**Myocardial Infarction**

**Diagnosis of right ventricular involvement in chronic inferior myocardial infarction by means of body surface QRS changes**

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**ABSTRACT**  ST segment elevation in right precordial leads is thought to be a good predictor of right ventricular involvement in patients with acute inferior myocardial infarction. This view, however, is rapidly disappearing. Therefore, using QRS changes in body surface potential maps in the chronic phase, we have attempted to differentiate patients with or without right ventricular involvement. Thirty patients with chronic inferior myocardial infarction (2 or more months after onset) were studied, in whom 87 unipolar ECGs and right ventriculograms were recorded. The patients were then divided into three groups depending on the locations of their abnormal QRS potentials (−2SD area) exceeding the normal range (mean − 2SD). In group A, the −2SD area was located predominantly on the right inferior chest, in group B on the left inferior chest, and in group N on both the right and left inferior chests equally. The results showed that group A had a lower right ventricular ejection fraction (RVEF) compared with group B (A, 40 ± 7%; B, 53 ± 10%; p < .001), while there was no difference in left ventricular ejection fraction between the two groups (49 ± 11% and 49 ± 11%, respectively). Moreover, right ventricular asynergy occurred in 14 of the 18 patients (78%) of group A but in only one of the 10 patients (10%) of group B. Group N was presumed to be intermediate between groups A and B. We also found that patients without Q waves in lead I of the 12-lead electrocardiogram (ECG) or patients with the 20 msec QRS vector in the left upper position on the vectorcardiogram (VCG) both had lower RVEF and tended to have a higher incidence of right ventricular asynergy. For recognition of right ventricular asynergy, each of the studies (mapping, ECG, and VCG) had high sensitivities (81% to 88%). For determining specificity, the mapping criteria were superior to both the VCG criteria and the ECG criteria. This QRS change was characterized as loss of electrical activity in right lower chest.

Information of this sort will be beneficial in managing such patients with myocardial infarction. *Circulation* 77, No. 6, 1283–1290, 1988.

**ST SEGMENT** elevation in right precordial lead is thought to be a good indicator of right ventricular involvement in patients with acute inferior myocardial infarction. However, because of the short duration of ST segment elevation, diagnostic accuracy was low after completion of the study period. Moreover, ST segment elevation does not identify necrotic tissue but myocardial ischemia. On the other hand, the loss of electrical activity in the right ventricle should alter the QRS potential. Some experimental results have shown the occurrence of QRS changes in the right precordial chest caused by right ventricular infarction. In human beings, however, the effect of right ventricular infarction on the QRS complex in the chronic stage has not been examined systematically. Therefore, in this study we have paid close attention to the body surface QRS changes during the chronic phase. We have attempted to identify patients with right ventricular involvement in inferior myocardial infarction.

Body surface mapping yields more information than the standard electrocardiographic technique in myocardial infarction. We also used the departure mapping technique to assess changes in the QRS potential, by which we were able to detect potential distribution out of the normal range. The purpose of this study was to differentiate inferior myocardial infarction with or without right ventricular involvement from the spatial distribution of abnormal QRS potentials. Then we were able to compare these QRS changes with the standard 12-lead electrocardiogram (ECG) and the vectorcardiogram (VCG).

**Materials and methods**

**Subjects.** Of the 930 consecutive patients who have under-
gone cardiac catheterization and radionuclide ventriculography at Yamagata University Hospital from August 1, 1980, to March 31, 1987, the following patients were selected.

**Inferior infarction group.** Thirty patients, ages 35 to 70 years (mean 56.4), who all satisfied the following criteria were selected as the group with inferior infarction: (1) a clinical diagnosis of myocardial infarction established by typical chest pain and serum enzyme changes, (2) no other heart disease such as congenital heart disease, myocardial disease, valvular heart disease, or hypertensive heart disease, (3) no conduction disturbances such as bundle branch block or Wolff-Parkinson-White syndrome, (4) abnormal Q waves (≥0.04 sec) in leads II and aVF or III and aVF but no abnormal Q waves in V₁ to V₄, (5) wall motion abnormalities in at least the inferior wall of the left ventricle (segments 4 and/or 5 according to the reporting system of the American Heart Association), and (6) significant stenosis of at least 75% or more in the right coronary artery and/or left circumflex artery.

**Anterior infarction group.** To examine the specificity of our ECG criteria, 32 patients, ages 34 to 71 years (mean 54.8), who all satisfied the following criteria were selected as the group with anterior myocardial infarction: criteria 1 through 3 as described above, (4) abnormal Q waves (≥0.04 sec) in leads V₁ and V₂ but no abnormal Q waves in II or aVF, (5) wall motion abnormalities in at least the anterior wall (segments 2 and/or 3) of the left ventricle, and (6) significant stenosis of at least 75% or more in the left anterior descending artery.

The time from the onset of myocardial infarction to cardiac catheterization ranged from 2 to 11 months (mean 5.1). Radionuclide ventriculography, body surface mapping, and ECG and VCG recording were performed within a week before or after cardiac catheterization.

Forty normal male volunteers, ages 22 to 51 years (mean 32), were examined, and body surface mapping was performed to evaluate normal values for ECG measurements. None of the volunteers had any previous history of cardiac disorders or systemic arterial hypertension, and all showed normal physical and 12-lead ECG findings. All subjects gave their consent before the study commenced.

**Body surface mapping**

**Map recording.** Body surface mapping was performed by use of a body surface potential mapping system, the HPM-5100 unit (Chunichi Denshi Co.). Eighty-seven body surface leads were arranged on the patient’s body in a latticelike pattern (13 × 7 matrix) except for the lead points in the midaxillary line. The body surface leads covered the patient’s entire thoracic surface (59 leads located on the anterior chest and 28 leads on the back). ECGs from these 87 unipolar leads, with Wilson’s central terminal used as reference, standard 12-lead ECGs, and Frank X, Y, and Z VCGs were sampled simultaneously. The stored signals of each of the ECGs were then displayed on a graphic terminal (Tektronix 4006-1). If noise was detected in any of the signals, data sampling was repeated. The flat portion of the PQ segment was chosen as the baseline. After baseline adjustment, data were recorded on a magnetic cassette tape in a digital format. This system had a resolution of 0.01 mV in the dynamic range ± 5 mV, with a sampling rate of 1000 samples/sec/channel. The data sampling was done at the resting expiratory level and in the supine position.

**Data analysis.** The mapping data were processed off-line on a minicomputer (DEC VAX 11/750) by means of a program developed by our institution. For mapping data analysis, the QRS onset and offset levels were determined from the superimposed Frank X, Y, and Z leads and the spatial magnitude. QRS potentials at 10, 20, 30, 40, 50, and 60 msec from the QRS onset were measured.

**Departure maps.** The mean (M) and standard deviation (SD) of the normal QRS potentials at each lead point were determined from 40 normal volunteers. To estimate the deviation of patient data from the normal value, the departure index (DI) at each lead was calculated as follows: DI = (X - M)/SD, where X represents the QRS potential at the corresponding lead of each of the patients. The body surface distribution of DI values was expressed by use of a map we called the “departure map.” Since we were interested in decreases of the QRS potential, the areas where the DIs were less than −2 on the departure map were designated as “−2SD areas.” On the maps, the rectangular areas represented torso surfaces, with the left half of the map reflecting the anterior chest and right half of the map reflecting the back. Therefore, both the right and left edges of the maps represented the right midaxillary line. Contour lines were drawn to connect points of equal DIs. The contour lines were separated by an interval of 1.

**Radionuclide ventriculography.** Radionuclide ventriculography with ⁹⁹ᵐTc-labeled human serum albumin was performed on all 62 patients. A 20 mCi bolus of technetium with saline solution was flushed into the antecubital vein.

Data were collected with an Ohio-Nuclear 410S scintillation camera interfaced to a DEC Gamma 11 processing system (PDP 11/34). With a low-energy, high-resolution slant-hole collimator, the first-pass studies were performed in a 30-degree right anterior oblique (RAO) projection soon after the technetium infusion, and the equilibrium studies were performed in a 60-degree left anterior oblique (LAO) projection 1 hr after the infusion. The left ventricular ejection fraction (LVEF) and right ventricular ejection fraction (RVEF) were calculated by a standard technique with the background-corrected time-activity curve from the left and right ventricular region of interest.

The 30-degree RAO projection was found to be suitable because it permitted good separation of the right ventricle, the right atrium, and pulmonary artery. To determine the ventricle perimeter, a 50% threshold was imposed on each image. Left and right ventricular wall motion was assessed qualitatively by three observers who had no prior knowledge of the ECG data. Wall motions were then evaluated separately in both anterolateral and posterior segments of the right ventricle, and the following segments of the left ventricle: (1) anterobasal, (2) anterolateral, (3) apical, (4) diaphragmatic, (5) posterobasal, (6) septal, and (7) posterolateral. The akinetik and the dyskinetic segments were defined as “asymmetry” (wall motion abnormality). Segments 1 through 5 of the left ventricle and both segments of the right ventricle were evaluated by a 30-degree RAO first-pass study, and segments 6 and 7 of the left ventricle were evaluated by a 60-degree LAO equilibrium study.

**Coronary angiography.** Selective coronary angiographic studies were performed in multiple projections by use of the Judkins technique. Coronary arterial narrowing of 75% or more in the luminal diameter was considered to be significant.

**Statistical analysis.** The quantitative data were expressed as the mean ± SD. Statistical comparisons were performed by analysis of variance and by an unpaired t test for the difference of the group mean; p < .05 was considered significant. The sensitivity and specificity of our criteria in detecting right ventricular asynergy were calculated by the standard formula.

**Results**

**Departure maps.** Thirty patients with inferior infarction were divided into three groups according to the site of the −2SD area on each patient’s departure map at a time of 20 or 30 msec from the QRS onset.

Group A was defined as the group in which peak negativity appeared on the right lateral chest; negative value contour lines were then drawn from the right
upper chest to the left lower chest. Group B was defined as the group in which peak negativity appeared on the left lateral chest; negative value contour lines were then drawn from the right lower chest to the left upper chest. Group N was defined as the group in which peak negativity occurred on the anterior lower chest; the contour lines were drawn nearly horizontal. These patients were thought to be midway between groups A and B. Figure 1 shows representative findings of each group. Of the 30 patients, 18 (60%) were classified as group A, 10 (33%) as group B, and only two (7%) as group N.

The map findings were then compared with the radionuclide ventriculographic findings for groups A and B. Group N was neglected because of its small population. Figure 2 shows the LVEFs and RVEFs of each group. Although there were no differences in LVEFs between groups A and B (A, 49 ± 11%; B, 49 ± 11%), remarkable differences in RVEFs were observed between the two groups. RVEFs in group A were significantly lower than those in group B (A, 40 ± 7%; B, 53 ± 10%; p < .001). Right ventricular asynergy was observed in 16 patients (53%). Figure 3 shows representative findings from patients with and without right ventricular asynergy. In group A, the incidence of right ventricular asynergy (14/18, 78%) was remarkably higher than that in group B (1/10, 10%; table 1). Using the spatial distribution of the abnormality in the early QRS potential, we could determine right ventricular involvement in these patients with chronic inferior myocardial infarction.

Segmental left and right ventricular wall motions of groups A, B, and N are listed in table 2. In the left ventricle, there were no significant differences in presence of asynergy between groups A and B in all segments. Right ventricular asynergy was observed in 16 patients. In group A patients with right ventricular asynergy, the asynergic site was located in the posterior segment only (9/14) or in both the posterior and anterolateral segments (5/14). One patient of group B had asynergy in both right ventricular sites, and one patient of group N showed right ventricular asynergy in the anterolateral segment.

All group A patients has stenosis of the right coronary artery. Dominance of the left circumflex artery was recognized in only one patient of group A. This patient had a 90% narrowing at the proximal portion of the left circumflex artery. Right ventricular involvement in this patient was assumed to be caused by the stenosis of the circumflex. Other major coronary arter-

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**FIGURE 1.** Departure maps at times of 10, 20, 30, 40, 50, and 60 msec from the QRS onset in representative patients. A, group A; B, group B; C, group N. The shaded area represents the -2SD area. Map classification was done by use of the 20 and 30 msec maps. Contour lines are separated by an interval of 1. See text for details.
ies, however, also showed stenosis (100% narrowing at the distal end of the left anterior descending artery and 90% narrowing at the distal portion of right coronary artery).

VCG and 12-lead ECG findings. From the mapping results, QRS changes suggesting right ventricular involvement were thought to be characterized by loss of electrical activity in the right inferior chest during the initial QRS complex. This change appeared as a leftward and upward shift of the early QRS vector in the VCG and as loss of the rightward force of the initial QRS complex in the 12-lead ECG. ECG and VCG findings were classified taking these effects into consideration (figure 4).

VCG findings. The VCG findings were divided into two groups according to the position of QRS vector 20 msec after QRS onset. Group VCG-A was defined as the group in which the 20 msec QRS vector was positioned in the left upper zone. Group VCG-B was defined as the group in which the 20 msec QRS vector was positioned in the right upper zone. Of the 30 subjects, 21 were in group VCG-A and nine were in group VCG-B.

RVEFs in group VCG-A were significantly lower than those in group VCG-B (VCG-A, 42 ± 8%; VCG-B, 54 ± 10%; p < .01). There were no differences in LVEFs between the two groups. Thirteen of the 21 patients (62%) of group VCG-A had right ventricular

FIGURE 2. LVEF and RVEF in each group classified by departure mapping. Although there were no differences in LVEF between groups A and B, remarkably lower RVEFs were observed in group A.

FIGURE 3. Wall motion image of the right ventricle in patients with and without right ventricular (RV) asynergy. In patients with right ventricular asynergy (left side), akinetic wall motion was observed in the posterior wall.
TABLE 1
Radionuclide ventriculographic results of the groups classified by body surface mapping

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>LVEF (%)</th>
<th>RVEF (%)</th>
<th>RV asynergy</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>18</td>
<td>49±11</td>
<td>40±7</td>
<td>14/18 (78%)</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
<td>49±11</td>
<td>53±10</td>
<td>1/10 (10%)</td>
</tr>
<tr>
<td>N</td>
<td>2</td>
<td>51</td>
<td>50</td>
<td>1/2</td>
</tr>
<tr>
<td>(A vs B)</td>
<td>NS</td>
<td></td>
<td>p&lt;.001</td>
<td>p&lt;.01</td>
</tr>
</tbody>
</table>

See text for description of groups.

asynergy, while only three of the nine patients (33%) of group VCG-B had right ventricular asynergy (table 3).

12-lead ECG findings. Patients were divided into the following two groups according to the existence of Q waves in lead I: QI(−) (20 patients) and QI(+) (10 patients).

RVEFs in the QI(−) group were significantly lower than those in the QI(+) group [QI(−), 41 ± 8%; QI(+), 53 ± 10%; p < .01]. There were no differences in LVEFs between the two groups. Thirteen of the 20 patients (65%) of the QI(−) group had right ventricular asynergy, while only three of 10 patients (30%) in the QI(+) group had right ventricular asynergy (table 3).

Sensitivity and specificity of the three studies: mapping, VCG, and 12-lead ECG. The sensitivity and specificity of these criteria for the recognition of right ventricular asynergy were investigated in two different populations: (1) patients with inferior infarction group and (2) patients with both inferior infarction and anterior infarction (table 4). In the mapping study, group N results were considered negative. The anterior myocardial infarction group, which was selected to be used in determining specificity, had a lower LVEF (41 ± 16%) than that of group A or group B of the inferior infarction group, but had an RVEF equal to that of group B (54 ± 10%). Moreover, no akinetic right ventricular sites were observed in the anterior infarction group. Therefore, the test sensitivities in both populations were equal.

Criteria for departure maps (group A), VCG (group VCG-A), and 12-lead ECG [group QI(−)] had relatively higher sensitivity (81% to 88%) for recognition of right ventricular asynergy. Thirteen of the 16 (81%) patients with right ventricular asynergy satisfied the criteria specified by all three test criteria. Fourteen of the 16 were detected by the mapping study; the sensitivity of mapping study criteria was therefore higher (88%).

On the other hand, the specificities of the criteria established by the three methods differed considerably. In the population with inferior infarction, the mapping study of group A had the highest specificity (71%) of the three techniques. Both VCG and 12-lead ECG studies showed relatively lower specificities (43% and 50%, respectively). In the population with inferior and anterior infarction, mapping studies exhibited the highest specificity (91%) of the three techniques. The VCG study showed the next highest specificity (67%), and the 12-lead ECG study showed a relatively lower specificity (52%). The diagnostic performance of the departure mapping method proved to be superior to the others.

Discussion

Myocardial infarction involving the right ventricle usually occurs in conjunction with inferior infarction. These patients, suffering from right ventricular dysfunction because of low cardiac output, may require fluid administration rather than fluid restriction.10 After coronary artery bypass surgery, right ventricular infarction often causes a continuing low cardiac output syndrome that is difficult to treat. The diagnosis of right ventricular infarction is important in order to be able to manage patients effectively.

Electrocardiographic diagnosis in these patients was done by assessing ST elevation in the right precordial leads.1-8 ST elevation in the right precordial leads, however, is not specific enough for patients with right ventricular infarction. Similar changes may also occur with pericarditis or pulmonary embolism. Moreover,

TABLE 2
Prevalence of asynergy at each segment in the groups classified by body surface mapping

<table>
<thead>
<tr>
<th>Left ventricle</th>
<th>Right ventricle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group</td>
<td>Ant-B</td>
</tr>
<tr>
<td>-------</td>
<td>--------</td>
</tr>
<tr>
<td>A</td>
<td>18</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
</tr>
<tr>
<td>N</td>
<td>2</td>
</tr>
</tbody>
</table>

Ant-B = antero-basal; Ant-L = antero-lateral; Diaph = diaphragmatic; Post-B = postero-basal; Sept = septal; Post-L = posterolateral; Post = posterior; A-L/Post = antero-lateral or posterior.

^p<.01 (A vs B).
since the ST elevation is transient, it may be missed if the ECG is not recorded during the acute phase. Furthermore, ST elevation indicates myocardial ischemia, not necrotic tissue. Therefore, close attention was paid to the QRS complex, and we tried to determine whether or not the QRS changes on the body surface could identify patients with right ventricular involvement in inferior myocardial infarction in the chronic phase.

**Departure maps.** By use of departure maps, body surface areas in which QRS values are out of the normal range (mean - 2SD) can be determined. The departure index [DI = (X - mean of normal)/SD of normal] used in this study is compatible with Mahalanobis' generalized distance. The DI indicates the extent of deviation from normal control at the lead point. Using the DIs, we can detect the extent of abnormal depolarization on the body surface, even where the potential amplitude is small. The location of the minimum DI indicates the area of the most prominent change.

Map classifications were determined according to the sites of the - 2SD area on the departure maps early in the QRS complex. Patients with the - 2SD area on the right inferior chest (group A) had remarkably lower RVEFs and a higher incidence of right ventricular asynergy than patients with the - 2SD area on the left inferior chest (group B). From these results, we were able to differentiate patients with or without right ventricular involvement in inferior myocardial infarction in the chronic stage. Group N could not be investigated for right ventricular involvement because of its small number of patients. Group N was believed to be midway between groups A and B based on its map pattern. To characterize group N with certainty, however, further examination of a larger group would be required.

**TABLE 3**

<p>| Radionuclide ventriculographic results of the groups classified by VCG and ECG |</p>
<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>LVEF (%)</th>
<th>RVEF (%)</th>
<th>RV asynergy</th>
</tr>
</thead>
<tbody>
<tr>
<td>VCG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>21</td>
<td>50 ± 11</td>
<td>42 ± 8</td>
<td>13/21 (62%)</td>
</tr>
<tr>
<td>B</td>
<td>9</td>
<td>49 ± 11</td>
<td>54 ± 10</td>
<td>3/9 (33%)</td>
</tr>
<tr>
<td>(A vs B)</td>
<td>NS</td>
<td></td>
<td></td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>QI(-)</td>
<td>20</td>
<td>50 ± 11</td>
<td>41 ± 8</td>
<td>13/20 (65%)</td>
</tr>
<tr>
<td>QI(+)</td>
<td>10</td>
<td>49 ± 11</td>
<td>53 ± 10</td>
<td>3/10 (30%)</td>
</tr>
<tr>
<td>[QI(-) vs QI(+)]</td>
<td>NS</td>
<td></td>
<td></td>
<td>p &lt; .01</td>
</tr>
</tbody>
</table>

See text for description of groups.

**TABLE 4**

<p>| Sensitivity and specificity of the three techniques in recognizing right ventricular asynergy |</p>
<table>
<thead>
<tr>
<th>Inferior MI</th>
<th>Anetor + inferior MI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sensitivity</strong></td>
<td><strong>Specificity</strong></td>
</tr>
<tr>
<td>Mapping</td>
<td>14/16 (88%)</td>
</tr>
<tr>
<td>VCG</td>
<td>13/16 (81%)</td>
</tr>
<tr>
<td>12-lead ECG</td>
<td>13/16 (81%)</td>
</tr>
</tbody>
</table>

MI = myocardial infarction; sensitivity = true positives correctly diagnosed/total true positives; specificity = true negatives correctly diagnosed/total true negatives.
The asynergic right ventricular site was located mainly on either the posterior segment only or on both the anterolateral and the posterior segments. This correlated with the right ventricular distribution of the right coronary artery. Six of seven patients with inclusion of the anterolateral segment had stenosis of both the right and left coronary arteries; the seventh had a dominant right coronary artery. It was believed that the disturbance of the dual artery supply to the right ventricle might have caused the anterolateral asynergy.

In this study, patients with right ventricular involvement in inferior myocardial infarction were successfully identified by the 20 or 30 msec potential departure maps. Infarction of other segments could influence the potential departure map distribution during these periods. Table 2 shows the extent of asynergy at each segment of the right and left ventricles. In both groups A and B, many patients had asynergy in the diaphragmatic, the posterobasal, and the septal segments. Septal asynergy was mainly located on the lower septum. Complicated anteroseptal infarction showing abnormal Q waves in leads V₁ to V₄ was not included in this population because of the selection criteria. With the exception of right ventricular asynergy, there were no significant differences in asynergy between groups A and B. Infarction of other segments did not influence the group separation. Loss of electrical activity in the right ventricle predominantly influenced group separation.

In the canine preparation, since the right coronary artery supplies almost exclusively the free wall of the right ventricle, its occlusion would result in an isolated right ventricular infarction. Sugiyama et al.⁹ recorded QRS changes in dogs for a week after experimentally inducing right ventricular myocardial infarction. They observed that the negative potentials occupy a large part of the right anterior chest surface. Chou et al.¹⁰ also observed both abnormal Q waves and regression of the R wave in the right precordial leads in 13 of 14 dogs. These studies indicated the usefulness of QRS changes in recognizing right ventricular involvement.

Clinically, some investigators have reported that QRS changes occur in the right ventricular infarction with ST elevation in the acute phase.³ ⁸ Coma-Canella et al.⁷ reported that Q waves in leads V₁ to V₃ were sometimes observed in patients with right ventricular infarction, but the diagnostic accuracy of these findings was low. Montague et al.⁸ demonstrated QRS changes in right anterior leads using time-integral analysis based on body surface mapping. On the group mean map, groups with right and left ventricular infarction tended to have a greater area of negative Q-
believed that the inclusion of combined infarction would have complicated the interpretation of the results for both the effect of primary right ventricular injury and the secondary influence of remote left ventricular infarction to right ventricular wall motion.

By evaluation of QRS changes in the chronic phase, patients with right ventricular involvement in inferior infarction can be identified noninvasively. These QRS changes are characterized by a loss of electrical activity in the right lower chest and can be recognized by body surface mapping. Moreover, right ventricular infarction can be diagnosed with these QRS changes in the conventional 12-lead ECG and the VCG. Information of this sort will be helpful in managing patients with myocardial infarction.

References


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