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## Results of hepatic resection for hepatocellular carcinoma invading major portal and/or hepatic veins

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Hepatocellular carcinoma (HCC) is one of the most common malignancies in Japan and worldwide [1]. Improvements in diagnostic imaging and clinical screening for HCC in high-risk patient populations have made it possible to diagnose small, asymptomatic HCC [2]. These improvements have led to better survival for patients with HCC who are treated with surgical and nonsurgical therapies. Many modalities have been applied to HCC. Local ablation therapies, such as percutaneous ethanol injection therapy (PEIT) and radiofrequency ablation therapy (RFA), are useful for patients with small HCCs [3,4]. Liver transplantation is another effective modality for patients with small HCCs and severe liver dysfunction [5]. Transcatheter arterial chemoembolization (TACE) is applied not only to patients with unresectable HCC but also to those with multiple intrahepatic tumors [6,7]. These modalities, however, are not indicated for patients with advanced HCC because of the presence of vascular invasion or large tumor size.

Surgical resection can be offered only to a minority of patients with HCC because most patients have chronic hepatitis or liver cirrhosis. Several improvements in preoperative evaluations [2,8], operative procedures [9–12], and perioperative management [13,14], however, have decreased the morbidity and mortality of hepatic resection in patients with underlying chronic liver disease. From these advances, the authors have applied hepatic

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resection aimed at complete tumor resection or cytoreductive treatment to patients with HCC with major vascular involvement [15].

This article retrospectively analyzes surgical results in a series of patients with HCC involving the major portal and/or hepatic veins to evaluate the feasibility of hepatic resection as a local treatment.

### Patients and methods

The clinical records of all patients who underwent hepatic resection for HCC at the Department of Gastroenterological Surgery, Kyoto University Hospital, were reviewed. Between January 1985 and December 2001, 845 patients with HCC underwent hepatic resection, excluding patients who underwent repeated hepatic resections or who had extrahepatic metastasis. The male-to-female ratio was 3.7 to 1, and patients' mean age was 61.2 years (range, 18–85 years). Preoperative liver dysfunction was evaluated using the clinical stage classification of the Liver Cancer Study Group of Japan [16]. The determinations were based on ascites, serum total bilirubin level, serum albumin level, indocyanine green retention rate at 15 minutes (ICGR<sub>15</sub>), and prothrombin time (%). Microscopic or macroscopic portal vein invasion was observed in 323 patients, and microscopic or macroscopic hepatic vein invasion was seen in 98 patients. One hundred and twelve patients with HCC invading the first branch of the portal vein and/or hepatic vein trunk were entered into this study.

Patients were observed in the outpatient clinic of Kyoto University Hospital or at the referring hospitals. Follow-up studies were performed with serum  $\alpha$ -fetoprotein or des- $\gamma$ -carboxyprothrombin once a month, and ultrasonography or CT once every 3 months. Patients with recurrent tumors underwent surgical resection, PEIT, RFA, TACE, and/or regional chemotherapy. Systemic chemotherapy was not given in the absence of extrahepatic recurrent tumors.

All deaths occurring within 30 days after hepatic resection were counted as operative deaths. Survival rates were determined by the Kaplan-Meier method, and the survival differences between groups were compared by the log-rank test. A *P* value less than 0.05 was considered to be significant.

### Results

The preoperative clinical features of the 112 patients included in the study are presented in Table 1. Subjects included 90 men and 22 women, with a mean age of 58 years (range, 31–85 years). The patients were observed until December 2001, for 0 to 131 months. Of 40 patients who underwent hepatic resection before 1989, 10 were positive for hepatitis B virus (HBV) surface antigen. Among 72 patients who underwent hepatic resection after 1990, 19 were positive only for HBV surface antigen, 33 were positive only for hepatitis C virus antibody, and 1 was positive for both.

Table 1  
Patient profiles and tumor characteristics of HCC with major vascular involvement

Variables	Characteristics
Gender	90 men, 22 women
Age range (mean $\pm$ SD)	31–85 y (58.1 $\pm$ 10.4 y)
Clinical stage I	70
Clinical stage II	41
Clinical stage III	1
ICGR <sub>15</sub> <15%	57
15% $\leq$ ICGR <sub>15</sub> <25%	30
25% $\leq$ ICGR <sub>15</sub> <40%	21
40% $\leq$ ICGR <sub>15</sub>	4
Solitary (IM0)	35
Multiple (IM1)	7
Multiple (IM2)	40
Multiple (IM3)	30
Maximal tumor size $\leq$ 5 cm	27
Maximal tumor size >5 cm	85
TT involvement <sup>a</sup>	
Vp3	46
Vp4	45
Vv2	23
Vv3	18

*Abbreviations:* HCC, hepatocellular carcinoma; ICGR<sub>15</sub>, indocyanine green retention rate at 15 min; TT, tumor thrombi.

<sup>a</sup> Total is 132 because 20 patients had TT involvement of both hepatic and portal veins.

IM0, no intrahepatic metastasis; IM1, intrahepatic metastasis to the sector in which the main tumor is located; IM2, intrahepatic metastases to 2 sectors; IM3, intrahepatic metastases to 3 or 4 sectors; Vp3, TT in the first branch of the portal vein; Vp4, TT in the portal trunk or extending to a branch on the opposite side; Vv2, TT in the hepatic vein trunk or the short hepatic vein; Vv3, TT in the inferior vena cava.

In the current study, 71 of the 112 patients with vascular invasion had tumor thrombi (TT) only in the major portal vein, 21 only in the major hepatic vein, and 20 in both the major portal and hepatic veins. Of 91 patients with TT in the major portal vein, 46 patients had TT in the first branch, and 45 patients had TT in the portal trunk. Among 23 of 41 patients with hepatic vein TT, the TT were found in the hepatic vein trunk, and in 18 patients the tumor had invaded the inferior vena cava (IVC).

The operative procedures are presented in Table 2, using Couinaud's anatomic nomenclature [17]. TT in the first branch of the portal vein or in the hepatic vein trunk could be removed using anatomic hepatic resection. TT in the portal trunk were removed by several different methods in the early years of the study, as reported by Kumada et al previously [9]. After 1991, however, TT were removed by the open method under simple occlusion of the portal trunk and both of the first branches of the portal vein because invasion of vessel wall was seldom observed histopathologically. For patients with TT in

Table 2  
Operative procedures performed in patients with HCC and major vascular involvement

Type	No. patients ( <i>n</i> = 112)
Partial resection	1
Sectorectomy	3
Median hepatectomy <sup>a</sup>	3
Left hepatectomy	25
Right hepatectomy	64
Extended left hepatectomy	3
Extended right hepatectomy	13

<sup>a</sup> Resection of segments 4, 5, and 8.

the IVC, the TT were removed under total hepatic vascular exclusion with or without veno-venous bypass. In one patient with TT extending to the right atrium, the TT were removed under cardiopulmonary bypass.

In 75% of patients, tumors were removed macroscopically during the operation. Patients with gross residual tumor received additional treatment based on the number, size, and location of the residual tumors and liver function. As a general principle, intraoperative ethanol injection of the residual tumors was performed. Patients underwent CT approximately 3 weeks after hepatic resection. If CT showed viable tumors, TACE using doxorubicin was performed within 1 month.

The 1-, 3-, and 5-year survival rates for the 845 patients overall were 78%, 57%, and 40%, respectively. The survival for patients with and without TT in the portal vein, and hepatic vein are presented in Figs. 1 and 2, respectively. In patients with HCC, progression of TT in the portal and hepatic veins was associated with a decline in survival rates. A characteristic difference, however, was noted in the prognosis of the subsets of patients with major portal vein involvement versus major hepatic vein involvement. No significant survival differences were seen between patients with TT in the first branch of the portal vein (5-year survival rate, 12%; *n* = 46) and those with TT in the portal trunk (7%, *n* = 45; *P* = 0.438; see Fig. 1). Patients with TT in the hepatic trunk (11%, *n* = 23), however, survived significantly longer than did those with TT invading the IVC (0%, *n* = 18; *P* = 0.008). The outcome of patients with TT in the IVC was poor, and no patient survived more than 2 years (see Fig. 2). TT sometimes were found in the portal and hepatic veins. No significant survival differences were detected among patients with TT only in the portal vein, only in the hepatic vein, or in both the portal and hepatic veins.

Tumor number and size and vascular involvement are important prognostic factors in patients with HCC. In patients with major vascular involvement, however, no significant survival differences were seen between patients with solitary and multiple tumors (Fig. 3a). The survival rate for patients with HCCs of 5 cm or less (26%, *n* = 27) showed a tendency to be better than that for patients with HCCs of more than 5 cm (6%, *n* = 85), but there were no significant differences (*P* = 0.053; Fig. 3b). In patients with

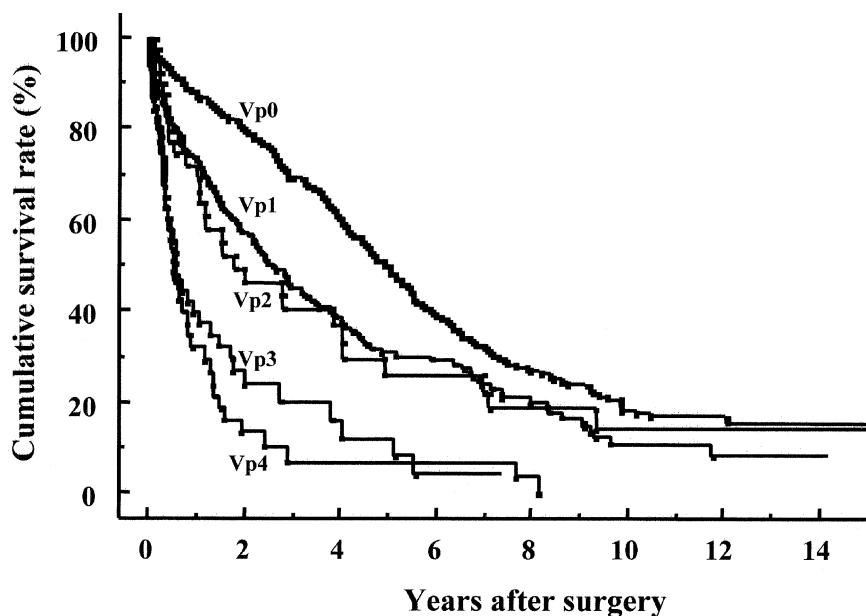


Fig. 1. Survival of patients with HCC with tumor thrombi (TT) in the portal vein. The 5-year survival rate was 50% in patients without TT in the portal vein (Vp0;  $n = 522$ ), 31% in patients with TT distal to the second branch of the portal vein (Vp1,  $n = 192$ ), 26% in patients with TT in the second branch of the portal vein (Vp2,  $n = 40$ ), 12% in patients with TT in the first branch of the portal vein (Vp3,  $n = 46$ ), and 7% in patients with TT in the portal trunk (Vp4,  $n = 45$ ). Regarding major portal vein involvement, no significant survival differences were noted between patients with TT in the first branch of the portal vein and those with TT in the portal trunk ( $P = 0.438$ ).

HCC with major portal and/or hepatic vein involvement, tumor number and size did not affect their prognosis.

Hepatic dysfunction is another significant prognostic factor in patients with HCC. In patients with hepatic vein involvement, no significant differences were noted between the survival rates of patients in clinical stage I and clinical stage II. Patients with portal vein involvement, however, showed a significantly better survival in clinical stage I than in clinical stage II ( $P = 0.001$ ; Figs. 4a,b).

The prognosis was not different between curative and palliative resections. The 5-year survival rate for patients with portal vein invasion who underwent curative resection was 11% ( $n = 70$ ), whereas the 5-year survival rate for patients undergoing palliative resection was 5% ( $n = 21$ ). The 5-year survival rate for patients with hepatic vein invasion who underwent curative resection was 6% ( $n = 29$ ), whereas it was 8% ( $n = 12$ ) for patients undergoing palliative resection.

Between 1985 and 1988, the postoperative mortality rate was 33% (9 of 27 patients). In 8 patients, death was related directly to hepatic resection,

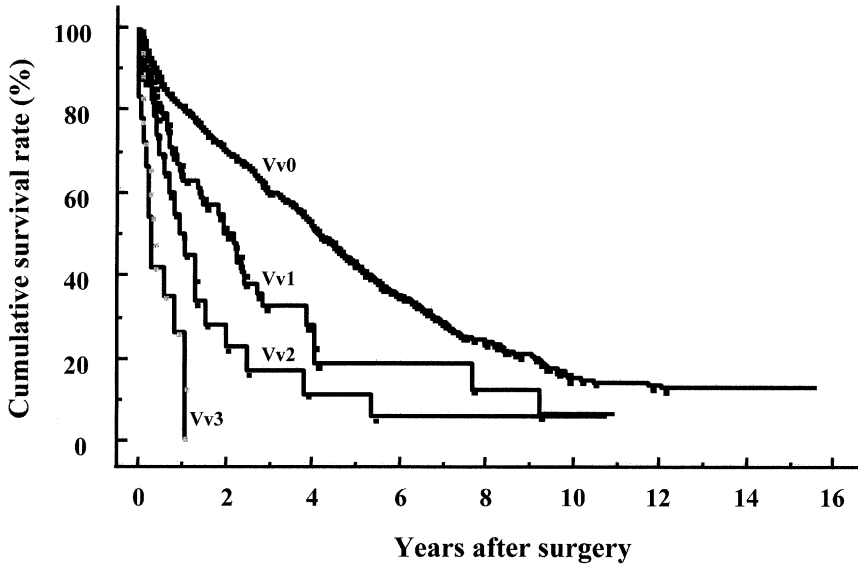


Fig. 2. Survival of patients with HCC with tumor thrombi (TT) in the hepatic vein. The 5-year survival rate was 43% (Vv0,  $n = 747$ ) in patients without TT in the hepatic vein, 19% in patients with TT in a branch of the hepatic vein (Vv1,  $n = 57$ ), 11% in patients with TT in the hepatic vein trunk or the short hepatic vein (Vv2,  $n = 23$ ), and 0% in patients with TT in the inferior vena cava (IVC; Vv3,  $n = 18$ ). Patients with TT in the hepatic vein trunk had a significantly better survival rate than did those with TT invading the IVC ( $P = 0.008$ ).

resulting from intra-abdominal bleeding in 4 patients and from postoperative liver failure or multiple organ failure in 4 others. One patient died of heart failure. After 1989, the postoperative mortality rate decreased to 2.4% (2 of 85 patients). One patient died of respiratory failure caused by bronchial bleeding. The other patient with TT in the IVC, who had undergone IVC plasty and metallic stent insertion in the IVC because of Budd-Chiari syndrome, died of multiple organ failure.

## Discussion

Vascular invasion is one of the most important prognostic factors for HCC because portal vein TT and hepatic vein TT lead to intrahepatic and systemic metastases [18–20]. The Liver Cancer Study Group of Japan reported that the incidence of macroscopic TT was 14.4% in the portal vein and 4.2% in the hepatic vein in surgically treated patients, and 62% in the portal vein and 26% in the hepatic vein in autopsy cases [21]. The median survival time of the nontreated patients with HCC and with TT in the portal vein was only 2.7 months [22]. Furthermore, the prognosis of patients treated by systemic chemotherapy also was poor [23].

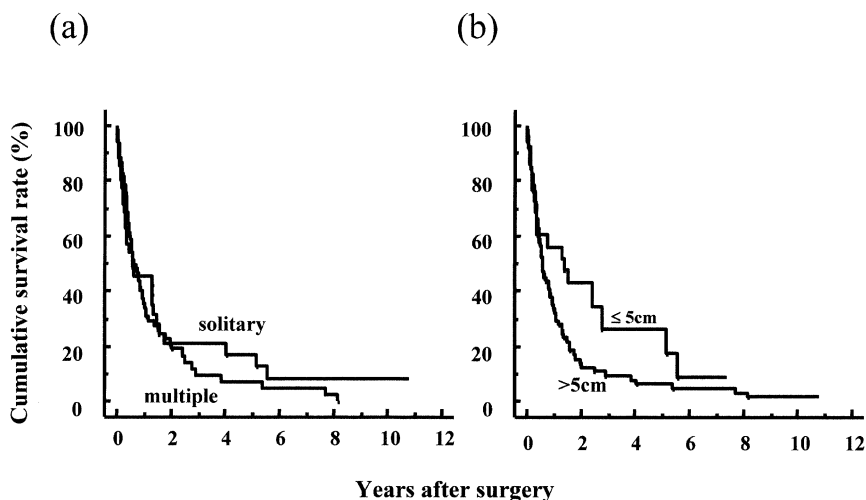


Fig. 3. Survival of patients with HCC with major vascular involvement according to the number of tumors (a) and tumor size (b). There were no significant differences between patients with solitary ( $n = 35$ ) and multiple tumors ( $n = 77$ ;  $P = 0.56$ ), and between patients with HCCs of 5 cm or less ( $n = 27$ ) and patients with HCCs of more than 5 cm ( $n = 85$ ;  $P = 0.053$ ).

Local ablation therapies, such as PEIT and RFA, and TACE are widely used as nonsurgical therapeutic modalities. Local tumor ablation is beneficial in treating a limited number of small HCCs without vascular invasion but is ineffective in patients with large lesions and portal vein TT [24]. TACE provides effective palliation for patients with advanced HCC and recurrent tumors. Preexisting TT of the portal trunk or the right first branch, however, have been considered a contraindication to TACE because occlusion of the hepatic arterial blood supply theoretically could result in liver failure from ischemia [7]. Among patients with HCC invading the portal and/or hepatic veins, the possibilities of surgical cure have been dismissed by some [25]. Only a few investigators have studied the surgical indication for patients with highly advanced HCC. TT in the portal trunk, however, sometimes lead to intrahepatic metastasis and portal hypertension. Occasionally, they induce a life-threatening complication (eg, bleeding from esophageal varices, which is the cause of death in 9% of patients with HCC) [21]. This serious complication can be avoided through cyto-reductive surgery combined with removal of TT in the portal vein. The latter recanalizes the portal vein with the option of further treatment with TACE.

The T category in macroscopic stage classification of HCC is defined by three tumor factors (tumor size, multiplicity of tumors, and vascular invasion) [16] because these three factors are important prognostic factors in patients with HCC [18]. In this study, however, tumor size and multiplicity of tumors did not affect the prognosis in patients with major vascular

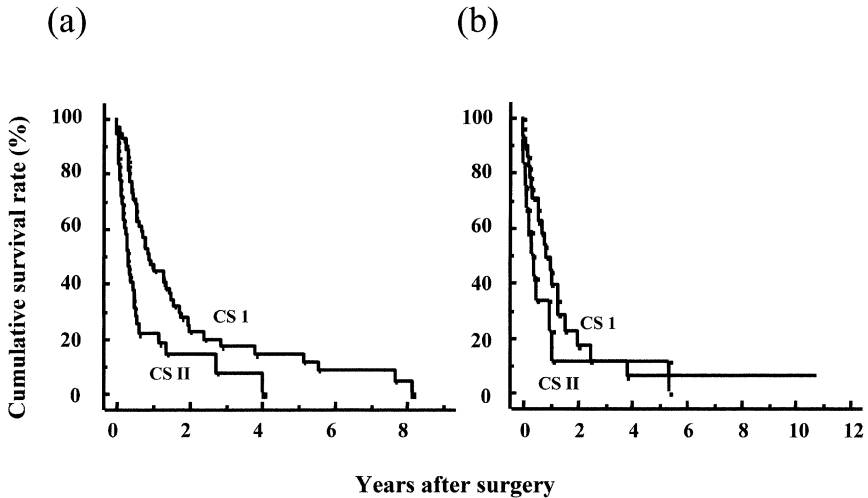


Fig. 4. Survival of patients with HCC with tumor thrombi (TT) in the portal vein (a) and the hepatic vein (b) according to clinical stage classification, excluding one patient in clinical stage III with TT both in the portal and hepatic veins. Patients with portal vein involvement in clinical stage I (CS I;  $n = 54$ ) had a significantly better survival than did patients in clinical stage II (CS II;  $n = 36$ ;  $P = 0.001$ ). Among patients with hepatic vein involvement, no significant differences were noted in the survivals of patients in CS I ( $n = 28$ ) versus CS II ( $n = 12$ ;  $P = 0.159$ ).

involvement. These results suggest that major vascular involvement may be the most important prognostic factor in patients with HCC.

For patients with major vascular invasion, a major hepatic resection is required to resect the main tumor and TT. Approximately 80% of patients with HCC in Japan have associated liver cirrhosis [21], increasing the difficulty of a major hepatic resection. Nagasue et al [26] recommended that a major hepatectomy be performed only in selected patients with Child's A status. Midorikawa et al [27] suggested that a major hepatic resection should not be performed if  $ICGR_{15}$  exceeds 20%. These indications are not always appropriate on evaluation of hepatic functional reserve for patients with major vascular invasion. In half the patients who had major vascular invasion in the present study,  $ICGR_{15}$  was more than 15%, and 37% of patients were in clinical stage II, which is similar to Child's B status. Improvements in surgical techniques and perioperative management also have decreased fatal complications markedly in the last 13 years. Among patients with vascular invasion, the mortality rate has been 2.4%, and surgical procedures for patients with TT in the major portal and/or hepatic veins have been established.

Liver dysfunction is another important prognostic factor for HCC [18,28]. Among patients with TT in the portal vein, the survival of patients in clinical stage I is significantly better than that of patients in clinical stage II.

In the current study, however, accompanying liver dysfunction did not affect the survival rate of patients with TT in the hepatic vein. Patients with TT in only the hepatic vein had a better prognosis than did those with IVC TT. No patients with TT invading the IVC survived more than 2 years; this was because of early distant organ metastasis, especially lung metastasis. These results suggest that systemic tumor spreading from the TT in the IVC has a deleterious effect on the prognosis of patients with hepatic vein invasion, even if their liver function is sufficient for a major hepatic resection. Therefore, surgical treatment is indicated for patients with TT in the portal vein or in the hepatic vein trunk, but not in the IVC.

For patients with liver dysfunction, liver transplantation offers the theoretic appeal of complete removal of the tumor-bearing liver and restoration of normal liver function for those with small HCCs [5] and sometimes for those with advanced HCC [29,30]. Mazzaferro et al [5] reported that patients with HCC with a limited size and number of tumors and without vascular involvement who undergo liver transplantation have a good prognosis. The prognosis for patients with vascular invasion treated by liver transplantation is poor, however. Ringe et al [30] reported that the 5-year survival rate of patients with HCC and with TT in the portal vein was 8.3% for those treated by hepatic resection and 5.3% for those treated with liver transplantation. Iwatsuki et al [29] reported a mean survival time of only 1.7 years after liver transplantation in patients with macroscopic vascular invasion. Given the serious shortage of donor organs, these results do not encourage liver transplantation for patients with advanced HCC.

Improvements in operative procedures and perioperative management have allowed safe resection in patients with advanced HCC. Although we have some patients who had TT in the major vessels and survived longer than 5 years, the prognosis in this subset of patients with HCC remains poor. Recently, Lee et al [31] reported that TACE can be a safe treatment modality for nodular type HCC with portal trunk obstruction when patients have good hepatic functional reserve and enough collateral circulation around the portal trunk. Minagawa et al [32] reported better survival in patients with TT in the portal vein treated with TACE who met the following criteria: no more than two primary nodules, no portal trunk occlusion, and an ICGR<sub>15</sub> of better than 20%. In that report, the 5-year survival of patients who met the criteria and underwent hepatic resection was 42%. The selection of patients for hepatic resection is one of the most important processes. In the same report, however, 60% of patients did not undergo hepatic resection and their mean survival time was only 4.3 months [32]. The outcome of these patients must be improved. Some pilot studies have reported that repeated regional chemotherapy through the hepatic artery is useful in treating patients with advanced HCC with TT in the portal vein [33–35]. New multidisciplinary treatments, such as surgical resection combined with preoperative or postoperative TACE and repeated

regional chemotherapy, are required to improve survival rates among patients with HCC with major vascular invasion.

## References

- [1] Okuda K. Hepatocellular carcinoma: recent progress. *Hepatology* 1992;15:948–63.
- [2] Takayasu K, Moriyama N, Muramatsu Y, et al. The diagnosis of small hepatocellular carcinomas: efficacy of various imaging procedures in 100 patients. *Am J Roentgenol* 1990;155:49–54.
- [3] Curley SA, Izzo F, Ellis LM, et al. Radiofrequency ablation of hepatocellular cancer in 110 patients with cirrhosis. *Ann Surg* 2000;232:381–91.
- [4] Livraghi T, Giorgio A, Marin G, et al. Hepatocellular carcinoma and cirrhosis in 746 patients: long-term results of percutaneous ethanol injection. *Radiology* 1995;197:101–8.
- [5] Mazzaferro V, Regalia E, Doci R, et al. Liver transplantation for the treatment of small hepatocellular carcinomas in patients with cirrhosis. *N Engl J Med* 1996;334:693–8.
- [6] Takayasu K, Wakao F, Moriyama N, et al. Postresection recurrence of hepatocellular carcinoma treated by arterial embolization: analysis of prognostic factors. *Hepatology* 1999;16:906–11.
- [7] Yamada R, Kishi K, Sato M, et al. Transcatheter arterial chemoembolization (TACE) in the treatment of unresectable liver cancer. *World J Surg* 1995;19:795–800.
- [8] Mori K, Ozawa K, Yamamoto Y, et al. Response of hepatic mitochondrial redox state to oral glucose load-redox tolerance test as a new predictor of surgical risk in hepatectomy. *Ann Surg* 1990;211:438–46.
- [9] Kumada K, Ozawa K, Okamoto R, et al. Hepatic resection for advanced hepatocellular carcinoma with removal of the portal vein tumor thrombi. *Surgery* 1990;180:821–7.
- [10] Kumada K, Shimahara Y, Fukui K, et al. Extended right hepatic lobectomy: combined resection of IVC and its reconstruction by ePTFE graft (Gore-Tex). *Acta Chir Scand* 1988;154:481–3.
- [11] Yamaoka Y, Ozawa K, Tanaka A, et al. New device for harvesting a hepatic graft from a living donor. *Transplantation* 1991;52:157–60.
- [12] Yamaoka Y, Ozawa K, Kumada K, et al. Total vascular exclusion for hepatic resection in cirrhotic patients. *Arch Surg* 1992;127:276–80.
- [13] Kiuchi T, Ozawa K, Yamamoto Y, et al. Changes in arterial ketone body ratio in the phase immediately after hepatectomy. *Arch Surg* 1990;125:655–59.
- [14] Ozawa K, Aoyama H, Yasuda K, et al. Metabolic abnormalities associated with postoperative organ failure. *Arch Surg* 1983;118:1245–51.
- [15] Ikai I, Yamaoka Y, Yamamoto Y, et al. Surgical intervention for patients with stage IV-A hepatocellular carcinoma without lymph node metastasis. *Ann Surg* 1998;227:433–9.
- [16] Liver Cancer Study Group of Japan. Classification of primary liver cancer (first English edition). Tokyo: Kanehara & Co; 1997.
- [17] Couinaud C. Plaidoyer pour une segmentation hépatique exacte et une technique anatomique de résection réglée du foie. *Presse Med* 1966;74:2849–52.
- [18] Liver Cancer Study Group of Japan. Predictive factors for long-term prognosis after partial hepatectomy for patients with hepatocellular carcinoma in Japan. *Cancer* 1994; 74:2772–80.
- [19] Tsai TJ, Chau GY, Lui WY, et al. Clinical significance of microscopic tumor venous invasion in patients with resectable hepatocellular carcinoma. *Surgery* 2000;127:603–8.
- [20] Vauthey JN, Klimstra D, Franceschi D, et al. Factors affecting long-term outcome after hepatic resection for hepatocellular carcinoma. *Am J Surg* 1995;169:28–35.
- [21] Liver Cancer Study Group of Japan. Primary liver cancer in Japan. *Ann Surg* 1990;211:277–87.

- [22] Llovet JM, Bustanante J, Castells A, et al. Natural history of untreated nonsurgical hepatocellular carcinoma: rationale for the design and evaluation of therapeutic trials. *Hepatology* 1999;29:62–7.
- [23] Okada S, Okazaki N, Nose H, et al. Prognostic factors in patients with hepatocellular carcinoma receiving systemic chemotherapy. *Hepatology* 1992;16:112–7.
- [24] Allgaier HP, Deibert P, Olschewski M, et al. Survival benefit of patients with inoperable hepatocellular carcinoma treated by a combination of transarterial chemoembolization and percutaneous ethanol injection—a single-center analysis including 132 patients. *Int J Cancer* 1998;79:601–5.
- [25] Schwartz ME, Sung M, Mor E, et al. Multidisciplinary approach to hepatocellular carcinoma in patients with cirrhosis. *J Am Coll Surg* 1995;180:596–603.
- [26] Nagasue N, Yukaya H, Ogawa Y, et al. Clinical experience with 118 hepatic resections for hepatocellular carcinoma. *Surgery* 1986;99:694–701.
- [27] Midorikawa Y, Kubota K, Takayama T, et al. A comparative study of postoperative complications after hepatectomy in patients with and without chronic liver disease. *Surgery* 1999;126:484–91.
- [28] Yasui M, Harada A, Torii A, et al. Impaired liver function and long-term prognosis after hepatectomy for hepatocellular carcinoma. *World J Surg* 1995;19:439–43.
- [29] Iwatsuki S, Dvorchik I, Marsh JW, et al. Liver transplantation for hepatocellular carcinoma: a proposal of a prognostic scoring system. *J Am Coll Surg* 2000;191:389–94.
- [30] Ringe B, Pichlmayr R, Wittekind C, et al. Surgical treatment of hepatocellular carcinoma: experience with liver resection and transplantation in 198 patients. *World J Surg* 1991;15:270–85.
- [31] Lee HS, Kim JS, Choi JJ, et al. The safety and efficacy of transcatheter arterial chemoembolization in the treatment of patients with hepatocellular carcinoma and main portal vein obstruction. *Cancer* 1997;79:2087–94.
- [32] Minagawa M, Makuuchi M, Takayama T, et al. Selection criteria for hepatectomy in patients with hepatocellular carcinoma and portal vein tumor thrombus. *Ann Surg* 2001;233:379–84.
- [33] Ando E, Yamashita F, Tanaka M, et al. A novel chemotherapy for advanced hepatocellular carcinoma with tumor thrombosis of the main trunk of the portal vein. *Cancer* 1997;79:1890–6.
- [34] Chung YH, Song IH, Song BC, et al. Combined therapy consisting of intra-arterial cisplatin infusion and systemic interferon- $\alpha$  for hepatocellular carcinoma patients with major portal vein thrombosis or distant metastasis. *Cancer* 2000;88:1986–91.
- [35] Toyoda G, Nakano S, Kumada T, et al. The efficacy of continuous arterial infusion of 5-fluorouracil and cisplatin through an implanted reservoir for severe advanced hepatocellular carcinoma. *Oncology* 1995;55:295–9.