Rationale and Objectives. Multiple cases of recurrence of aneurysms after endovascular treatment have been reported. The purpose of the current hemodynamic study was to identify changes in shear stress and pressure associated with the recurrence of terminal intracranial aneurysms after endovascular occlusion.

Methods. Using a finite element method, a pulsed flow with a non-Newtonian viscosity of blood was simulated within the aneurysm cavity. A recurrent terminal intracranial aneurysm of a patient originally treated with balloon occlusion was then studied. This was based on a physiologic pulsatile flow, which was observed in the middle cerebral artery. Before and after the balloon occlusion, local maximum wall shear stress and pressure drop at the neck of the aneurysm were calculated and compared with the normal shear stress.

Results. Although the maximum shear stress at the right neck of the aneurysm was significantly reduced after balloon insertion, it was still 2.5 times greater than the normal maximum shear stress. This was attributable to the presence of a portion of the aneurysmal neck (residual neck), which was not obliterated by the balloon. The balloon also helped to reduce the maximum pressure inside the aneurysm by approximately 15%. Hemodynamic changes in the residual aneurysm neck, where the shear stress and the pressure are high, and other factors may be responsible for the recurrence of aneurysms after balloon or coil occlusion.

Conclusion. Residual necks after balloon occlusion, coil occlusion, or both are attributable to the geometric orientation of the aneurysm with respect to parent and daughter vessels and the variety of configurations of the balloon and coils used to occlude the aneurysms. Inadequate reduction in local shear stress found in these residual necks is an important factor in the recurrence and rupture of the aneurysm after endovascular occlusion.

Key Words. Intracranial aneurysm; endovascular treatment; balloon occlusion.
cases, occlusion of the aneurysm is obtained. However, the most disturbing limitation of the endovascular approach—the recurrence of aneurysms after treatment by this procedure—still remains. Although there are reports of regrowth of aneurysm sacs from a residual neck after aneurysm clipping, there are not many in the neurosurgical literature [1–4]. Because of the geometric configuration, size, and location of the aneurysm, complete endovascular occlusion is difficult to obtain from within the vessel. The use of this technique is problematic because the recurrent aneurysm is more difficult to treat and a second endovascular procedure or surgery may carry more risk to the patient than the first one. This type of complication may in turn reduce or even preclude the use of the endovascular technique for treating intracranial aneurysms in the future.

In the current study, we hypothesized that the hemodynamic changes in the nonoccluded area of the aneurysm are responsible for the recurrence and rupture of the aneurysm unless complete occlusion of the aneurysm is obtained. In view of this, we studied a recurrent terminal intracranial aneurysm originally treated by balloon occlusion. Pulsatile flow of the aneurysm is numerically simulated on the basis of the physiologic pulsatile flow observed in the middle cerebral artery. Angiograms of the aneurysm are used for the computer modeling of blood flow in the aneurysmal cavity.

The nature of flow within the aneurysm has been investigated by several researchers. The intraaneurysmal flow pattern is primarily governed by the geometric relation between the aneurysm and the parent vessel [5–9]. In vitro steady-flow experiments in lateral aneurysms have indicated that the inflow into the aneurysm arose from the downstream lip and was directed toward the center of the fundus, whereas the backflow to the parent vessel ran along the wall of the fundus [8, 9]. During the study of the origin and progression of the lateral aneurysm, rapid changes in blood flow direction were observed that resulted in frequent variation in wall shear stress and pressure at the proximal and distal walls of the aneurysmal cavity, rendering continuous damage to the intima at the cavity neck [10]. It also has been reported that partial coil embolization in lateral aneurysms may prevent early rebleeding; however, it may induce additional mural stresses resulting from new hemodynamic forces and compliance mismatch [11]. By contrast, in terminal aneurysms, the flow patterns are different and a stagnant flow region is observed at the center core of the aneurysm [12]. To our knowledge, a quantitative estimate of flow parameters (e.g., velocity profile, shear rate, non-Newtonian viscosity, and wall shear stress before and after occlusion of a terminal aneurysm) has not been documented. The final purposes of our research were to evaluate the effects of the endovascular treatment and to determine how recurrences can be avoided.

MATERIALS AND METHODS

Geometry

The numeric analysis reported in this article was based on preocclusion and postocclusion angiograms of a patient who had a large terminal internal carotid aneurysm. The patient's terminal aneurysm was originally treated by balloon occlusion (Figs. 1A and 1B). However, in an arteriogram performed 2 years after the original treatment, it was noted that there had been a recurrence of the aneurysm, which had a lobulated appearance (Fig. 1C). Subsequent embolization using a detachable coil was performed, resulting in complete occlusion of the aneurysm (Fig. 1D). The films were obtained in a biplane angiographic unit, and the magnification used was approximately 8%. From the angiographic image, digitized information about the geometric coordinates were obtained. The width of the neck of the large terminal aneurysm was 4.9 mm. The maximum width and length of the terminal aneurysm were 12 and 19 mm, respectively. Digitized x- and y-coordinate data were used to create the finite element mesh. The mesh plots for the terminal aneurysm without and with balloon occlusion are shown in the left and right sides of Figure 2, respectively. The vessel diameter at the parent branch was 3.3 mm; at the daughter branches, the diameters were 2.5 and 2.8 mm. The axis of the left branch was at an angle of 112° when measured from the flow axis of parent artery. Similarly, the axis of the right branch made an angle of 293° with respect to the flow axis of the parent artery. The terminal aneurysm could be categorized as a wide-neck aneurysm.

Numeric Formulation

We obtained the time-dependent solution of an incompressible non-Newtonian fluid for selected geometry (Fig. 2). The flow is described by the conservation equations of fluid mass and momentum. We used a finite element method to solve the two conservation
FIGURE 1. Angiogram of a middle cerebral terminal aneurysm of a patient without (A) and with (E) balloon occlusion. Postembolization angiogram, after balloon insertion, is shown in B. Notice the residual neck (open arrows). Recurrence of the aneurysm, after 2 years, has a lobulated appearance (C). A subsequent embolization was performed using the detachable coil technique (D).

FIGURE 2. The mesh plots for the terminal aneurysm without (left) and with (right) balloon occlusion. The illustration on the right includes the residual neck, as can be seen in Figure 1B.

Equations and thus to obtain the velocity, wall shear rate and stress, and pressure distributions. These two equations are as follows:

\[ u_{j,j} = 0 \tag{1} \]

and

\[ \rho \left[ \frac{\delta u_i}{\delta t} + u_j u_{i,j} \right] = \sigma_{ij,j} + \rho f_i \tag{2} \]

where \( i,j = 1,2 \) for axisymmetric flows, \( u_i \) is the component of the velocity vector, \( \rho \) is density, \( \sigma_{ij} \) is stress tensor, and \( f_i \) is the body force. The stress vector, \( s_i \), at a point on the boundary of a fluid element is defined by

\[ s_i = \sigma_{ij} n_j \tag{3} \]

For a known element and its solution field, the stress component, \( s_i \), on the boundary at the Gaussian integra-
tion points is evaluated. Subsequently, the normal and tangential components of stress vectors are obtained after applying the appropriate transformations.

The Galerkin formulation [13], using nine nodal quadrilateral elements, was applied to make discrete the continuity and momentum equations; this resulted in a set of nonlinear algebraic equations of the following form:

$$M \frac{du}{dt} + K(u) u = F,$$

where $K(u)$ is the global system matrix developed from the momentum balance, $M$ is the mass matrix, $u$ is the unknown velocity, and $F$ is the forcing function (including body forces and boundary conditions) [13].

The matrix equation, representing a discrete analog of the original equations for an individual fluid element, was constructed, assembled, and solved. For spatial integration, the number of iteration steps was limited to 10 at each time step with a combination of the successive substitution and quasi-Newton scheme. The numeric simulation of a pulsatile flow requires a time integration method. The implicit time integration scheme used in the current study was the second-order trapezoidal method with a variable time step, which depends on the magnitude of temporal inlet velocity and its gradient change. Depending on the physiologic velocity pulse shape, the time steps vary between $1 \times 10^{-4}$ to $1 \times 10^{-5}$ sec. The finite-element computer code FIDAP (Fluid Dynamics International, Evanston, IL) was used to formulate and solve this matrix equation. An IBM-3090 (IBM, Danbury, CT) was used, and the results were downloaded to a Macintosh II computer (Apple, Cupertino, CA).

For validation of the numeric computation, we performed two separate computer modeling runs at peak systolic flow with different convergence criteria; both the relative velocity error with respect to the previous step and the relative residue error compared with the initial value were set at 2% and 1%, respectively, which was similar to the values used by Banerjee et al. [14]. Furthermore, the overall convergence was confirmed by increasing the total number of meshes by 20% over that of the previous run and by comparing the two results to check for accuracy. When the improvement with 20% more meshes was less than 1% in velocity vectors, wall shear stress, and pressure, the computation was considered to be accurate. (For additional detail on the formulation, see Banerjee et al. [15].) Although the current study was limited and we did not explore the complete three-dimensional flow phenomenon, the flow simulation for the two-dimensional flow offers additional insights into terminal aneurysm flow characteristics. Compared with a two-dimensional pulse flow simulation, a three-dimensional pulse flow requires central processing unit time and memory that were beyond the capacities of our computation facility.

### Boundary Conditions

The numeric calculations were performed for the center line instantaneous velocity and with uniform spatial inlet flow conditions. This was based on configuration of the short entry length of the parent artery. Padayachee [16] showed the in vivo velocity profile at the core region as measured by an ultrasound Doppler flow cuff. The inlet velocity profile was normalized as shown in Figure 3 and was used as an input for the present numeric simulation. The peak inlet Reynolds number during systole was 440, whereas during diastole the Reynolds number was 200. The outflow boundary condition does not need to be specified at the exit, because its values are effectively determined by extrapolation similar to the finite difference schemes. A no-slip boundary condition is specified on the rigid arterial wall.

### Non-Newtonian Blood Model

We used the Carreau model to represent the shear-rate-dependent, non-Newtonian blood viscosity whose model constants were obtained by curve fitting of available shear-rate-dependent blood viscosity data described by Cho and Kensey [17]:

![FIGURE 3. Normalized form of the in vivo velocity pulse in the middle cerebral artery.](image-url)
where \( \lambda \) (characteristic time) = 3.313 sec, \( n = 0.3568 \), \( \eta_0 = 0.56 \) poise, and \( \eta_\infty = 0.0345 \) poise. To calculate viscosity in the flow field locally, the local shear rate, \( \dot{\gamma} \), was calculated from the velocity gradient through the second invariant of the rate of strain tensor, \( \Pi_7 \), as follows:

\[
\dot{\gamma} = \sqrt{\frac{1}{2} \Pi_7} = \sqrt{\frac{1}{2} \left[ \sum_i \sum_j \dot{\gamma}_{ij} \dot{\gamma}_{ij} \right]}
\]

After the local viscosity was determined using the Carreau model, equation 6, the local shear stress, \( \tau (-\eta \dot{\gamma}) \), was calculated. Blood with a constant density of 1.05 g/cm\(^3\) and infinite shear-rate kinematic viscosity, \( \nu_\infty = (\eta_\infty / \rho) \), were used in the calculations. The pulse rate was 75 bpm. Hence, the dimensionless frequency parameter, \( \alpha = 0.5d(\omega \nu)^{0.5} \), for the artery was found to be 4.87, where \( d \) is the diameter, \( \omega \) is the circular frequency, and \( \nu \) is the kinetic velocity.

**RESULTS**

We included spatial distribution of velocity, the temporal distribution of shear rate, non-Newtonian viscosity, shear stress, and pressure drop during the systolic part of the pulse cycle. The flow calculations are presented for the terminal aneurysm with and without the balloon. The diastolic part of the pulse is not reported here because the changes in flow parameters were insignificant. By contrast, during the systolic part of the pulse (Fig. 3), sharp changes in instantaneous velocity created significant variations of calculated flow parameters.

**Velocity Profile**

At time step \( t = 0.105 \) sec, the spatial distributions of the axial velocity profiles, \( u_x \), from the neck to the interior core of the terminal aneurysm are shown in Figure 4. The magnitude of velocity inside the core of the aneurysm was less than that in the main or daughter branch arterial velocities. The peak velocity in the main lumen was 44.6 cm/sec, whereas it was only 3 cm/sec near the right neck of the aneurysm (locations IIIR in Fig. 4, top). The nature of the axial velocity profiles, \( u_x \), indicated that the flow was positive at the core region, whereas it was negative at the wall region of the aneurysm. This finding indicates that for a terminal aneurysm, the flow enters through the core and leaves it along the aneurysm wall.

However, the axial flow distribution was asymmetric in nature because it skewed more toward the right wall of the aneurysm, with the peak velocity and sharper velocity gradient were near the right wall region (locations IIIR and IIR in Fig. 4) rather than the left wall. The asymmetric distribution of the velocity was attributable to the geometric orientation (i.e., the angle between the
Aneurysm and the main and daughter arteries. It is more common to find asymmetric terminal aneurysms than symmetric ones.

Shear Rate

The top left panel of Figure 5 shows the magnitude of an instantaneous shear rate at critical right wall locations (left side of Fig. 2) of the aneurysm prior to balloon treatment, and the top left panel of Figure 5 shows the shear rates at the right wall for the aneurysm after insertion of the balloon (right side of Fig. 2).

Plot 1R in the top left panel of Figure 5 shows the normal instantaneous shear rate at the right wall location IR (left side of Fig. 2) during the systolic part of the pulse cycle. The maximum normal shear rate of 1,020 sec\(^{-1}\) occurred at \(t = 0.16\) sec. Plot 2R in the top left panel of Figure 5 shows the shear rate at the right neck location IIR (left side of Fig. 2) of the aneurysm. The maximum shear rate at the right neck of the aneurysm increased to 5,560 sec\(^{-1}\) at \(t = 0.17\) sec, which is more than five times the maximum value at location IR (arrow 1 in the top left panel of Fig. 5). Plot 3R shows the shear rate at location IIIR (left side of Fig. 2), where the maximum shear rate is 1,230 sec\(^{-1}\) at \(t = 0.16\) sec. The instantaneous shear rate at this location reaches a value of the normal shear rate as observed for plot 1R. As indicated by arrows 1 and 2, a phase shift is observed in which the time for maximum shear rate is different between plots 1R and 2R. In the left side of Figure 2, the shear rate at location IVR is insignificant because a sharp reduction in flow occurred from location 1R to IVR in the aneurysm sac. From the neck to the inside core of the aneurysm, the velocity gradient at the inside wall reduced and a stagnant flow region developed.

Because of the identical geometric location, plot 1R-b in the top right panel of Figure 5 shows a similar temporal variation of normal wall shear rate as observed in plot 1R in the top left panel of Figure 5. Plot 2R-b in the top right panel of Figure 5 represents the shear rate at the right neck of the aneurysm. The maximum shear rate at the right neck region with the inserted balloon is 2,730 sec\(^{-1}\) at \(t = 0.16\) sec (arrow 1-b in the top right panel of Fig. 5), which is approximately half the maximum shear rate observed for the aneurysm without the balloon. It is evident that the maximum shear rate at the neck, even with the balloon, is approximately 2.5 times the maximum normal shear rate obtained in locations IR and IR-b in the right side of Figure 2. The wall shear rate of the aneurysm close to the junction of the balloon wall was significantly less than the normal value, as shown in plot 3R-b in the top right panel of Figure 5 (arrow 2-b).

Plot 1L in the bottom left panel of Figure 5 and plot 1L-b in the bottom right panel of Figure 5 show the

![Figure 5](image-url)
normal instantaneous shear rate at the left wall locations IL and IL-b (Fig. 2). In general, we observed that the left wall had a lower level of shear rate than the normal value. The left wall of the aneurysm with and without the balloon showed a reduction in the magnitude of shear rate (arrows 3 and 3-b in the bottom left and right panels of Fig. 5, respectively). Compared with the maximum value of the shear rate of 988 sec\(^{-1}\) at \(t = 0.12\) sec (plot 1L in the bottom left panel of Fig. 5 and plot 1L-b in the bottom right panel of Fig. 5), the maximum shear rates for plot 3L in the bottom left panel of Figure 5 and 3L-b in the bottom right panel of Figure 5 were reduced to values of 429 sec\(^{-1}\) at \(t = 0.23\) sec and 18 sec\(^{-1}\) at \(t = 0.10\) sec, respectively. Clearly, a reduction in shear rate was observed at the left aneurysm wall with the balloon. Away from the neck, the inside wall of the aneurysm sac showed a further reduction of shear rate. In addition, a phase shift was observed between locations 1L and 3L in the bottom left panel of Figure 5 and between 1L-b and 3L-b in the bottom right panel of Figure 5.

Non-Newtonian Viscosity of Blood

An instantaneous value of non-Newtonian blood viscosity was calculated at the right wall (the top left panel of Fig. 6 for the aneurysm without the balloon and the top right panel of Fig. 6 for the aneurysm with the balloon) and at the left wall (bottom left panel of Fig. 6 for the aneurysm without the balloon and the bottom right panel of Fig. 6 for the aneurysm with the balloon). The non-Newtonian blood viscosity was inversely dependent on the shear rate (i.e., the blood viscosity was less at high shear rates, whereas it was high at low shear rates).

For plots 1R to 3R in the top left panel of Figure 6 and plots 1R-b to 3R-b in the top right panel of Figure 6, the shear rate is generally high during the systolic part of the pulse cycle, especially at the neck of the aneurysm where the blood shows infinite shear rate viscosity (i.e., 0.0345 poise). A reduction in the non-Newtonian viscosity of blood was observed at peak systolic location because at this location, the shear rate increased because of a sharp increase in the velocity gradient (arrows 4 and 4-b in the top left and right panels of Fig. 6). The non-Newtonian viscosity was progressively higher from the neck to the inside core of the aneurysm because the shear rate became significantly lower (top left and right panels of Fig. 5). When the balloon was inserted in the aneurysm, the increase in blood viscosity was significant because of the decrease in the shear rate (arrows 4 and 4-b in the top left and right panels of Fig. 6).

The bottom left and right panels of Figure 6 represent the non-Newtonian viscosity at the left wall of the terminal aneurysm. In Figure 6, the variation of non-Newtonian blood viscosity is depicted by arrow 5 in the

![FIGURE 6. Instantaneous non-Newtonian blood viscosity (Visc.) at the right and left walls for the aneurysm without and with the balloon.](image-url)
bottom left panel and arrow 5-b in the bottom right panel. Because of the sharper reduction of shear rate within a short distance, the increase of non-Newtonian viscosity was much faster for the aneurysm with the balloon than without it. Compared with the left wall, the reduction in non-Newtonian viscosity was higher at the right wall.

**Wall Shear Stress**

Based on the shear rate and non-Newtonian viscosity calculations in the previous sections, the shear stress is calculated. In this section the magnitude of maximum value of normal wall shear stress is compared with the shear stress at the aneurysm neck with and without balloon.

Table 1 shows that the normal value of the maximum shear stress calculated at the main inlet arterial wall was 37.7 dynes/cm². At the right neck (IIIR in the left side of Fig. 2) of the aneurysm, the maximum shear stress increased to a value of 194.6 dynes/cm². However, when the balloon was inserted in the aneurysm, the maximum shear stress at the right neck (IIIR-b in the right side of Fig. 2) decreased to 98.3 dynes/cm². We ignored the shear stress calculations at the left neck because the magnitude of the shear rate at this location was significantly lower than that of the right neck.

**Pressure**

The top and bottom left panels of Figure 7 show the calculated pressure drop for the aneurysm without the balloon, whereas the top and bottom right panels of Figure 7 show the pressure drop with the balloon. Because the aneurysm acted as a dead end, the flow was significantly reduced, leading to a high-pressure region inside the aneurysm sac. The pressure drop, when compared with the main inlet artery, increased from the neck region to the inside core of the aneurysm (arrow 6 in the top left panel of Fig. 7). The right neck of the aneurysm without the balloon showed less of a pressure drop, approximately 1,107 dynes/cm² at t = 0.18 sec, compared with the core region of the aneurysm, where the pressure drop was 1,959 dynes/cm². When the balloon was inserted, a reduction of maxi-

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**TABLE 1: Normal Peak Wall Shear Stress Versus Peak Wall Shear Strain at the Right Neck of the Aneurysm With and Without the Balloon**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Normal</th>
<th>Without Balloon</th>
<th>With Balloon</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shear rate (1/sec)</td>
<td>1,020</td>
<td>5,560</td>
<td>2,730</td>
</tr>
<tr>
<td>Viscosity (poise)</td>
<td>0.037</td>
<td>0.035</td>
<td>0.036</td>
</tr>
<tr>
<td>Shear stress (dynes/cm²)</td>
<td>37.7</td>
<td>194.6</td>
<td>98.3</td>
</tr>
</tbody>
</table>

**FIGURE 7.** Instantaneous pressure drops at the right and left walls for the aneurysm without and with the balloon.
mum pressure drop, from 1,959 dynes/cm² to 1,665 dynes/cm², was observed for the right wall of the aneurysm (arrows 7 and 7-b in the top left and right panels of Fig. 7, respectively). A similar phenomenon was observed at the left wall (bottom left and right panels of Fig. 7) of the aneurysms. It is evident that the aneurysm experienced high pressure and that the balloon helped to reduce the maximum pressure inside the aneurysm by approximately 15%.

**DISCUSSION**

Gobin et al. [11] demonstrated that partial coil embolization of a lateral aneurysm did not prevent regrowth or rebleeding of the aneurysm. However, the hemodynamics of bifurcation and terminal aneurysms differ. Unlike lateral aneurysms, the circulation of terminal aneurysms is rapid, without vortex formations. The dynamics of the inflow–outflow zones depend on the angle between the aneurysm and its inlet and outlet branches. A qualitative analysis was performed by Strother et al. [12] using angiography and Doppler studies on terminal aneurysms produced in dogs. The calculated flow field in the terminal aneurysm for the current study was similar to the qualitative observations of Strother et al. [12], who reported that hemodynamic changes occurred mainly at the neck region without the presence of an intraaneurysmal vortex. Our results also indicate that hydrodynamic flow parameters at the neck of the aneurysm play a significant role in recurrence after balloon occlusion.

Physiologically, terminal aneurysms with an asymmetric flow distribution occur more often than symmetric ones [9]. The asymmetry of the two daughter branches with respect to the flow axis of the main artery creates an unequal distribution of hydraulic loss factors [18]. In our study, the hydraulic loss factor associated with the left branch of the aneurysm was about 30% higher than the right branch, which then resulted in a greater flow to the right branch than to the left branch. This caused a higher velocity gradient and hence elevated shear stress at the right neck compared with the left neck.

Because of the geometric shape of the terminal aneurysm, we could not obtain complete endovascular occlusion after balloon insertion. A small residual area at the right neck was left uncovered (Fig. 1B). Compared with the normal maximum value of shear stress, a fivefold increase occurred at the right neck area of the aneurysm before balloon occlusion. By contrast, when a balloon was inserted, the maximum shear stress at the right neck area decreased to half the value of the maximum shear stress calculated before insertion of the balloon. Although the maximum shear stress at the right neck of the aneurysm with the balloon was significantly reduced, it was still 2.5 times more than the normal maximum shear stress calculated at the main inlet arterial wall. It is evident that when inserted inside the aneurysm, the balloon restricted the flow in the aneurysm sac, retarding overall flow velocity, which resulted in a reduced level of shear rate, shear stress, and pressure in the neck.

Because the aneurysm recurred 2 years after the balloon occlusion, the results of our study indicate that the high wall shear stress in association with increased pressure are at least partially responsible for the development and regrowth of a daughter aneurysm. Other factors, however, may be involved in this process. Physiochemical changes such as increased intimal strain [19] and changes in the endothelial cell also may play a significant role. An elevated level of shear stress, for instance, may result in physiochemical changes in the endothelial cells that induce vascular remodeling (e.g., vascular dilation associated with sustained high blood flow or the cell loss and matrix proteolysis that result in aneurysm formation) [20].

One advantage of our computational simulation is the simultaneous solving of time-dependent, nonlinear momentum and mass equations for any irregular geometry such as the present case. Another advantage is the shear-rate–dependent, non-Newtonian viscosity of blood and pulsatile inflow boundary conditions. However, a limitation of our study was the use of a rigid wall model. To improve the accuracy of results in the future, viscoelasticity of the vessel wall needs to be included in the calculations. Moreover, to determine the complete flow field in the aneurysm, numeric studies using three-dimensional models will be more appropriate.

**REFERENCES**

HEMODYNAMIC CHANGES IN TERMINAL ANEURYSM


Announcement

The Japan Radiological Society (JRS) has organized several scientific meetings or assemblies to be held in the coming years:

• The 55th General Assembly of the JRS will be held April 2–4, 1996, at the Pacifico Yokohama Conference Center in Yokohama, Japan. The official languages are English and Japanese. For more information, contact Tsutomu Takashima, Department of Radiology, Kanazawa University Medical School, 13-1 Takara-machi, Kanazawa 920, Japan; 0762-62-8151, fax 0762-34-4256.

• The Autumn Assembly of the JRS will be held October 7–9, 1996, at the Sapporo Medical College in Hokkaido, Japan. For more information, contact Kazuo Morita, Department of Radiology, Sapporo Medical College, S1 W17, Chu-o-ku, Sapporo 060, Japan; 011-611-2111, fax 011-613-9920.

• The 56th General Assembly of the JRS will be held April 4–6, 1997, at the Pacifico Yokohama Conference Center in Yokohama, Japan. The official languages are English and Japanese. For more information, contact Koichi Yamaguchi, Department of Radiology, Yamagata University Medical School, 2-2-2 Iida-nishi, Yamagata 990-23, Japan; 0236-33-1122, fax 0236-28-5389.

• The 57th General Assembly of the JRS will be held April 9–11, 1998, at Port Island in Kobe, Japan. The assembly will be held in conjunction with the 1998 Asian and Oceanian Congress of Radiology. The official languages are English and Japanese. For more information, contact Michio Kono, Department of Radiology, Kobe University Medical School, 7-5 Kusunoki-cho, Kobe 650, Japan; 078-341-7451, fax 078-371-7143.

• The Eighth Asian and Oceanian Congress of Radiology will be held April 5–8, 1998, at Port Island in Kobe, Japan. The meeting is being organized by the JRS in conjunction with its 1998 meeting. For more information, contact Hitoshi Katayama, Department of Radiology, Juntendo University Medical School, 3-1-3 Hongo, Bunkyo-ku, Japan; 03-3813-3111, fax 03-3812-6035/03-3816-0958.