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Purpose: We designed this study to evaluate the feasibility of using mangafodipir trisodium enhanced functional liver imaging (MT-FLI) for assessing the viable fraction of hepatocytes (VFH) in the liver.

Materials and Methods: We evaluated the change of VFH with using MT-FLI before and after inducing acute hepatic necrosis (AHN) with CCl4 in the liver of 15 beagle dogs. The MR imaging was performed on a 1.5T MRI unit with using the EFGRE-3D sequence (TR/TE = 4.7/1.1 msec; flip angle: 20.0°). We evaluated the linear dependence of the density of viable hepatocytes in AHN on the MR images as compared to that in the corresponding normal livers (DAHN/DN) on the VFH in their pathologic specimens, and the change of the VFH from the MT-FLI on that of the laboratory findings (AST, ALT, albumin, bilirubin, PT, ICG-R15) before and after AHN induction.

Results: The mean ± SD of the VFH from the MT-FLI in AHN was 61.2 ± 10.7% of that of the normal ones. The DAHN/DN showed strong positive linear dependence on the VFH in their pathologic specimens (β = .769, p < .05). The change of the VFH from MT-FLI showed high linear dependence on that of all the above-mentioned laboratory findings (β < .933 or > .938, p < .05).

Conclusion: The MT-FLI seems to be a feasible method for measuring VFH in the liver.

Index words: Liver, Liver, function, Magnetic resonance (MR), volume measurement, Animals

Hepatic resection for treating hepatocellular carcinoma is now being performed on patients suffering with chronic liver disease (1, 2); however, postoperative liver failure is still an important cause of death after hepatic resection (3-5). In this setting, the liver has a decreased volume of viable hepatocytes that causes delayed regeneration of the remnant liver. For this reason, even a small volume of partial hepatic resection can be fatal in those cases with severe underlying liver cirrhosis or chronic hepatitis. Thus, estimating the hepatic functional reserve based on the actual volume of functioning viable hepatocytes is very important in the therapeutic management of these patients for preventing postoperative hepatic failure.

Various methods have been suggested to evaluate the hepatic functional reserve. The Child-Pugh’s classifica-
tion [6-8] has been widely used to predict the post-hepatectomy mortality. Other dynamic liver tests include the indocyanine green (ICG) retention test [9], the arterial ketone body ratio evaluation method [10] and the liver resection index as calculated from the data of the 14C-aminopyrine breath test. However, all of previously recommended methods to evaluate the hepatic functional reserve are based on the results of indirect examinations, and no laboratory finding-based test can directly provide information on the volume of viable hepatocytes. Therefore, the development of a direct method for evaluating the viable fraction of hepatocytes in the liver is increasingly required.

Many efforts to evaluate the functioning volume of hepatocytes with performing hepatic scintigraphy have been reported [11, 12]. A few previous studies reported that the hepatic functional impairment could be detected by hepatocyte-specific contrast agent-enhanced MR imaging [13, 14]. However, no studies involving direct evaluation of the viable fraction of hepatocytes in the liver on MR images with using hepatocyte-specific contrast agents have yet been performed.

Mangafodipir trisodium was formerly referred to as manganese dipyridoxyl diphosphate (Mn-DPDP), and it undergoes specialized intracellular uptake by hepatocytes [15]. The majority of the Mn in this contrast agent is taken up by the hepatocytes in the normally functioning liver [16]. For this reason, mangafodipir trisodium has the potential to generate functional T1-weighted MR images that consist of selectively enhanced viable hepatocytes.

Mangafodipir trisodium enhanced functional liver imaging is a newly designed method for measuring the actual fractional volume of viable hepatocytes in a selected liver, as compared with that of the normal liver, with using mangafodipir trisodium enhanced MR imaging and volumetry.

The purpose of this study was to evaluate the feasibility of mangafodipir trisodium enhanced functional liver imaging for assessing the viable fraction of hepatocytes in the liver.

**Materials and Methods**

**Experimental Model**

Fifteen healthy adult female beagle dogs (average weight: 13.5 kg) were used in this study, and the experimental protocol was approved by the animal research committee for the use of laboratory animals at our institute. All of our subjects were anesthetized with intramuscular injections of ketamine (Ketalar; Yuhan Yanghang, Seoul, Korea) 50 mg/kg of body weight and xylazine hydrochloride (Rompun; Bayer Korea, Seoul, Korea) 5 mg/kg, and they were intubated using a 7-mm cuffed endotracheal tube. Small supplementary doses of ketamine (total dose range: 10-20 mg/kg) were given intermittently during the course of the experiment as needed to maintain adequate levels of sedation.

Under general anesthesia, we obtained initial blood samples to evaluate the baseline liver function. The liver function test included the serum levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), albumin and total bilirubin and the prothrombin time. After initial sampling of blood, we intravenously injected 0.5 mg/kg of indocyanine green (ICG), and we obtained blood samples to measure the retention rate of ICG 15 minutes after administration (ICG-R15). After those blood samplings, all of our subjects underwent the initial mangafodipir trisodium enhanced MR imaging and volumetry for their livers.

Magnetic Resonance Imaging

All of our subjects were imaged with a 1.5-T MR unit (Signa Horizon, GE Medical System, Milwaukee, WI, U.S.A.) using a phased-array torso coil and a 3-dimensional enhanced fast gradient recalled echo (EFGRE-3D) sequence. Each anesthetized dog was placed in the supine position and it was tightly fixed using cotton tape to extend their anterior limbs upwardly. The MR imaging was performed with the following parameters: repetition time/echo time: 6.3/1.1 msec, flip angle: 20 degree, sampling bandwidth: 62.5 kHz, field of view: 28×21 cm, matrix: 256×128, section thickness: 7 mm, and the number of excitations: 1. The number of sections was individually adapted to ensure coverage of the entire liver and this ranged between 13 and 24 sections. The axial non-enhanced T1-weighted images (NE-T1WI) and axial contrast-enhanced T1-weighted images (CE-T1WI) were obtained in all cases. The CE-T1WI was acquired 30
Intravenous injection of doses of triiodinated contrast medium (Teslascan™, Amersham Health, Oslo, Norway) in the same planes with the NE-T1WI. During the image acquisition, breath-holding of the animals was performed with the tidal expiration level, and this was kept steady at this level without compression of the air bag connected to the intubated endotracheal tube.

**Histologic Examination**

All the animal subjects were euthanized by an intravenous injected overdose of thiopental sodium (Pentothal; Choong Wae Pharmacy, Seoul, Korea) just after the acquisition of the second MR images on the fifth experimental day. Then, the whole liver was removed and fixed in 10% buffered formalin. Histologic examination was performed on the hematoxylin-eosin (H & E)-stained axial sections, which corresponded to the MR imaging plane. We also measured the fractions of viable hepatocytes on the histologic specimens under a light microscope (Carl Zeiss, Jena, Germany) that was linked to a computer-assisted image analysis system (KS400, ver. 2.0; Carl Zeiss, Jena, Germany) with 512-pixel resolution. Three randomly selected areas were analyzed and averaged in each specimen. The images were collected at a magnification of 40X, with the maximum area examined in the horizontal X-Y plane of 400 μm X 400 μm. The area occupied by viable hepatocytes was calculated by the area of the viable hepatocytes/the area of the X-Y plane X 100; this was expressed as the percent of the area of viable hepatocytes.

**Analysis of the Functional Liver Imaging and Statistical Methods**

Post-processing was carried out on a workstation (SUN SPARC II; Sun Microsystems, Mountain View, CA, U.S.A.) with using computer software (Advantage Windows 4.0; GE Medical Systems, Milwaukee, WI, U.S.A.). We measured the differences of the signal intensity (SI) from the volume of interest (VOI) of the whole liver between the NE-T1WI and the CE-T1WI. We also measured the SI of the subcutaneous fat in the back from the 2 mm² sized region of interest (ROI) in all the imaging planes. We calculated the standardized SI (SSI) of the liver for quantifying the SI. The SSI of the liver was defined as (the SI of the liver/the SI of the subcutaneous fat of the back) X 100 in each imaging plane. The SSI from the VOI of the whole liver was calculated by averaging of the summed SSI of the liver in all imaging planes. To determine the boundary of the liver where the SI was acquired, a radiologist manually traced the outline of the liver in each section. The tracing excluded the gallbladder and the retro-hepatic cava, but it included the intra-hepatic biliary and vascular structures. We also performed three dimensional volumetry with the same sets of data that were used when acquiring the SI. The computer stacked the data from each section, allowing three dimensional volumetric reconstructions and measurements to be obtained.

The measurement of the volumetric fraction that consists of the viable hepatocytes could be accomplished by calculating the density of the viable hepatocytes in a unit volume multiplied by the total liver volume.

We established the following two equations for defining the density of the viable hepatocytes in a unit volume and the total volumetric fraction of the viable hepatocytes in the liver. The density of viable hepatocytes in a certain liver (DC) could be defined by measuring the differences of the SSI of the whole liver between NE-T1WI (SSI_NE) and CE-T1WI (SSI_CE) with using the following equation:

\[
D_C = SSI_{CE} - SSI_{NE}
\]

We could obtain the density of the viable hepatocytes in the normal liver (DN) by applying the above equation to the normal livers before treatment with CCl₄. We could also obtain the volume of the liver by 3-D volumetry, and the volume before treatment with CCl₄ was used as a normal liver volume (VN) in a selected case.

With using the DN and VN as the standards of the compactness of the viable hepatocytes and the normal liver volume in the cases before CCl₄ treatment and with the VC as the volume of a certain liver, the viable fraction of hepatocytes in a certain liver (FC) compared to the normal one could be calculated by the following equation:

\[
F_C = \frac{|D_C/D_N| \times (V_C/V_N)|}{100}\%
\]

Linear regression analysis was performed to assess the linear dependence of the ratio of the calculated density of viable hepatocytes in the liver with acute hepatic necrosis to the normal one (DAHN/DN) for the proportions of those variables on the histologic specimens that were evaluated by an image analyzer with using commercially available software (SPSS version 11.0; SPSS, Chicago, IL, U.S.A.). We also evaluated the linear dependence of the viable fraction of hepatocytes, as calculated by mangafodipir trisodium enhanced functional liver imaging, on the various criteria of the liver function tests and the ICG-R15 with using linear regression analysis. A p value of less than .05 was considered a sta-
Fig. 1. Estimation of the density of viable hepatocytes and the liver volume

The representative MR images of the liver of a dog (dog 1) before [A, B, C] and after [D, E, F] the CCl₄ treatment: The SI from the VOI of the whole liver is calculated by averaging of the summed signal intensities of the liver in all imaging planes of NE-T1WI [A, D] and CE-T1WI [B, E]. Each MR image shows the outlined liver by manually traced lines and a small rectangle in the subcutaneous fat in the back in order to obtain the SSI of the whole liver. They show (20.7 - 15.0)/20.7 difference in SSI between the images before and after the CCl₄ treatment, which means a 27.5% (5.7/20.7) decrease in the density of viable hepatocytes. The three dimensional volumetric reconstructions of the liver with the same sets of data that were used in acquirement of the SI show the volumetric data of the livers before [C] and after [F] the CCl₄ treatment. They show a decrease in liver volume from 947 to 812 ml. Therefore, the calculated viable fraction of hepatocytes of the liver after the treatment of CCl₄ is 62.1% (72.5% × 85.7%) of that before the treatment.
Results

In all cases, we successfully performed mangafodipir trisodium enhanced functional liver imaging and we obtained the calculated viable fraction of hepatocytes on the MR images (Fig. 1). The clinical characteristics of all the subjects with normal liver and the subjects with acute hepatic necrosis are described in Table 1. All of the subjects showed diminished $D_{AHN}$ as compared to the corresponding $D_N$. They showed a $D_{AHN}$ of 47.8%-88.2% in comparison to their corresponding $D_N$ with a mean ± standard deviation of 70.7±10.2%. The cases in the acute hepatic necrosis group also showed a more decreased liver volume than that of the corresponding cases in the normal group. They showed 75.3%-93.9% of the volume of their corresponding normal cases with a mean of 86.2±5.3%. The range of the calculated viable fraction of hepatocytes in the livers with acute hepatic necrosis, as determined by mangafodipir trisodium enhanced functional liver imaging, was 36.0%-73.9% in comparison with that of the corresponding normal cases with a mean of 61.2±10.7%. The fraction of the viable hepatocytes in the liver of the acute hepatic necrosis group, with using an image analyzer, was 51.2%-86.1%, and the ratio of $D_{AHN}/D_N$ showed a strong positive linear dependence on the proportion of viable hepatocytes in their corresponding pathologic specimens ($\beta = .769, p < .05$) (Fig. 2). The serum level of AST, ALT and bilirubin in the cases of the acute hepatic necrosis group were markedly increased as 342.8%-605.3%, 2342.9%-10121.4%, and 766.7%-2500.0% as compared to their corresponding normal cases, respectively. The serum level of albumin in the acute hepatic necrosis group was decreased to 14.3%-45.5% of their normal levels. The cases of the acute hepatic necrosis group showed a prothrombin time of 27.0%-77.0% in comparison to those in the normal group. The ICG-R15 value was increased from 1.0%-7.6% in the normal group to 20.0%-28.7% in the acute hepatic necrosis group. The change of the viable fraction of hepatocytes on the mangafodipir trisodium enhanced functional liver images showed a significant linear dependence to that of all the criteria of our laboratory findings used to evaluate liver function, including the serum level of AST ($\beta = -.944, p < .05$), ALT ($\beta = -.972, p < .05$), albumin ($\beta = .938, p < .05$), bilirubin ($\beta = -.933, p < .05$), the prothrombin time ($\beta = .957, p < .05$), and ICG-R15 ($\beta = -.944, p < .05$) (Fig. 2).

Discussion

In this study, we successfully evaluated the viable

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<th>Subj.</th>
<th>AST</th>
<th>ALT</th>
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Abbreviations: Pre, in the liver before inducing acute hepatic necrosis; Post, in the liver after inducing acute hepatic necrosis; AST, serum level of aspartate aminotransferase; ALT, serum level of alanine aminotransferase; Bil, bilirubin, mg/dL; Alb, albumin, g/dL; PT, prothrombin time, %; ICG-R15, the retention rate of ICG 15 minutes after administration, %; $D_N$, the density of viable hepatocytes in the normal liver; $D_{AHN}$, the density of viable hepatocytes in the liver with acute hepatic necrosis; Liver Vol., the volume of the liver, ml; VFH-FLI (post), viable fraction of hepatocytes in the liver with acute hepatic necrosis as calculated by the mangafodipir trisodium enhanced functional liver imaging, (%)
fraction of hepatocytes on the MR images by the method of mangafodipir trisodium enhanced functional liver imaging and we correlated it with the histopathologic results. That result implied that the amount of viable hepatocytes in the liver could be evaluated by mangafodipir trisodium enhanced functional liver imaging and it might be a possible method for getting the volumetric information of viable hepatocytes in the liver, which is considered as one of the very important prognostic factors in partial hepatectomy.

Various methods have been suggested to predict the postoperative prognosis of patients after partial hepatectomy. One of the most widely used methods is the Child-Pugh classification system. Franco et al. [7] reported that the postoperative mortality rate of the Child-Pugh class A patients was 3.7%, whereas that in the Child-Pugh class B and C patients was 16.7%. Nagasue et al. [17] also reported that there were significant differences in the postoperative mortality rate between the groups with different Child-Pugh classes. Among the criteria of the Child-Pugh classification system, the prothrombin time is known as a universal indicator of the
severity of liver failure. For liver diseases, the prothrombin time is just a measurement of synthetic liver function. Indeed, the prothrombin time is incorporated in almost all the prognostic models used worldwide for making major therapeutic decisions such as liver transplantation for patients with acute liver failure and cirrhosis or for administering steroid therapy to alcoholic hepatitis (18–20). The results of our study also revealed that the significant linear dependence of the change of the viable fraction of hepatocytes on the mangafodipir trisodium enhanced functional liver images to that of the prothrombin time in the acute hepatic necrosis group. This implied that the mangafodipir trisodium enhanced functional liver imaging could provide information on the synthetic liver function as well. In our results, the change of the viable fraction of hepatocytes on the mangafodipir trisodium enhanced functional liver images also showed a significant linear dependence to that of the serum levels of AST and ALT, and these 2 parameters represented the severity of the destruction of the hepatocytes. Those results indicated that mangafodipir trisodium enhanced functional liver imaging also provided the functional information based on the viable fraction of hepatocytes.

Attempts to evaluate the hepatic function with various imaging modalities have been reported. 99mTc-galactosyl-human serum albumin (Tc-GSA) scintigraphy has been reported to be useful for evaluating the hepatic function, as based on the hepatocyte volume in various physiologic and pathological conditions (12). Kwon et al. (11) have reported that the functional hepatic volume could be measured with Tc-GSA scintigraphy. However, scintigraphy has the disadvantages of hazardous radiation and poor spatial resolution. The mangafodipir trisodium enhanced functional liver imaging is safe from any radiation hazard, as well as it provides the hepatic functional information. Moreover, the mangafodipir trisodium enhanced MR image could provide additional information about anatomical change, and so it has an advantage in detecting and characterizing the focal lesions in the liver with high spatial resolution. Recent studies have reported that diffusion weighted MR imaging and perfusion imaging were useful in evaluating the severity of chronic hepatitis (21, 22). However, those researchers could not estimate the volume of the functioning hepatocytes, whereas the volumetric measurement of viable hepatocytes could be achieved by mangafodipir trisodium enhanced functional liver imaging.

Despite its limited capability for investigating the hepatic functional reserve, the ICG test is widely used for preoperative evaluation of liver function, and it is the mainstay for making decisions about performing hepatectomy or its extent (23). ICG as an organic anion is rapidly and completely bound by plasma protein, mainly beta-lipoprotein, after venous administration; it is exclusively removed by the hepatic parenchymal cells. The ICG uptake into the cell through the membrane involves active transport using ATP and then ICG is secreted into the bile (24). The mechanism of transferring mangafodipir trisodium to the liver and its uptake to the hepatocytes are similar to those of ICG. In the bloodstream, the manganese of the contrast material is removed from the DPDP ligand; it is bound to plasma proteins and then transferred to the liver with active uptake by the hepatocytes. It is excreted into bile, and this induces prominent and prolonged T1 shortening of the liver and bile ducts (25). Therefore, we can theoretically measure the SI of the hepatic parenchyma on MR images as a result of its selective uptake by viable hepatocytes. In our study, the change of the viable fraction of hepatocytes on mangafodipir trisodium enhanced functional liver images showed a significant linear dependence to that of the ICG-R15. This is thought to happen via a similar mechanism of transfer to the liver and excretion between that of the ICG and the mangafodipir trisodium. That result implied that the mangafodipir trisodium enhanced functional liver imaging might have the potential to indicate the functional reserve of the liver.

One of the most important results of this study was that the $D_{MIN}/D_N$ showed a strong positive linear dependence to the proportion of viable hepatocytes in the histopathologic specimens. This revealed that the change of the SI of the hepatic parenchyma was a direct result of the uptake of manganese by viable hepatocytes. The good correlation between the densities of viable hepatocytes on the MR images and on the histologic results might suggest that the mangafodipir trisodium could selectively enhance the viable hepatocytes on the MR images. That result implied that the actual volume consists of the viable hepatocytes in the whole liver, that is, the functioning liver volume could be measured on MR images by mangafodipir trisodium enhanced functional liver imaging. From the above results, we could analogize that the functioning volume of the viable hepatocytes in the remnant liver after partial hepatectomy might be predicted with mangafodipir trisodium en-
enhanced functional liver imaging if the measurement of the viable fraction of hepatocytes is performed for the future remnant liver, after the partial resection, on the image simulations with MRI for those patients who are being considered for undergoing hepatic surgery.

There were some possible limitations in this study. First, we used the randomly sampled three areas of the liver with acute hepatic necrosis to evaluate the viable fraction of hepatocytes in the pathologic specimens. If hepatic necrosis was to develop unevenly, then the real volume of the viable hepatocytes could have been incorrect. However, acute hepatic necrosis induced by CCl4 is known to be a diffuse parenchymal lesion. Thus, the minimal difference of the actual volume of viable hepatocytes seemed to be acceptable. Second, we manually traced the outline of the liver on each slice to determine the boundary where the SI was acquired. In that procedure, some peripheral region of the liver might have been excluded or a portion of the extra-hepatic structures could have been included in the traced boundary. It might result in incorrectly outlining the liver, which could be improved by the development of automatic organ tracing software. Third, we obtained the three dimensional image reconstruction and volumetric data by summation of the volume of multiple 7 mm-thick image slabs. During the three dimensional image reconstructions, volume averaging might have developed at the margin of each slab. The volume averaging effect could lead a difference in the liver volume between that on the MR images and the real one. This is thought to be a common limitation of current commercial MR machines, which may improved by the development of the imaging sequences with a faster image acquisition time and higher image resolution, which would result in producing more images with finer resolution and thickness in a shorter period of image acquisition. This may also be improved by combined use of an alternative modality that provides more precise three dimensional reconstruction results in shorter periods, such as multi-detector row CT. Fourth, the mangafodipir trisodium enhanced functional liver imaging is more expensive and time consuming to obtain the results, as compared with the conventional methods for evaluating liver function such as the ICG test. However, these qualities seemed to be offset by its advantages for evaluating the anatomical change as well as the hepatic functional status. Moreover, the current post-processing steps are expected to be replaced by simpler ones according to the development of computer technology. Finally, manganese has potential hepatic toxicity. If a manganese compound like mangafodipir trisodium were used as a contrast media for MR imaging, then it could possibly induce aggravation of hepatic dysfunction when it’s applied to human applications. This situation could be improved by developing a non-toxic hepatocyte-specific contrast media. Therefore, further study with other hepatocyte-targeting contrast media and the development of new hepatocyte-specific contrast media without hepatotoxicity are needed.

In summery, the D_{AHV}/D_{N} showed strong positive linear dependence on the viable fraction of hepatocytes in the patients' pathologic specimens (β = .769, \( p < .05 \)). The change of the viable fraction of hepatocytes on mangafodipir trisodium enhanced functional liver images showed high linear dependence on the laboratory findings, including the serum levels of AST, ALT, bilirubin and albumin, the prothrombin time and ICG-R15 (β < .933. or >.938, \( p < .05 \)).

In conclusion, the D_{AHV}/D_{N} well represented the viable fraction of hepatocytes in the patients’ pathologic specimens, and the change of the viable fraction of hepatocytes on the mangafodipir trisodium enhanced functional liver images showed high linear dependence on the findings of liver function tests. Therefore, mangafodipir trisodium enhanced functional liver imaging seems to be a feasible method for measuring the viable fraction of hepatocytes in the liver.

References

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ÀÎÇÏ´ëÇб³ Àǰú´ëÇÐ ÀÀ±ÞÀÇÇб³½Ç

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ÀúÀÚµéÀº °£ ³» »ýÁ¸ °£¼¼Æ÷ºÐÀ² Æò°¡¿¡¼­ ¸Á°¡Æ÷µðÇǸ£ Æ®¸®¼Òµð¿ò Á¶¿µÁõ°­ ±â´ÉÀû °£ ¿µ»ó¹ýÀ» ÀÌ¿ëÇÑ

´ë»ó°ú ¹æ¹ý:
ÀúÀÚµéÀº 15¿¹ÀÇ °³ °£¿¡¼­ CCl4¸¦ ÀÌ¿ëÇÏ¿© À¯¹ßÇÑ ±Þ¼º °£±«»ç ÀüÈÄÀÇ »ýÁ¸ °£¼¼Æ÷ºÐÀ² º¯È­¸¦ ±â´ÉÀû °£¿µ»ó¹ýÀ» ÀÌ¿ëÇÏ¿© ¾Ë¾Æº¸¾Ò´Ù. ÀÚ±â°ø¸í¿µ»ó¼Ò°ß¿¡¼­ Á¤»ó°ú ºñ±³ÇÑ »ýÁ¸ °£¼¼Æ÷ºÐÀ²ÀÇ ¿ó°ü°ü°è ¹× ±Þ¼º °£ ±«»ç À¯¹ß ÀüÈÄÀÇ °£±â´É°Ë»ç¼Ò°ß (AST, ALT, albumin, bilirubin, PT, ICG- R15) º¯È­¿Í ±â´ÉÀû °£¿µ»ó¼Ò°ß¿¡¼­ »ýÁ¸ °£¼¼Æ÷ºÐÀ² º¯È­°£ÀÇ »ó°ü°ü°è¸¦ ¾Ë¾Æº¸¾Ò´Ù.

°á°ú:
±Þ¼º °£ ±«»ç°¡ À¯¹ßµÈ °£ÀÇ ±â´ÉÀû °£¿µ»ó¼Ò°ß¿¡¼­ »ýÁ¸ °£¼¼Æ÷ºÐÀ²ÀÇ Æò±Õ°ªÀº Á¤»ó»óÅÂÀÇ 61.2±10.7% ¿´´Ù. DAHN/DNÀº Á¶Á÷ º´¸®Ç¥º»¿¡¼­ÀÇ »ýÁ¸ °£¼¼Æ÷ºÐÀ²°ú ³ôÀº »ó°ü°ü°è¸¦ º¸¿´´Ù(¥â= .769, p< .05). ±â´ÉÀû °£¿µ»ó¼Ò°ß¿¡¼­ »ýÁ¸ °£¼¼Æ÷ºÐÀ²ÀÇ º¯È­´Â °£ ±«»ç À¯¹ß ÀüÈÄÀÇ °£±â´É°Ë»ç¼Ò°ß º¯È­¿Í ³ôÀº »ó°ü°ü°è¸¦ º¸¿´´Ù(¥â< -.933 or > .938, p< .05).

°á·Ð:
±â´ÉÀû °£¿µ»ó¹ýÀº °£ ³» »ýÁ¸ °£¼¼Æ÷ºÐÀ²ÀÇ ÃøÁ¤¿¡ À¯¿ëÇÑ ¹æ¹ýÀ¸·Î »ý°¢ÇÑ´Ù.