39
Multiple adjusted*	1.00	1.70 (0.89–3.29)	0.85 (0.43–1.70)	0.95 (0.49–1.84)	0.43

*Adjusted for age, sex, BMI, self-reported medical conditions, lack of perceived social support, respectively, smoking and drinking habits/history, educational level, cognitive status, pain, physical activity and impaired instrumental activities of daily living and the number of HBP measurements. CI, confidence interval; BMI, body mass index; HBP, home blood pressure.

more possibility that might explain the inverse relationship between SBP and depressive symptoms was the issue of measurement timing. HBP was measured in the early morning, and a question on the GDS (Question 27: "Do you enjoy getting up in the morning?") was in harmony with symptoms of hypotension in the morning. Therefore, participants with morning hypotension tended to score higher on the GDS scale. This also makes it possible to explain the inverse association of BP with depression symptoms. To understand the causal relation of BP in nonmedical settings with depressive symptoms, prospective studies using ambulatory BP measurements that can assess not only morning BP but also daytime BP are required.

The biological mechanisms involved in the relationship between HBP and depression are not well known. Recent findings have indicated that many neuropeptides play a major role in mediating the response to stress-related diseases, including depression and anxiety disorders (*38*). The overexpression of endogenous neuropeptide Y in transgenic rats has been associated with lower BP at baseline and during stress (*39*). Thus, these findings suggested that neuropeptide Y may be a possible link between low BP and depressive symptoms. Further study is required to clarify the biological mechanism underlying the association between lower BP and depressive symptoms.

A previous study reported that chronic hypotensives have a female preponderance (*40*). In contrast, the current results show that the proportion of males was higher in the lowest SBP group than in the other SBP groups (Table 1). Differences in participant population may partly explain the incon-

sistencies. Further study is required for clarification.

This study had several limitations. First, the participants were sufficiently active and healthy to participate in the survey; therefore, it is possible that our results do not apply to subjects at higher risk. Second, because this study was cross-sectional, we could not conclude that lower HBP increases the occurrence of depressive symptoms or that depressive symptoms lead to the decline of HBP among subjects aged 70 years and over. Therefore, a prospective study or trial should be undertaken to confirm the relationship between HBP and depressive symptoms. Third, since screening BP was not measured, we could not infer the influence of screening BP on depressive symptoms in this study population. Finally, the GDS scale was designed for measuring the intensity of depressive symptoms and not for making a clinical diagnosis of depressive episodes. Therefore, a larger sample population using a standardized comprehensive structured diagnostic interview should be undertaken to confirm the relation between HBP and depressive episodes.

In conclusion, home SBP, but not home DBP, was independently related to a lower prevalence of depressive symptoms in a community-dwelling elderly population not taking antihypertensive drugs. A prospective study or randomized trials are required to clarify the causality in this relationship.

Acknowledgements

References

1. Wada T, Ishine M, Sakagami T, et al: Depression in Japanese community-dwelling elderly—prevalence and association with ADL and QOL. *Arch Gerontol Geriatr* 2004; 39: 15–23.

2. Shimamoto K, Kita T, Mabuchi H, et al: Effects of hypertension and type 2 diabetes mellitus on the risk of total cardiovascular events in Japanese patients with hypercholesterolemia: implications from the Japan Lipid Intervention Trial (J-LIT). *Hypertens Res* 2007; 30: 119–123.

3. Ishiguro K, Sasamura H, Sakamaki Y, Itoh H, Saruta T: Developmental activity of the renin-angiotensin system during the "critical period" modulates later L-NAME–induced hypertension and renal injury. *Hypertens Res* 2007; 30: 63–75.

4. Nakamura K, Okamura T, Kanda H, et al: Impact of hypertension on medical economics: a 10-year follow-up study of national health insurance in Shiga, Japan. *Hypertens Res* 2005; 28: 859–864.

5. Owens PE, Lyons SP, O'Brien ET: Arterial hypotension: prevalence of low blood pressure in the general population using ambulatory blood pressure monitoring. *J Hum Hypertens* 2000; 14: 243–247.

6. Lucas KE, Rowe PC, Coresh J, Klag MJ, Meoni LA, Ford DE: Prospective association between hypotension and idiopathic chronic fatigue. *J Hypertens* 2004; 22: 691–695.

7. Wessely S, Nickson J, Cox B: Symptoms of low blood pressure: a population study. *BMJ* 1990; 301: 362–365.

8. Pilgrim JA, Stansfeld S, Marmot M: Low blood pressure, low mood? *BMJ* 1992; 304: 75–78.

9. Pemberton J: Does constitutional hypotension exist? *BMJ* 1989; 298: 660–662.

10. Duschek S, Meinhardt J, Schandry R: Reduced cortical activity due to chronic low blood pressure: an EEG study. *Biol Psychol* 2006; 72: 241–250.

11. Duschek S, Schandry R: Cognitive performance and cerebral blood flow in essential hypotension. *Psychophysiology* 2004; 41: 905–913.

12. Barrett-Connor E, Palinkas LA: Low blood pressure and depression in older men: a population based study. *BMJ* 1994; 308: 446–449.

13. Stroup-Benham CA, Markides KS, Black SA, Goodwin JS: Relationship between low blood pressure and depressive symptomatology in older people. *J Am Geriatr Soc* 2000; 48: 250–255.

14. Hildrum B, Mykletun A, Stordal E, Bjelland I, Dahl AA, Holmen J: Association of low blood pressure with anxiety and depression: the Nord-Trondelag Health Study. *J Epidemiol Community Health* 2007; 61: 53–58.

15. Jorm AF: Association of hypotension with positive and negative affect and depressive symptoms in the elderly. *Br J Psychiatry* 2001; 178: 553–555.

16. Paterniti S, Verdier-Taillefer MH, Geneste C, Bisserbe JC, Alperovitch A: Low blood pressure and risk of depression in the elderly. A prospective community-based study. *Br J Psychiatry* 2000; 176: 464–467.

17. Gilmore C, Green BH, Copeland JR, et al: Blood pressure as a risk factor for depression in elderly people: a prospective study. *Acta Psychiatr Scand* 1995; 91: 126–129.

18. Kabir AA, Whelton PK, Khan MM, Gustat J, Chen W: Association of symptoms of depression and obesity with hypertension: the Bogalusa Heart Study. *Am J Hypertens* 2006; 19: 639–645.

19. Artinian NT, Washington OG, Flack JM, Hockman EM, Jen KL: Depression, stress, and blood pressure in urban African-American women. *Prog Cardiovasc Nurs* 2006; 21: 68–75.

20. Chen R, Wei L, Hu Z, Qin X, Copeland JR, Hemingway H: Depression in older people in rural China. *Arch Intern Med* 2005; 165: 2019–2025.

21. Shkarin VV, Shkarina IN, Malygin VL: Psychological aspects of systemic approach to intra-nosological diagnosis in arterial hypertension. *Ter Arkh* 2000; 72: 43–46.

22. MacDonald MB, Laing GP, Wilson MP, Wilson TW: Prevalence and predictors of white-coat response in patients with treated hypertension. *CMAJ* 1999; 161: 265–269.

23. Imai Y, Okubo T, Asayama K, et al: Epidemiology of hypertension based on ambulatory blood pressure monitoring and self-measurement of blood pressure at home. *J Health Sci* 2004; 50: 113–119.

24. O'Brien E, Asmar R, Beilin L, et al: European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens* 2003; 21: 821–848.

25. Hozawa A, Ohkubo T, Kikuya M, et al: Blood pressure control assessed by home, ambulatory and conventional

blood pressure measurements in the Japanese general population: the Ohasama study. *Hypertens Res* 2002; **25**: 57–63.

26. Brink T, Yesavage J, Lum O, Heersema P, Adey M, Rose T: Screening tests for geriatric depression. *Clin Gerontol* 1982; **1**: 37–44.

27. Hozawa A, Ebihara S, Ohmori K, *et al*: Increased plasma 8-isoprostane levels in hypertensive subjects: the Tsurugaya Project. *Hypertens Res* 2004; **27**: 557–561.

28. Imai Y, Satoh H, Nagai K, *et al*: Characteristics of a community-based distribution of home blood pressure in Ohasama in northern Japan. *J Hypertens* 1993; **11**: 1441–1449.

29. Williams S, Dale J: The effectiveness of treatment for depression/depressive symptoms in adults with cancer: a systematic review. *Br J Cancer* 2006; **94**: 372–390.

30. Folstein MF, Folstein SE, McHugh PR: "Mini-mental state." A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res* 1975; **12**: 189–198.

31. Niino N, Imaizumi T, Kawakami N: A Japanese translation of the Geriatric Depression Scale. *Clin Gerontol* 1991; **10**: 85–87.

32. Chonan K, Kikuya M, Araki T, *et al*: Device for the self-measurement of blood pressure that can monitor blood pressure during sleep. *Blood Press Monit* 2001; **6**: 203–205.

33. Muraoka Y, Oiji A, Ihara K: The physical and psychological and social background factor of elderly depression in the community. *Jpn J Geriatr Psychiatry* 1996; **7**: 397–407 (in Japanese).

34. Koizumi Y, Awata S, Seki T: Association between social support and depression in the elderly Japanese population. *Jpn J Geriatr Psychiatry* 2004; **41**: 426–433 (in Japanese).

35. Koyano W, Shibata H, Haga H, Suyama Y, Nakazato K: Measurement of competence in the elderly living at home: development of an index of competence. *Nippon Koshueiseigaku Zasshi* 1987; **34**: 109–114 (in Japanese).

36. Niu K, Hozawa A, Fujita K, *et al*: Influence of leisure-time physical activity on the relationship between C-reactive protein and hypertension in a community-based elderly population of Japan: the Tsurugaya project. *Hypertens Res* 2005; **28**: 747–754.

37. Donner-Banzhoff N, Chan Y, Szalai JP, Hilditch JR: Low blood pressure associated with low mood: a red herring? *J Clin Epidemiol* 1997; **50**: 1175–1181.

38. Holmes A, Heilig M, Rupniak NM, Steckler T, Griebel G: Neuropeptide systems as novel therapeutic targets for depression and anxiety disorders. *Trends Pharmacol Sci* 2003; **24**: 580–588.

39. Fuxe K, Aguirre JA, Agnati LF, *et al*: Neuropeptide Y and central cardiovascular regulation. Focus on its role as a cotransmitter in cardiovascular adrenergic neurons. *Ann N Y Acad Sci* 1990; **611**: 111–132.

40. Akahoshi M, Hida A, Imaizumi M, *et al*: Basic characteristics of chronic hypotension cases: a longitudinal follow-up study from 1958 through 1999. *Hypertens Res* 2006; **29**: 1–7.

SEX DIFFERENCES IN THE PREFERENCE FOR PLACE OF DEATH IN COMMUNITY-DWELLING ELDERLY PEOPLE IN JAPAN

To the Editor: The Ministry of Health, Labor, and Welfare in Japan is strongly promoting "death at home."[1] Research indicates that honoring the treatment preferences of terminally ill patients is critical for the provision of high-quality care at the end of life,[2,3] but few studies have investigated the preference for place of death in elderly people in Japan. In this study, we found that only half of community-dwelling elderly people studied wanted to die at home when they become terminally ill. Especially in elderly women, there is a strong desire for not wanting to inconvenience others, and therefore many of them prefer "hospital deaths."

The survey was conducted from November 2006 to April 2007. Face-to-face questionnaires were given to all members aged 70 and older who belonged to four nonreligious affiliated seniors clubs. Two of the seniors clubs were rurally located in Iwanuma village, Miyagi prefecture, northern Japan (n = 55 and 71), whereas the other two were located in Kawasaki city, an urban area in central Japan (n = 84 and 76). Subjects were aged 70 to 102, with a mean age ± standard deviation of 77.0 ± 6.1. Questionnaires focused on the place where the subjects want to be cared for when they die and demographic factors (housing status, personal medical history, degree of physical and mental independence). One hundred fifty men (aged 75.9 ± 5.3) and 136 women (aged 78.2 ± 6.6) responded.

Results showed that 50% of respondents preferred to die at home, 33.6% in a hospital, and 5.2% in a nursing facility. There were more multigenerational households in rural areas than in urban areas, and many of them were two- and three-generation families. No significant differences were found in the preferences of place to die according to the number of family members living together or age, but 60% of men preferred to die at home, whereas 40% each of women wanted to die at home and in the hospital.

In addition, for the following question "What is the most important thing when you are on your deathbed?" in which respondents were asked to choose two answers, approximately 110 men and 110 women chose the answer "dying without pain (or suffering)," followed by "dying at home" (n = 77) for men and "not bothering family and friends" (n = 58) for women. This highlighted the differences between the sexes regarding feelings toward life and death. Women, who did not want to bother others, significantly preferred hospital deaths.

The study revealed that individuals who have a higher degree of physical and mental independence tend to desire to die in a hospital. Women showed a marked tendency toward this. In contrast, many men are mentally and physically dependent on their spouse.

More than 80% of men and women are independent in terms of fundamental activities of daily living, but fewer than 50% of men did any housework, whereas nearly 70% of females did all the housework. More than 70% of men wanted to be cared for by their spouses when needed, whereas women wanted to be cared for by their daughters, daughter-in-laws, and spouses and in hospitals.

These results suggest that, although an individual's personal values and the degree of physical and mental dependence can affect the desire of death at home or in a hospital, sex differences need to be taken into account. The Ministry of Health, Labor and Welfare in Japan has been promoting home-based medical care after the medical service fee system was revised in 2006, but only half of elderly people want to die at home. This revision may not meet all of the needs of elderly people in Japan. End-of-life care for elderly people should not be only home-based medical care but also other types of care. More surveys on the actual conditions will be needed in the future.

Miyako Yamasaki, MD
Satoru Ebihara, MD
Shannon Freeman, BA
Takae Ebihara, MD
Masanori Asada, MD
Shinsuke Yamanda, MD
Hiroyuki Arai, MD
Department of Geriatrics and Gerontology
School of Medicine
Tohoku University
Sendai, Japan

REFERENCES

1. The 22nd Social Welfare Deliberation Committee, Medicare Section Meeting [on-line]. Available at http://www.mhlw.go.jp/shingi/2005/11/s1110-6c.html.
2. Council on Scientific Affairs . American Medical Association: Good care of the dying patients. JAMA 1996;275:474–478.
3. Lynn J. Measuring quality of care at end of life: A statement of principles. J Am Geriatr Soc 1997;45:526–527.

DIAGNOSTIC ACCURACY OF CRITERIA FOR URINARY TRACT INFECTION IN A COHORT OF NURSING HOME RESIDENTS

To the Editor: We are commenting on "Diagnostic Accuracy of Criteria for Urinary Tract Infection in a Cohort of Nursing Home Residents."[1] Table 1 in the article infers that 54 of 100 cases of suspected urinary tract infection (UTI) had neither fever nor urinary signs or symptoms. The discussion includes the unproven hypothesis that residents with UTI frequently exhibit nonspecific "geriatric manifestations" of acute disease such as confusion, falls, or functional decline *without* fever or urinary symptoms such as frequency, dysuria, or suprapubic tenderness. This is a widespread belief that is not supported by data or expert

科学的看護・介護による
嚥下障害・誤嚥性肺炎に対する予防
──食の楽しみの維持をめざして

Preventing aspiration pneumonia in the dysphagic elderly by scientific nursing and caring
── Towards maintaining of gratification of tasting and eating

海老原孝枝(写真)　海老原　覚
Takae EBIHARA and Satoru EBIHARA
東北大学加齢医学研究所加齢老年医学研究分野

◎一般的な高齢者の誤嚥は，咽喉頭の器質的な病態が原因であるというよりも，脳あるいは消化管に起因する，神経の知覚や伝達の障害といっても過言でない．であるから誤嚥の予防あるいは治療は，神経に対するアプローチが鍵となる．そのアプローチ法はアンジオテンシン酵素阻害剤やドパミン剤などによる投薬だけでは困難で，transient receptor potential 刺激，嗅覚刺激などの介護的アプローチが重要である．これらをうまく駆使することにより，嚥下障害高齢者は摂食する喜びを取り戻せる，あるいは維持できることが期待できる．われわれ医療人は誤嚥についてメディカル分野のみ診るのではなく，家人や介護者への教育に重きをおくべきであると考える．

Key word　誤嚥，ドパミン-サブスタンス P 系ニューロン，transient receptor potential，嗅覚刺激，摂食

　19 世紀に Osler 先生は「肺炎は老人の友である」と指摘したが，わが国における高齢者肺炎の前向き調査結果(1 年間，全 20 施設)によると，全肺炎症例の約 67%，院内肺炎の 87%が誤嚥由来であった[1]．実際，高齢者は誤嚥リスクの高い状態にある．このことから，「高齢者をみたら陰に誤嚥あり」で日常診療・看護・介護に臨むことが重要である．

脳と誤嚥

　2006 年からの 2 年間における東北大学病院老年科誤嚥外来受診高齢者の大多数が脳血管障害を有しており，その約半数が認知症を呈していた．Alzheimer 病の高齢者も脳血管障害認知症のほぼ同数おり，嚥下障害・誤嚥性肺炎に遭遇したら脳の診断が肝要となる．

　肺炎を繰り返し罹患した高齢者の脳ではラクナ梗塞が大脳基底核・内包近傍の穿通枝領域に多発しており，この部位でのドパミン代謝障害が観察されるとともに，その下位のサブスタンス P 神経系の障害，さらに食物や唾液などの口腔内分泌物が咽頭に接したときに惹起する嚥下反射および誤って気管内に入り込んだものを気管外に排出するための咳反射の劣化が認められる[2](図 1)．とくに，ラクナ梗塞が大脳基底核など穿通枝領域に存在する群では，片側・両側梗塞に従って覚醒時に比べ夜間睡眠中における嚥下反射の劣化が認められ，それに応じて肺炎の発症率も増加する．無症候性脳梗塞でも同様で，2 年間の肺炎発症率は対照群の 4 倍に上る．また，延髄嚥下中枢のその上位中枢として島回が嚥下運動に重要にかかわる．被験者が水や唾液を飲んだとき(自発性嚥下運動)，島回・中心前回などの部位が活性化する．これまで誤嚥性肺炎罹患で当科入院患者の脳解析で
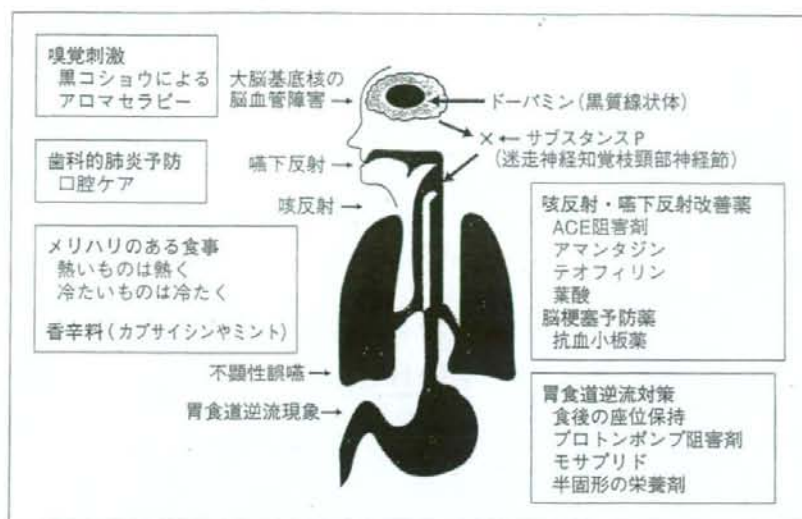
図 1 高齢者摂食嚥下障害および誤嚥性肺炎の予防・治療法

は島回の血流低下が認められ, 島回関連の血管病変の有無も誤嚥の発症に重要である[3]. Alzheimer病においても, 初期は反射的な嚥下機構は比較的保たれているが, 進行とともに肺炎を合併してくる.

著者らの調査では, 繰り返す肺炎罹患の高齢者は嚥下反射潜時5秒以上(正常値3秒以内), 咳反射感受性検査(クエン酸法)3.75 log mg/ml 以上であった[4]. 実際の診療では嚥下反射の低下があり, おそらく誤嚥していても咳反射が保持されている場合は肺炎になりにくい. 嚥下および咳の両反射が低下している場合は肺炎を高率に発症しやすい.

治療(図1)

誤嚥性肺炎の治療としては, 絶食下での抗生物質投与が必要である. 誤嚥のおもな原因が脳血管障害である場合はドパミン-サブスタンスP系ニューロンの賦活が主体である. サブスタンスP分解抑制作用をもつアンジオテンシン変換酵素阻害剤(ACE阻害剤), およびドパミン遊離促進剤, 抗血小板剤のシロスタゾールを投与する. さらにわれわれの誤嚥外来の日常診療では, 脳起因に次いで食道裂孔ヘルニア, 胃切除, 円背による胃食道逆流(GER)起因の誤嚥が多い. GERはたとえ少量でも誤嚥の機会を増やす. 著者らの研究では,

pH4以下の液体でヒト嚥下反射は有意に負の相関を示す. GERをきたしやすい病態を有するときは, 食後2時間以上の座位とともにプロトンポンプ阻害剤およびセロトニン受容体作動薬を投与する.

肺炎を罹患した場合, 当初は抗生物質が奏功しても背後に不顕性誤嚥が存在する場合には肺炎はなかなか軽快しない. そのうち喀痰よりMRSAの出現をみることがよくある. このことから抗生物質の投与後, 熱・炎症マーカーの改善をもって図1に示した薬剤投与あるいはケアが有用と考えられる. 当院に誤嚥性肺炎で入院した高齢者に, 抗生物質投与単独群と, ACE阻害剤およびドパミン剤の投与追加群と比較すると, 投与群において抗生物質使用日数, 入院日数, MRSA感染率, 医療費, 院内死亡率が抑制できた[4].

看護・介護ケア(図1)

誤嚥防止には看護・介護によるケアが重要な位置を占める.

1. Transient receptor potential(TRP)刺激

末梢知覚神経終末に, 6回膜貫通領域をもつ, 非選択性カチオンチャネルである温度感受性受容体(transient receptor potential：TRP)スーパーファミリーの存在が判明している. 温度感受性として, 6つの $TRPV_1$, $TRPV_2$, $TRPV_3$, $TRPV_4$,
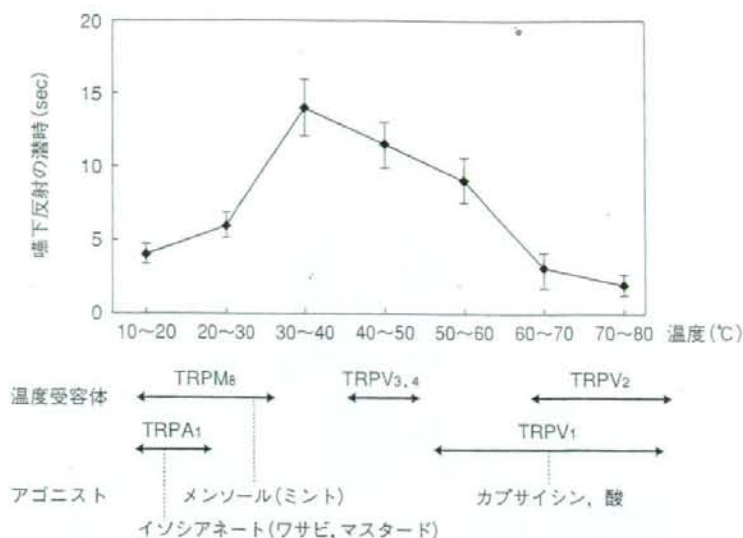
図 2 TRPと嚥下反射

TRPM$_8$, TRPA$_1$が知られており，それぞれ活性化温度閾値が存在する（TRPV$_1$＞43℃，TRPV$_2$＞52℃，TRPV$_3$＞32〜39℃，TRPV$_4$＞27〜35℃，TRPM$_8$＜25〜28℃，TRPA$_1$＜17℃）．TRP は無髄 C 線維に発現すると思われ，温度だけでなくその他の刺激でも直接活性化されるポリモーダル受容器である．そして，この末梢感覚神経に入力された温度受容が電気信号に変換されてその情報が中枢まで伝達されると考えられる．

① 食事の温度……誤嚥しにくい食事として"物粘性"が重要である一方，経口摂取時の食物温度も重要である．著者らが調査したヒト嚥下反射と食事温度の関係はベル型（図 2 上部）であった．これは，咽頭知覚神経上の TRPV$_1$, TRPV$_2$, TRPM$_8$, TRPA$_1$が嚥下反射の活性化に関与する可能性を示唆する（図 2 下部）[5]．つまり，体温に近い温度受容では嚥下反射の劣化が認められていても，熱温度あるいは冷温度受容では嚥下反射は有意に改善する．これは，「温かいお料理は熱い，または温かい温度で，冷たいお料理は冷たい温度で食するのがよい」ということを意味する．つくりおきはいけない．

② 味付け……嚥下反射は，唐辛子の辛味成分であるカプサイシン（TRPV$_1$アゴニスト）による急性直接刺激により濃度依存性に改善する[2]．さらに，カプサイシン含有トローチを用いた慢性刺激

効果の調査についても，脱感作を示唆するような症状なく嚥下・咳反射の改善が認められた[6]．一方，清涼感をもたらすミントの主成分のメンソール（TRPM$_8$アゴニスト）は，氷水と同様，嚥下反射潜時を濃度依存性に改善した[7]．メンソール味付けの食品（例：ミント入りゼリー）が摂食開始時の嚥下訓練食として適していると思われる．

③ 口腔ブラッシング……施設で 1 カ月間，歯・歯肉・舌など口腔内を 1 カ所当り 10 回ずつブラッシングを行ったところ，平均約 9 秒の嚥下反射潜時が介入後第 3 日より改善し，第 30 日は約 4.3 秒と唾液中のサブスタンス P 増加とともに改善した[8]．また，咳反射の改善を認められた．また，全国老人保健施設で行った口腔ケア介入試験（2 年間）では歯のある・なしにかかわらず，非介入群は肺炎発症リスクが 1.7 倍高いことが示された[9]．この研究では非介入群は認知機能の自然低下が認められたのに対し，介入群では認めなかった．口腔ケアはグラム陰性嫌気性菌など口腔内雑菌を排除し，口腔内環境を改善するだけでなく，ブラッシングという機械的刺激が口腔内知覚神経のTRP に入力し，その情報が嚥下や咳の関連中枢へと伝達され，末梢・中枢性に神経伝達物質の放出を促し，上気道防御反射の改善・認知機能の低下抑制をもたらす．

2. 嗅覚刺激

誤嚥予防の薬物療法は誤嚥リスクのある人に薬を内服させるという矛盾を含んでおり，低ADL・意識レベルの人には難しい．そこで著者らは，このような経口摂取が困難な高齢者に対する摂食嚥下改善法として嗅覚刺激による方法を考案した．黒コショウ精油による1日3回の嗅覚刺激は，上位嚥下中枢の島回の脳血流を回復させ，嚥下反射および嚥下運動を改善させる(図3)[10]．ドラッグガスデリバリーシステムにより，この有効芳香成分を24時間連続して成分をターゲットまで輸送する，黒コショウ精油の芳香成分を含有したパッチタイプシートを首周辺の下着や衣服に貼ることにより夜間の不顕性誤嚥を防ぐことができる(異食症の高齢者には衣服の襟足などに貼る)(図4)．また，この黒コショウ精油による嗅覚刺激は前帯状回も活性化する．長期経管栄養施行の重症心身障害児において黒コショウ精油嗅覚刺激によって経口摂取量が回復した(図5)．食べる意欲そのものがない高齢者にも効果が期待できる．

摂食再開プログラム

誤嚥性肺炎など絶食下での治療を要する段階か
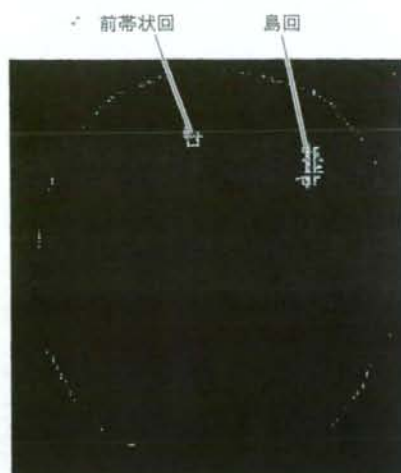

図3　黒コショウ芳香による脳血流改善効果

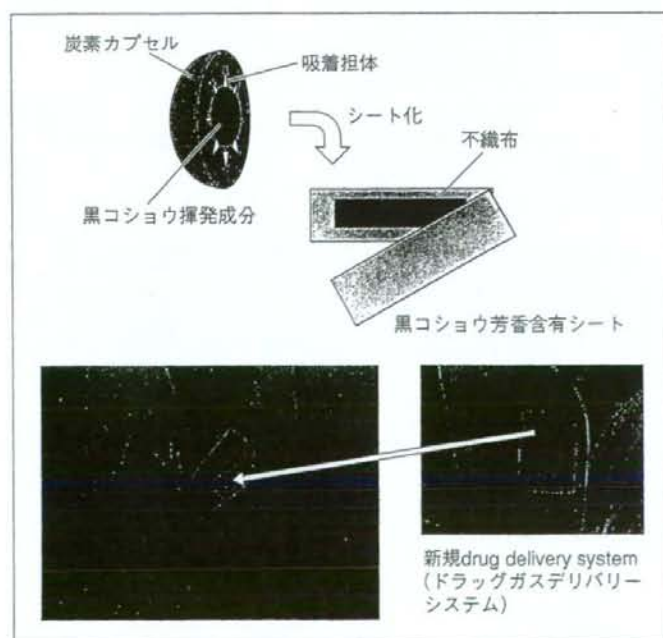ら回復し，摂食を再開する際に，誤嚥を引き起こすことがよくある．そこで，東北大学老年科における摂食再開プログラムを紹介する(図6)．急性期をすぎたころから絶食下のまま黒コショウ芳香含有シート貼布による不顕性誤嚥防止を行う．つぎに，カプサイシントローチによる口腔内の知覚神経刺激を行う．その後，メンソール含有ゼリー

図4　黒コショウ芳香含有シートのドラッグガスデリバリーシステム