

recurrent Graves' hyperthyroidism. Furthermore, thyroid glands that previously have not responded to RAI treatment may be less susceptible to early hypothyroidism than RAI-naive thyroid glands.

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This project is dedicated to the memory of Dr. J.P. Maclean, a respected colleague and dear friend who devoted much of his professional life to the care of the patients on whom this article is based. We also recognize the important contribution of D. McDonald, in performing much of the extraction and entry of data.

REFERENCES

- Ross DS. Current therapeutic approaches to hyperthyroidism. *Trends Endocrinol Metab* 1993;4:281-285.
- Sridama V, McCormick M, Kaplan EL, Fauchet R, DeGroot LJ. Long-term follow-up study of compensated low-dose ¹³¹I therapy for Graves' disease. *N Engl J Med* 1984;311:426-432.
- Kung AWC, Choi P, Lam KSL, Pun KK, Wang C, Yeung RTT. Discriminant factors affecting early outcome of radioiodine treatment for Graves' disease. *Clin Radiol* 1990;42:52-54.
- DeGroot LJ, Manglabruks A, McCormick M. Comparison of RA ¹³¹I treatment protocols for Graves' disease. *J Endocrinol Invest* 1990;13:111-118.
- Ratcliffe GE, Fogelman I, Maisey MN. The evaluation of radioiodine therapy for thyroid patients using a fixed-dose regime. *Br J Radiol* 1986;59:1105-1107.
- Watson AB, Brownlie BEW, Frampton CM, Turner JG, Rogers TGH. Outcome following standardized 185 MBq dose ¹³¹I therapy for Graves' disease. *Clin Endocrinol (Oxf)* 1988;28:487-496.
- Nordyke RA, Gilbert FI. Optimal iodine-131 dose for eliminating hyperthyroidism in Graves' disease. *J Nucl Med* 1991;32:411-416.
- Harbert JC. Radioiodine therapy of hyperthyroidism. In: Harbert JC, Eckelman WC, eds. *Nuclear medicine diagnosis and therapy*. New York: Thieme Medical Publishers; 1996:951-973.
- Hayes AA, Akre CM, Gorman CA. Iodine-131 of Graves' disease using modified early iodine-131 uptake measurements in therapy dose calculations. *J Nucl Med* 1990;31:519-522.
- Hennessey JV, Berg LA, Ibrahim MA, Markert RJ. Evaluation of early (5-6 hours) iodine 123 uptake for diagnosis and treatment planning in Graves' disease. *Arch Intern Med* 1995;155:621-624.
- Kung AWC, Pun KK, Lam KSL, Choi P, Wang C, Yeung RTT. Long-term results following ¹³¹I treatment for Graves' disease in Hong Kong Chinese—discriminant factors predicting hypothyroidism. *Q J Med* 1990;76:961-967.
- Goolden AWG, Stewart JSW. Long-term results from graded low dose radioactive iodine therapy for thyrotoxicosis. *Clin Endocrinol* 1986;24:217-222.
- Berglund J, Christensen SB, Dymling JF, Hallengren B. The incidence of recurrence and hypothyroidism following treatment with antithyroid drugs, surgery or radioiodine in all patients with thyrotoxicosis in Malmo during the period 1970-1974. *J Intern Med* 1991;229:435-442.
- Lowdell CP, Dobbs HJ, Spathis GS, McCready VR, Cosgrove DO, Harmer CL. Low-dose ¹³¹I in treatment of Graves' disease. *J R Soc Med* 1985;78:197-202.
- Törning O, Tallstedt L, Wallin G, et al. Graves' hyperthyroidism: treatment with antithyroid drugs, surgery, or radioiodine—a prospective randomized study. *J Clin Endocrinol Metab* 1996;81:2986-2993.
- Willemsen UF, Knesewitsch P, Kreisig T, Pickardt CR, Kirsch CM. Functional results of radioiodine therapy with a 300-Gy absorbed dose in Graves' disease. *Eur J Nucl Med* 1993;20:1051-1055.
- Hardisty CA, Jones SJ, Hedley AJ, Munro DS, Bewsher PD, Weir RD. Clinical outcome and costs of care in radioiodine treatment of hyperthyroidism. *J R Coll Physicians Lond* 1990;24:36-42.
- De Bruin TWA, Croon CDL, De Klerk JMH, Van Isselt JW. Standardized radioiodine therapy in Graves' disease: the persistent effect of thyroid weight and radioiodine uptake on outcome. *J Intern Med* 1994;236:507-513.
- Crooks J, Buchanan WW, Wayne EJ, McDonald E. Effect of pretreatment with methylthiouracil in results of ¹³¹I therapy. *Br Med J* 1960;1:151-154.
- Connell JMC, Hilditch TE, Robertson J, Coghill G, Alexander WD. Radioprotective action of carbimazole in radioiodine therapy for thyrotoxicosis—influence of the drug on iodine kinetics. *Eur J Nucl Med* 1987;13:358-361.
- Velkeniers B, Cytryn R, Vanhaelst L, Jonckheer MH. Treatment of hyperthyroidism with radioiodine: adjunctive therapy with antithyroid drugs reconsidered. *Lancet* 1988;i:1127-1129.
- Solomon B, Glinoe D, Lagasse R, Wartofsky L. Current trends in the management of Graves' disease. *J Clin Endocrinol Metab* 1989;70:1518-1522.
- Barandes M, Hurley JR, Becker DV. Implications of rapid intrathyroidal iodine turnover for ¹³¹I therapy: the small pool syndrome. *J Nucl Med* 1973;14:79.
- Becker DV, Zanzonico PB, Hurley JR, et al. Treatment of hyperthyroidism with ¹³¹I: continuation of antithyroid drugs does not adversely affect therapy and in "small pool" patients reduces the extrathyroidal radiation dose. In: Gordon A, Gross J, Henneman G, eds. *Progress in thyroid research*. Rotterdam, The Netherlands: Balkema; 1991:223-226.
- Aktay R, Rezaei K, Seabold JE, Bar RS, Kirchner PT. Four- to twenty-four-hour uptake ratio: an index of rapid iodine-131 turnover in hyperthyroidism. *J Nucl Med* 1996;37:1815-1819.

Prediction of Prognosis in Peripheral Facial Nerve Paralysis Using Submandibular Gland Scintigraphy

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In this study, we evaluated the ability of submandibular gland scintigraphy to predict the prognosis of peripheral facial nerve paralysis. **Methods:** Submandibular gland scintigraphy was performed in 78 patients with acute peripheral facial nerve paralysis. After injection of 180-370 MBq [^{99m}Tc]pertechnetate, serial 1-min images were acquired for 25 min. At 15 min after injection of radionuclide, ascorbic acid was administered intraorally to stimulate salivary secretion. Regions of interest were set manually on both submandibular glands, and time-activity curves were generated. The ratios of peak count density (PCR) and washout (WR) of the affected side to the normal side were calculated. Parameters of ≥ 0.8 suggested normal affected submandibular function and indicated a good prognosis. **Results:** Complete recovery of facial nerve paralysis was observed in 52 of 78 patients. The sensitivity, speci-

ficity and accuracy of PCR for a good prognosis were 79%, 50% and 69%, and those of WR were 85%, 77% and 82%, respectively. Positive and negative predictive values for a good prognosis were 76% and 54% in PCR and 88% and 71% in WR, respectively. When WR obtained within 14 days of the onset was used, positive and negative predictive values for a good prognosis were 94% and 73%, respectively. None of the eight patients who had values of < 0.8 for both parameters within 14 days of the onset recovered completely. **Conclusion:** Submandibular gland scintigraphy can serve as a reliable indicator to predict the prognosis of acute peripheral facial nerve paralysis in its early symptomatic period.

Key Words: salivary gland scintigraphy; submandibular gland; Bell's palsy; Ramsay Hunt syndrome; facial nerve paralysis

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The prognosis of peripheral facial nerve paralysis is usually good (1-4); however, once irreversible pathological changes occur and neurolysis develops, it is almost impossible to

recover facial nerve function. Therefore, it is important to assess the prognosis in the early symptomatic period to plan treatment, including surgical intervention. For this purpose, several procedures, including nerve excitability tests (5), maximal stimulation tests (6), evoked electromyography (7,8) or electroneurography (5,9), stapedial reflex tests, lacrimation tests (10), salivary flow tests (6,11) and blink tests (12), have been used.

Salivary secretion from the submandibular glands depends on the integrity of parasympathetic division of the facial nerve (chorda tympani nerve). Salivary flow test was introduced by Magielski and Blatt (11), and May (6) reported that the test offered prognostic information at an early stage. However, this test requires administrator skill and is uncomfortable for patients (13). In contrast, salivary scintigraphy with [^{99m}Tc]pertechnetate can be performed easily in physiologic conditions and only requires that the patient remain in the supine position for about 30 min (14–16).

This study was performed to evaluate the ability of submandibular gland scintigraphy to predict prognosis in patients with peripheral facial nerve paralysis.

MATERIALS AND METHODS

Patients

The study group consisted of 78 consecutive patients (43 men, 35 women; age range 3–71 yr; mean age = 45 ± 17 yr) with peripheral unilateral acute facial nerve paralysis who underwent submandibular scintigraphy. Sixty-two patients were diagnosed as having Bell's palsy, and 16 patients were diagnosed as having Ramsay Hunt syndrome by clinical and immunological findings (in acute and convalescent serum antibody titers to varicella-zoster virus and herpes simplex virus). Forty-two patients had facial paralysis on the left side, and 36 had paralysis on the right side.

Submandibular Gland Scintigraphy

Just after the intravenous administration of 180–370 MBq [^{99m}Tc]pertechnetate, anterior sequential imaging was performed every minute for 25 min using a gamma camera equipped with a low-energy, high-resolution, parallel-hole collimator, with energy discrimination centered on 140 keV and a 20% window. To stimulate salivary secretion, ascorbic acid (250 mg) was administered intraorally 15 min after an injection of [^{99m}Tc]pertechnetate.

Data Analysis

A region of interest (ROI) was set manually on the submandibular gland of the normal side using the image obtained by summing the 1- to 15-min images, and a symmetrical ROI was set on the affected side. Then, the time-activity curves of both submandibular glands were generated. Percent washout (WO) was calculated by the following formula: $WO = 100 \cdot (PC - LC) / PC$, where PC = peak count before ascorbic acid administration and LC = lowest count after ascorbic acid administration. Because [^{99m}Tc]pertechnetate accumulation and washout patterns are variable even in normal populations, we used each patient's normal side as a control; accordingly, the ratios of peak count density (PCR) and washout ratio (WR) of the affected side to normal side were calculated as functional parameters. When WR and PCR were equal to or >0.8, each parameter as a submandibular gland function of the side of facial paralysis was considered normal. These parameters obtained within and after 14 days of the onset of facial paralysis were analyzed with respect to the prognosis.

Evaluation of Facial Paralysis

The degree of facial nerve paralysis was scored using a modified method (12) based on the system proposed by May (17). Ten inspections, including tone, wrinkling of forehead, closing eyes

gently, closing eyes tightly, blinking, wrinkling nose, grinning, whistling, blowing out cheeks and depressing lower lip, were performed. In each analysis, a score of 4 points was awarded if the function was normal, 2 points if it was present but weak and 0 if it was absent, giving a total of 40 points if the involved side was exactly like the normal side. The degree of facial nerve paralysis was evaluated at the first examination and re-evaluated at least up to 6 mo later if the paralysis had not completely recovered. When the total score was 36 points or more and without synkinetic movement, facial spasm and gustatory lacrimation in the follow-up examination, facial paralysis was considered to be completely recovered.

Electroneurography

Thirty-three patients underwent electroneurography several times within 3 wk of the onset of the palsy. Using a pair of electrodes on the stylomastoid foramen region, the facial nerve was stimulated by a square wave of 0.2 msec duration at an intensity exceeding the maximal stimulation. Compound muscle action potential was recorded from a surface electrode on the orbicularis oris muscle, and the ratio of compound muscle action potential of the affected side to normal side was calculated (5,9). When the minimal ratio in each patient was <0.1, the nerve function was considered to be severely damaged.

Statistical Analysis

All data are expressed as mean ± s.d. Unpaired Student's t-test (two-tailed) was used to compare differences between the two groups. A p value of < 0.05 was considered to be significant.

RESULTS

All patients were treated conservatively, including steroid therapy, vitamin B complex administration and stellate ganglion block. Antiviral therapy was administered in all patients with Ramsay Hunt syndrome. Complete recovery of facial nerve paralysis was observed in 52 of 78 patients. Initial score of facial paralysis was significantly higher in the patients with complete recovery than in those with incomplete recovery (9.4 ± 7.6 compared to 4.5 ± 5.4 , $p < 0.01$). Final scores of facial nerve paralysis in patients with and without complete recovery were 39 ± 1.8 and 21 ± 10 , respectively.

Submandibular gland scintigraphy was performed 12 ± 5.9 days (range 3–28 days) after the onset of facial paralysis. The WR in patients without complete recovery of facial paralysis was 0.67 ± 0.20 and was significantly lower than that in patients with complete recovery (0.91 ± 0.17 , $p < 0.0001$). The WR obtained within 14 days of the onset of facial paralysis was also lower in patients without complete recovery than in those with complete recovery (0.62 ± 0.17 compared to 0.89 ± 0.14 , $p < 0.0001$), but WR obtained after 14 days of the onset of the symptom was not significantly lower in patients without complete recovery (0.77 ± 0.22 compared to 0.95 ± 0.23 , $p = 0.08$) (Fig. 1). The PCR in patients without complete recovery of facial paralysis was also significantly lower than in patients with complete recovery (0.79 ± 0.20 compared to 0.93 ± 0.19 , $p < 0.005$). The PCR obtained within 14 days of the onset of facial paralysis tended to be lower in patients without complete recovery than in those with complete recovery (0.82 ± 0.21 compared to 0.92 ± 0.16 , $p = 0.06$), and PCR obtained after 14 days of the onset of the symptom was significantly lower in patients without complete recovery (0.73 ± 0.18 compared to 0.96 ± 0.26 , $p < 0.05$) (Fig. 2).

The relationship between submandibular gland functional parameters (WR and PCR) and prognosis of facial paralysis is summarized in Table 1. Diagnostic values for prognosis are summarized in Table 2. Sensitivity, specificity and accuracy of

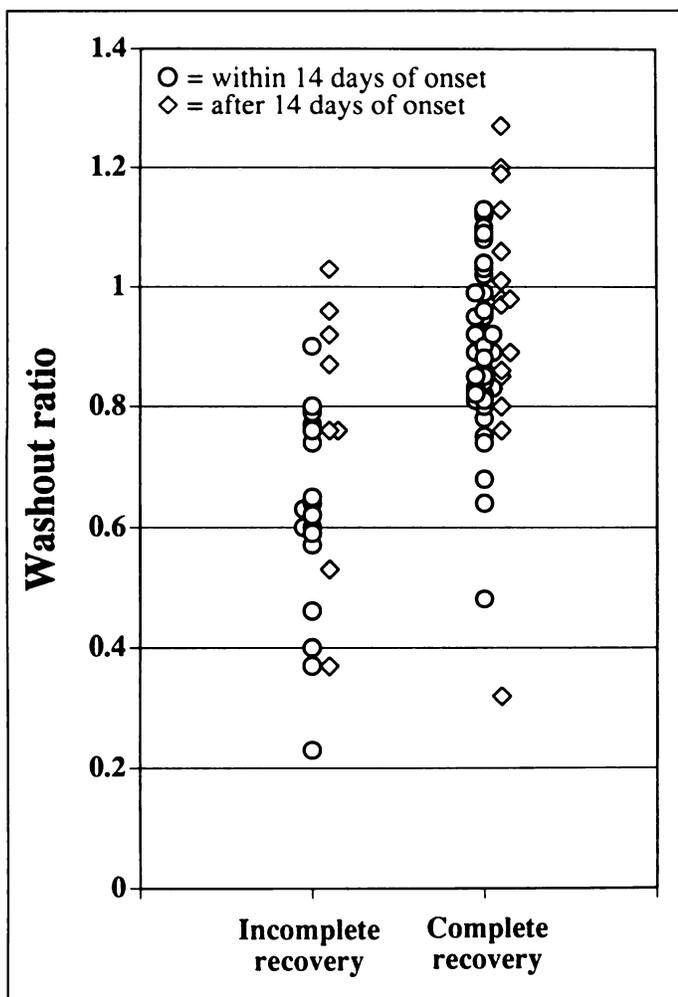


FIGURE 1. WRs were plotted in patients with complete and incomplete recovery of acute peripheral facial nerve paralysis. Open circles and rhombuses demonstrate WRs obtained within 14 days of the onset and >14 days after the onset of the symptom, respectively. The WR in patients without complete recovery of facial paralysis was significantly lower than that in patients with complete recovery (0.67 ± 0.20 compared to 0.91 ± 0.17 , $p < 0.0001$). The WR obtained within 14 days of the onset of facial paralysis was also lower in patients without complete recovery than in those with complete recovery (0.62 ± 0.17 compared to 0.89 ± 0.14 , $p < 0.0001$), but the WR obtained after 14 days of the onset of the symptom was not significantly lower in patients without complete recovery (0.77 ± 0.22 compared to 0.95 ± 0.23 , $p = 0.08$).

WR for complete recovery of facial paralysis were 85% (44/52), 77% (20/26) and 82% (64/78), respectively, and those of PCR were 79% (41/52), 50% (13/26) and 69% (54/78), respectively. Positive and negative predictive values of WR were 88% (44/50) and 71% (20/28), and those of PCR were 76% (41/54) and 54% (13/24), respectively. When the parameters of submandibular gland scintigraphy performed within 14 days of the onset of facial paralysis were analyzed, sensitivity, specificity and accuracy of WR for complete recovery of facial paralysis were 84% (31/37), 89% (16/18) and 85% (47/55), respectively, and those of PCR were 81% (30/37), 44% (8/18) and 69% (38/55), respectively. Positive and negative predictive values of WR obtained within 14 days of the onset of facial paralysis were 94% (31/33) and 73% (16/22), and those of PCR were 75% (30/40) and 53% (8/15), respectively.

When both parameters were ≥ 0.8 , most patients' facial paralysis recovered completely (89%, 34 of 38 patients), especially when scintigraphy was performed within 2 wk of the onset (92%, 24/26). When both parameters were < 0.8 , only 1

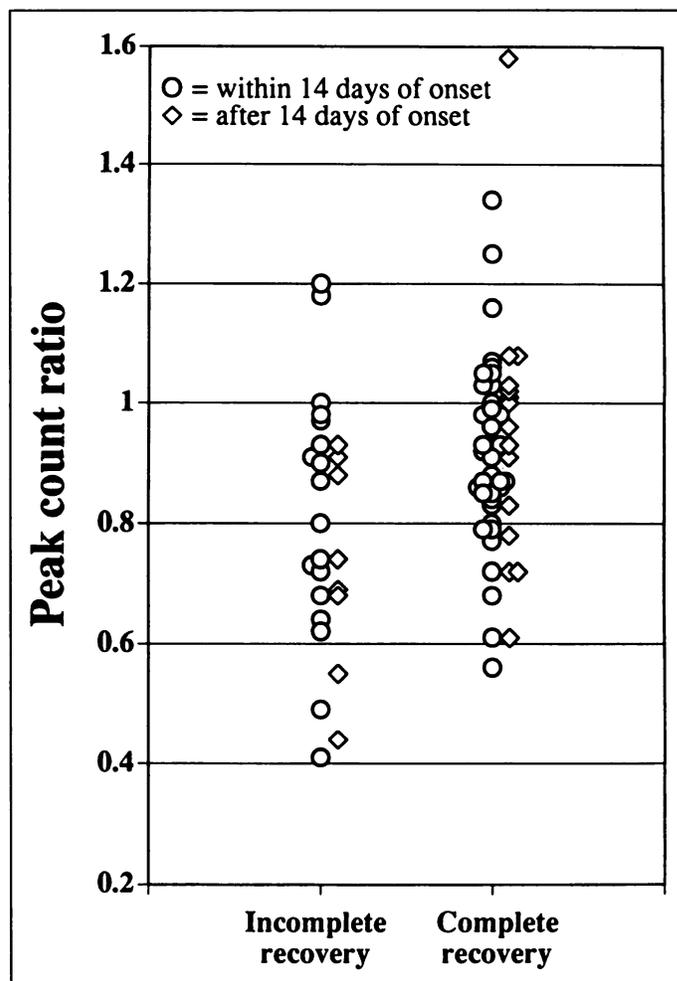


FIGURE 2. PCRs were plotted in patients with complete and incomplete recovery of acute peripheral facial nerve paralysis. Open circles and rhombuses demonstrated the PCRs obtained within 14 days of the onset and >14 days after the onset of the symptom, respectively. The PCR in patients without complete recovery of facial paralysis was significantly lower than in patients with complete recovery (0.79 ± 0.20 compared to 0.93 ± 0.19 , $p < 0.005$). PCR obtained within 14 days of the onset of facial paralysis tended to be lower in patients without complete recovery than in those with complete recovery (0.82 ± 0.21 compared to 0.92 ± 0.16 , $p = 0.06$), and PCR obtained after 14 days of the onset was significantly lower in patients without complete recovery (0.73 ± 0.18 compared to 0.96 ± 0.26 , $p < 0.05$).

of 12 patients showed complete recovery, and no patients recovered completely when scintigraphy was performed within 2 wk of the onset (zero of eight patients).

In Bell's palsy, sensitivity, specificity, accuracy, positive and

TABLE 1
Relationship Between Submandibular Gland Function and Prognosis of Facial Nerve Paralysis

	Prognosis of facial paralysis		Total
	Complete recovery	Incomplete recovery	
Both WR and PCR ≥ 0.8	34 (24)	4 (2)	38 (26)
WR ≥ 0.8 and PCR < 0.8	10 (7)	2 (0)	12 (7)
WR < 0.8 and PCR ≥ 0.8	7 (6)	9 (7)	16 (13)
Both WR and PCR < 0.8	1 (0)	11 (8)	12 (8)
Total	52 (37)	26 (17)	78 (54)

Numbers in parentheses denote patients who underwent submandibular gland scintigraphy within 2 wk of the onset of the facial nerve paralysis. WR = washout ratio; PCR = peak count density ratio.

TABLE 2
Prognostic Diagnosis by Submandibular Gland Scintigraphy

	Submandibular gland scintigraphy		
	Within 14 days	After 14 days	Whole study
WR			
Sensitivity	84% (31/37)	87% (13/15)	85% (44/52)
Specificity	89% (16/18)	50% (4/8)	77% (20/26)
Accuracy	85% (47/55)	74% (17/23)	82% (64/78)
Positive predictive value	94% (31/33)	76% (13/17)	88% (44/50)
Negative predictive value	73% (16/22)	67% (4/6)	71% (20/28)
PCR			
Sensitivity	81% (30/37)	73% (11/15)	79% (41/52)
Specificity	44% (8/18)	63% (5/8)	50% (13/26)
Accuracy	69% (38/55)	70% (16/23)	69% (54/78)
Positive predictive value	75% (30/40)	79% (11/14)	76% (41/54)
Negative predictive value	53% (8/15)	56% (5/9)	54% (13/24)

WR = washout ratio; PCR = peak count density ratio.

negative predictive values of WR for complete recovery of facial paralysis were 84% (36/43), 74% (14/19), 81% (50/62), 88% (36/41) and 67% (14/21), respectively, and those of PCR were 77% (33/43), 58% (11/19), 71% (44/62), 80% (33/41) and 52% (11/21), respectively. In Ramsay Hunt syndrome, sensitivity, specificity, accuracy, positive and negative predictive values of WR for complete recovery of facial paralysis were 89% (8/9), 86% (6/7), 88% (14/16), 89% (8/9) and 86% (6/7), respectively, and those of PCR were 89% (8/9), 29% (2/7), 63% (10/16), 62% (8/13) and 67% (2/3), respectively.

In electroneurography, sensitivity, specificity and accuracy for complete recovery of facial paralysis were 74% (14/19), 36% (5/14) and 58% (19/33), respectively. Positive and negative predictive values were 61% (14/23) and 50% (5/10), respectively.

Representative cases are presented in Figures 3 and 4. Salivary scintigraphy performed 6 days after the onset of left Bell's palsy in a 53-yr-old woman demonstrated poor visualization and poor stimulatory secretion of the left submandibular gland (Fig. 3A). The time-activity curve of submandibular gland activity showed that the PCR was 0.72 and WR was 0.37 (Fig. 3B). The score of facial paralysis was 0 when the scintigraphy was performed. Recovery of the facial paralysis was incomplete (score of 20 6 mo later). A 20-yr-old woman with left complete facial paralysis showed normal function of submandibular gland with PCR (0.93) and WR (0.88) 9 days after the onset of Bell's palsy (Fig. 4). The patient's paralysis recovered from a score of 0 to 38 about 2.5 mo later.

DISCUSSION

This study demonstrated that submandibular gland scintigraphy could predict the prognosis of peripheral facial nerve paralysis in the early symptomatic period. As a parameter, WR was more reliable and showed an excellent positive predictive value for complete recovery (94% when scintigraphy was performed within 14 days of the onset) and a good negative predictive value (73% when scintigraphy was performed within 14 days of the onset). When both PCR and WR obtained within 14 days of the onset were <0.8 , no patients showed complete recovery (zero of eight patients), suggesting that these criteria are good indications for surgical intervention.

The salivary flow test has been considered to be useful for predicting prognosis, especially in the very early symptomatic period, even when electrical tests are normal (18). However, the

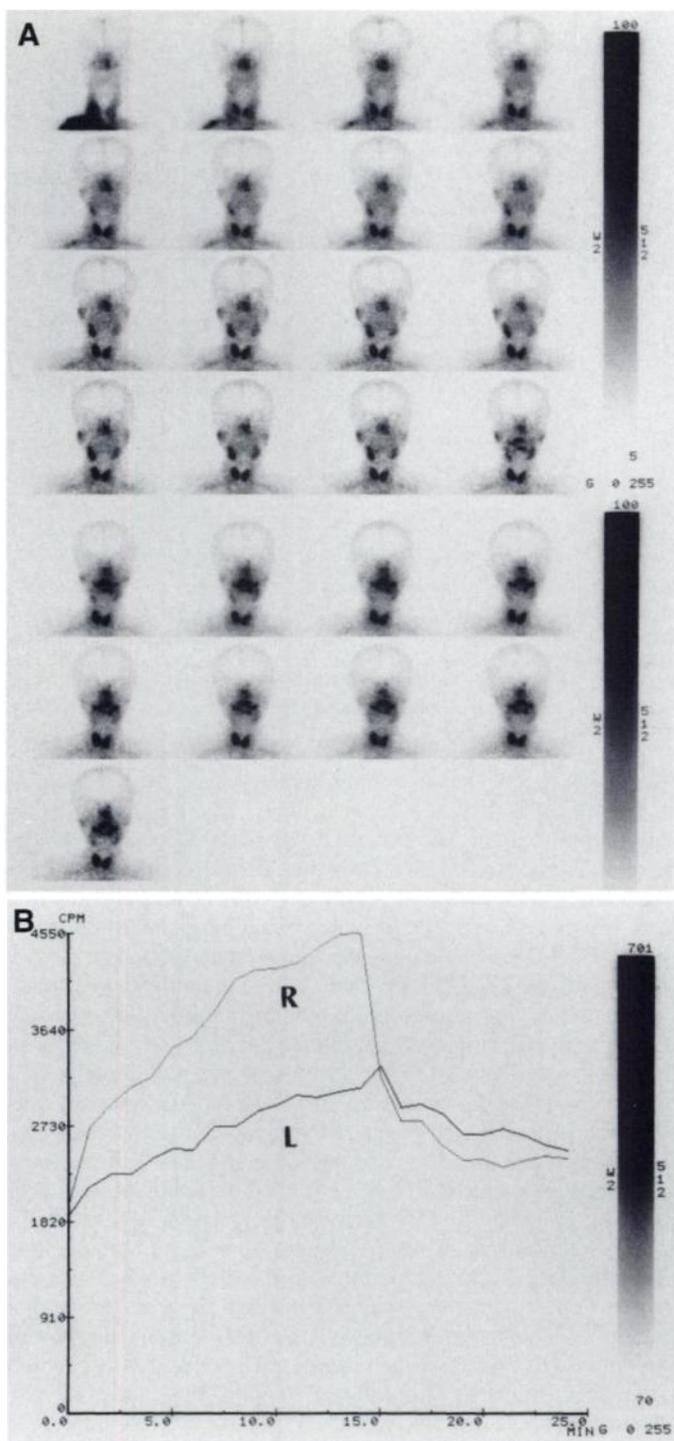


FIGURE 3. Salivary scintigraphy performed 6 days after the onset of left complete facial paralysis (Bell's palsy) in a 53-yr-old woman. Sequential 1-min images are shown from the upper left to the lower right. Stimulation of salivary secretion was performed at 15 min after administration of radionuclide (end of the 15th imaging). (A) Poor visualization and poor stimulatory secretion of the left submandibular gland were observed. (B) Time-activity curve of the submandibular gland indicated that PCR was 0.72 and WR was 0.37. Recovery of the facial paralysis was incomplete (score improved insufficiently from 0-20 at final examination). R = right submandibular gland; L = left submandibular gland.

salivary flow test is rarely used at present because it is difficult to perform, making it relatively inaccurate sometimes. Also, it is uncomfortable for the patient (13). The insertion of polyethylene tubing into Wharton's duct openings may be difficult and unpleasant to the patient. Repetition of the test may also become impossible because of edema of the ducts. Pressure or kinking

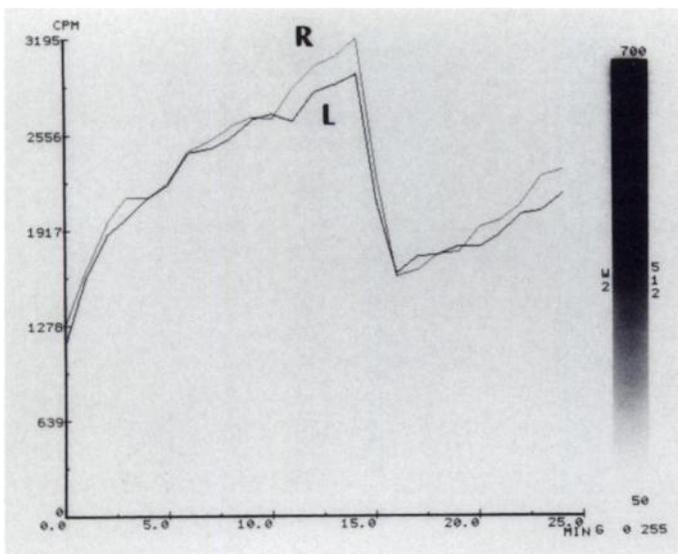


FIGURE 4. Time-activity curve obtained 9 days after the onset of left Bell's palsy (score 0) in a 20-yr-old woman. After oral administration of ascorbic acid at 15 min, radionuclide activity of both submandibular glands decreased rapidly. The PCR (0.93) and WR (0.88) indicated a good prognosis. This patient's paralysis recovered completely about 2.5 mo later. R = right submandibular gland; L = left submandibular gland.

of the tubing may lead to an unreliable count of salivary drops. In children, it is difficult to perform (13,15). Because salivary scintigraphy can be performed easily with the patient in the supine position with intravenous administration of [^{99m}Tc]pertechnetate, submandibular gland function can be evaluated more objectively and precisely in physiologic conditions as an uptake of [^{99m}Tc]pertechnetate and washout induced by a salivary secretion stimulant, such as lemon and ascorbic acid.

Few studies focusing on prediction of the prognosis of facial nerve paralysis using submandibular scintigraphy have been reported. Rosen et al. (15) reported that submandibular gland count symmetry (accumulation of [^{99m}Tc]pertechnetate) was related to prognosis; however, they stated that more study was necessary to obtain more conclusive evidence. With salivary scintigraphy, Yamashita et al. (16) used the stimulatory secretion ratio calculated by dividing the peak count before stimulatory secretion by the residual count of submandibular gland after stimulation and compared with the early partial recovery (5th wk) after the onset of facial paralysis. They observed that prediction of early partial improvement was successful in 44 of 56 patients. In this study, we used PCR and WR as functional parameters. Because stimulation of parasympathetic activity produces profuse watery salivary flow, we used a degree of washout of submandibular gland count by stimulation as a functional parameter rather than the ratio of the peak count before stimulation to the residual count after stimulation. Furthermore, we validated the outcome at 6 mo after the onset of the paralysis because very few patients will recover after 6 mo (1,19), and delayed improvement within 6 mo is not thought to be an indication for surgical intervention.

In all of the methods for predicting prognosis, parameters are obtained by evaluating facial nerve function. Electrical tests, including nerve excitability tests, maximal stimulation tests and evoked electromyography or electroneurography, stimulate the facial nerve electrically distal to the stylomastoid foramen and evaluate the electrical response of the facial nerve or facial muscle movement (5-9). The blink test evaluates the muscle movement itself (12). The stapedial reflex test evaluates the stapedius muscle response to strong sound. The lacrimal and

salivary flow tests evaluate the function of the parasympathetic division of the facial nerve (greater superficial petrosal nerve and chorda tympani nerve respectively) indirectly through lacrimal and submandibular gland function (6,10,11). Intraoperative-evoked electromyography demonstrated that the lesion producing Bell's palsy lies within the facial canal and is situated at the meatal foramen in 94% of patients (8,10). In electrical tests, the nerve is stimulated distal to the stylomastoid foramen; however, submandibular gland function reflects nerve damage, including that proximal to the mastoid segment. Therefore, early after the onset of facial paralysis, the electrical test shows a good response because 4-7 days are needed for the facial nerve to develop Wallerian degeneration (9). On the other hand, submandibular scintigraphy shows abnormal findings at this early stage of the disease (18).

The therapy for facial nerve paralysis is usually conservative. For Bell's palsy, steroid therapy may lessen synkinesis, prevent progression of palsy and shorten the recovery time when given in the first 3 wk after the onset of the palsy (19,20). In Ramsay Hunt syndrome, steroid therapy is also considered to be effective in addition to administration of an antiviral agent (acyclovir) (3,21,22). Recently, acyclovir also has been tried based on the hypothesis that herpes simplex causes Bell's palsy and has been proven to be effective for Bell's palsy (23). However, in both Bell's palsy and Ramsay Hunt syndrome, some patients receiving conservative treatment have a poor outcome and would have been indicated for decompression surgery.

There is some controversy about the significance of decompression surgery in patients with Bell's palsy and Ramsay Hunt syndrome. Studies with surgical treatment did not demonstrate any improvement in prognosis when the decompression was limited to the portion distal to the labyrinthine segment (13,18,24). However, when the meatal segment was decompressed, significant improvement of the prognosis was obtained, and the results were consistent with the observation that the lesion producing Bell's palsy is situated at the meatal foramen in most cases (8,10). Surgery is indicated when the chance of satisfactory return of facial function is unlikely and the majority of endoneural tubes are still intact. Therefore, a reliable prognostic indicator is essential for the determination of surgical treatment within 2-3 wk after the onset of the symptom (8).

Electroneurography is currently considered the most sensitive method for the determination of the surgical indication in the field of otorhinolaryngology (8,13,19). When the compound action potential on the involved side was <10% of normal, unsatisfactory recovery of the facial palsy was observed in 50% of cases in our study and around 30%-79% in the literature (7,8,13). In this study, submandibular gland scintigraphy showed a negative predictive value of >70% for prognosis, and therefore, the finding obtained by this method is considered a reliable indicator of surgical indication. Accordingly, submandibular gland scintigraphy should be added as one of the routine diagnostic tests for predicting prognosis.

A pitfall of the method might be the presence of prior salivary gland disease. The method we used is based on the assumption that both submandibular glands' function was normal before the onset of facial paralysis and evaluates the submandibular function of the affected side by comparing it with other normal side. Therefore, prior disease may influence the analysis. Careful interpretation is needed when a patient has salivary gland disease or a poor uptake of pertechnetate on the side contralateral to that showing facial paralysis.

CONCLUSION

Submandibular scintigraphy revealed excellent diagnostic ability to predict the prognosis in patients with acute peripheral facial nerve paralysis in its early symptomatic period. Consequently, submandibular scintigraphy should be added as one of the routine tests to predict the prognosis of peripheral facial nerve paralysis and, especially, to determine of the indications for surgical therapy.

REFERENCES

1. Peiterson E. The natural history of Bell's palsy. *Am J Otol* 1982;4:107-111.
2. Devriese PP, Schumacher T, Scheide A, DeJongh RH, Houtkooper JM. Incidence, prognosis and recovery of Bell's palsy; a survey of about 1000 patients. *Clin Otolaryngol* 1990;15:15-27.
3. Robillard RB, Hilsinger RL, Adour KK. Ramsay Hunt facial paralysis: clinical analysis of 185 patients. *Otolaryngol Head Neck Surg* 1986;95:292-297.
4. Heathfield KWG, Mee AS. Prognosis of the Ramsay Hunt syndrome. *Br Med J* 1978;1:343-344.
5. Fisch U. Maximal nerve excitability testing versus electroneurography. *Arch Otolaryngol* 1980;106:352-357.
6. May M, Hardin WB, Sullivan J, Wette R. Natural history of Bell's palsy: the salivary flow test and other prognostic indicators. *Laryngoscope* 1976;86:704-712.
7. Sinba PK, Keith RW, Pensak ML. Predictability of recovery from Bell's palsy using evoked electromyography. *Am J Otol* 1994;15:769-771.
8. Fisch U. Surgery for Bell's palsy. *Arch Otolaryngol* 1981;107:1-11.
9. Tojima H, Aoyagi M, Inamura H, Koike Y. Clinical advantages of electroneurography in patients with Bell's palsy within two weeks after onset. *Acta Otolaryngol (Stockh)* 1994;511(suppl):147-149.
10. Fisch U, Esslen E. Total intratemporal exposure of the facial nerve. *Arch Otolaryngol* 1972;95:335-341.
11. Magielski JE, Blatt IM. Submaxillary salivary flow: a test of chorda tympani nerve function as an aid in diagnosis and prognosis of facial nerve paralysis. *Laryngoscope* 1958;68:1770-1789.
12. Mizukoshi K, Watanabe Y, Aso S, Asai M. Prognostic value of blink test in patients with facial paralysis. *Acta Otolaryngol (Stockh)* 1988;446(suppl):70-75.
13. May M, Klein SR, Taylor FH. Idiopathic (Bell's) facial palsy: natural history defies steroid or surgical treatment. *Laryngoscope* 1985;95:406-409.
14. Mishkin FS. Radionuclide salivary gland imaging. *Semin Nucl Med* 1981;11:258-265.
15. Rosen G, Vered IY, Fedcheyn SC. Submandibular salivary gland scan: a prognostic indicator of Bell's palsy. *J Laryngol Otol* 1980;94:1021-1024.
16. Yamashita T, Ino C, Tomoda K, Kumazawa T. Prognostic determination and submandibular function in Bell's palsy. *Arch Otolaryngol* 1989;111:244-248.
17. May M. Facial paralysis, peripheral type: a proposed method of reporting. *Laryngoscope* 1970;80:331-390.
18. May M, Blumenthal F, Taylor F. Bell's palsy: surgery based upon prognostic indicators and results. *Laryngoscope* 1981;91:2092-2103.
19. Hughes GB. Practical management of Bell's palsy. *Otolaryngol Head Neck Surg* 1990;102:658-663.
20. Stankiewicz JA. Steroid and idiopathic facial nerve paralysis. *Otol Head Neck Surg* 1983;91:672-677.
21. Stafford FW, Welch AR. The use of acyclovir in Ramsay Hunt syndrome. *J Laryngol Otol* 1986;100:337-340.
22. Inamura H, Aoyagi M, Tojima H, Koike Y. Effects of acyclovir in Ramsay Hunt syndrome. *Acta Otolaryngol (Stockh)* 1988;446(suppl):111-113.
23. Adour KK, Ruboyanes JM, Von Doerstein PG, et al. Bell's palsy treatment with acyclovir and prednisone compared with prednisone alone: a double-blind, randomized, controlled trial. *Ann Otol Rhinol Laryngol* 1996;105:371-378.
24. Aoyagi M, Koike Y, Ichige A. Results of facial nerve decompression. *Acta Otolaryngol (Stockh)* 1988;466(suppl):101-105.

Pulmonary SPECT Imaging and the Stripe Sign

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A patient with high clinical suspicion for pulmonary embolism underwent a diagnostic scintigraphic ventilation/perfusion scan. The planar images revealed an unmatched perfusion defect with a stripe sign in the right middle lobe. A stripe sign is the appearance of normally perfused tissue between the defect and the pleural surface suggesting a nonpleural-based abnormality. SPECT images acquired in the same study period, however, failed to demonstrate normally perfused tissue between the defect and the pleural surface. Previous studies have compared planar ventilation/perfusion studies with stripe sign perfusion defects to pulmonary angiography. The results suggest that stripe sign perfusion defects are generally not due to emboli. However, planar imaging is projectional and may miss pleural contact in some perfusion lesions depending on the projection. In the absence of SPECT data, the significance of the stripe sign may need to be reassessed.

Key Words: pulmonary embolism; pulmonary angiography; ventilation/perfusion scan

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A stripe sign in a ventilation/perfusion (V/Q) scan is the appearance of normally perfused tissue between a lesion and the pleural surface. The presence of a stripe sign may imply that there is a region of normally perfused tissue distal to the defect. In contradistinction, embolic lesions are believed to be pleural based, with the defect contacting the pleural surface. Nonpleural-based lesions with a stripe sign are thought to be less likely

due to pulmonary embolism (PE). We report a case of a young woman with a clinical history suggestive of PE. A scintigraphic V/Q study was ordered to evaluate segmental ventilation and perfusion. Planar images revealed a large perfusion lesion with a stripe sign in the right middle lobe on the right posterior oblique (RPO) projection. This defect was not matched with a ventilation abnormality. Using ^{81m}Kr as the ventilation agent, our laboratory performed dual-isotope SPECT acquisitions in all patients who could safely undergo the procedure. This technique eliminates the positioning discrepancies created when the perfusion and ventilation images are acquired separately. Images were visualized in the transaxial, sagittal and coronal planes.

The perfusion defect and its ventilation mismatch were well visualized, but a rim of normally perfused tissue could not be identified between the lesion and the pleural surface. This suggests that the limited number of views acquired in planar imaging may fail to locate the point where a perfusion defect contacts the pleural surface. Also, normally perfused tissue adjacent to the lesion, or in the contralateral lung, could mimic a stripe between the defect and the pleural surface. This could be incorrectly interpreted as a less-suspicious, nonpleural-based lesion, and the patient may not get proper treatment. The definition of a stripe sign may need to be reassessed to include the caveat that the stripe must be seen in all of the available perspectives to be labeled a nonpleural-based abnormality. Under these conditions, SPECT imaging should be considered because it is less likely to misrepresent pleural contact in perfusion defects than traditional planar imaging.

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