Pulsatile hepatomegaly in constrictive pericarditis

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SUMMARY In a study of 30 consecutive patients with constrictive pericarditis diagnosed by clinical, radiological, and echocardiographic criteria 21 (70%) were found to have pulsatile hepatomegaly. The pulsations were felt clinically and confirmed by external hepatic recordings. These pulsations conformed almost identically to the jugular venous pulsations in the neck. The hepatic pulsations disappeared after successful pericardiectomy. Persistence of the hepatic pulsations was associated with poor postoperative relief suggesting that it is another useful sign in the assessment of the adequacy of pericardiectomy. Thus this poorly appreciated clinical finding appears to be present in a high proportion of patients with constrictive pericarditis.

The clinical criteria for the diagnosis of constrictive pericarditis are well established.¹⁻³ Of the various criteria, the jugular venous pulsations have received the greatest attention.^{2,3} Although hepatomegaly has been reported to be almost always present, it has been claimed to be non-pulsatile.⁴ Nevertheless, except for isolated reports of a few patients with constrictive pericarditis in whom hepatic pulsations have been felt or recorded these pulsations have received scant attention.^{5,6} The present study was undertaken to assess the prevalence and relation of the hepatic pulsations to the jugular venous pulse.

Patients and methods

Thirty patients (24 men, six women; age range 28-60 (mean 38) years) in whom the diagnosis of constrictive pericarditis was based on clinical, electrocardiographic, radiological, and echocardiographical evidence were studied. In two patients the diagnosis was confirmed by cardiac catheterisation, and in the remainder the diagnosis was confirmed at operation for pericardiectomy.

External hepatic pulsations and jugular venous pulsations were recorded simultaneously using two

piezoelectric transducers on a Cambridge multi-channel photographic recorder at a paper speed of 100 mm/s. The patients were placed in a recumbent or semirecumbent position. One transducer was held firmly over the liver in the area of maximum pulsation, while the other was held over the distended jugular vein. Care was taken to keep the transducer well away from the epigastrium, and the recordings were made during the held mid-inspiratory phase. A surface electrocardiogram was used as a reference tracing. The hepatic tracings were compared with those obtained from the jugular venous pulse, and they were further analysed for their respective wave patterns.

Results

All 30 patients had a raised jugular venous pressure, and the characteristic venous pulsations were observed in all patients in one position or the other.

Hepatic pulsations were detected clinically in 21 of the 30 patients, a prevalence of 70%. These pulsations were best appreciated in the held mid-inspiratory or end-inspiratory phase and were felt as a double pulsation with a prominent diastolic dip after the carotid pulse. Two of the 21 patients had doubtful pulsations in that they were faint and could not be clearly distinguished from aortic or cardiac pulsations.

External hepatic tracings recorded from all 21 patients showed both the "a" and "v" waves, as well as the characteristic "x" and "y" descents of constrictive pericarditis. These tracings correlated almost identically with those obtained from the jugular ven-

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Figure Simultaneous pressure tracings of externally recorded jugular venous (JVP) and hepatic pulsations in a patient with constrictive pericarditis. Note the almost identical phasic relation between the two tracings.

ous pulse (Figure). Interestingly, the two patients who had doubtful clinical pulsations had clearly defined recordable hepatic pulsations. Analysis of these recordings showed that the heights of the "a" and "v" waves were equal in 75% of the patients, whereas the "a" wave was more prominent in the remaining 25%. The "y" descent was deeper than the "x" descent in 67% of patients, and in 33% the "x" descent was equal to or slightly deeper than the "y" descent.

Discussion

In constrictive pericarditis the presence of hepatic enlargement and a raised jugular venous pressure are an invariable finding. Liver pulsations have not, however, received much attention as being a common finding in these patients. In fact, it was once stated that absent hepatic pulsations in the presence of a large hepatomegaly was a diagnostic criterion for the disease.⁴ Only two reports draw attention to the presence of recordable hepatic pulsations in constrictive pericarditis.^{5,6} In the first report, a study of 11 cases, six patients had pulsatile hepatomegaly, a prevalence of 54%.⁵ In the second report, 71%—that is, 10 out of 14 patients—had hepatic pulsations.⁶ This correlates closely with the 70% prevalence we found in our series.

It is conceivable that these pulsations could be mistakenly thought to represent aortic pulsations transmitted through an enlarged liver. Nevertheless, there is usually no obstruction at the mouths of the vena cavae in constrictive pericarditis.⁷ This allows the characteristic changes in the right atrium to be faithfully transmitted to the superior and inferior vena cavae. Thus the phasic form of the hepatic tracings,

showing well marked "a" and "v" waves as well as the rapid "x" and "y" descents, is clear evidence that the pulsations are venous and not arterial. This phasic nature of the hepatic pulsations is manifested clinically by a double pulsation and, in most of our patients, with a prominent diastolic dip after the carotid pulse, coinciding with the "y" descent of the jugular venous pulse.

Analysis of the hepatic recordings showed that in most cases (75%) the "a" and "v" waves were of equal amplitude and that there was a tendency to a deeper "y" descent than the "x" descent. These findings are somewhat different from the two previously reported studies of hepatic pulsations in that they found that the "a" wave was more prominent and that the "x" descent tended to be deeper than the "y" descent.^{5,6} These discrepancies could possibly be explained by the differences in the technique used and the fidelity of the recording equipment in the different studies.

Although we did not perform cardiac catheterisation except in two of our cases, we found no difference in the height of the jugular venous pulse as judged clinically between those with pulsatile hepatomegaly and those without. Similarly, the presence or absence of hepatic pulsations could not be related to the severity of constrictive pericarditis as judged by the increase in the right atrial mean pressure.⁵ It is, however, possible that in patients with milder forms of constrictive pericarditis and thus lower jugular venous pressures, palpable hepatic pulsations may be less common or absent.

There are other factors which may influence the presence or absence of hepatic pulsations in constrictive pericarditis. Liver pulsations may be absent owing to the presence of associated liver disease. The high systemic venous pressure in constrictive pericarditis almost always causes severe hepatic congestion, which when prolonged can lead to so called cardiac cirrhosis. In fact, in the presence of significant centrilobular fibrosis or cardiac cirrhosis the hepatic systolic pulsation may be lost.⁸ There appears to be no current biochemical means of distinguishing a congested non-fibrotic liver from a congested cirrhotic one.⁹ Thus in the absence of a liver biopsy we could not confirm the presence of cirrhosis in our patients as the liver enzymes were variably increased.

Another factor that may play a role is that some patients with constrictive pericarditis may not have a free communication between the right atrium and the inferior vena cava. The pericarditis may cause some constriction around the junction of the right atrium and the inferior vena cava, leading to attenuation of pressure transmitted from the right atrium to the inferior vena cava. In such patients hepatic pulsations may be absent. In most cases of constrictive pericarditis there is no gradient between the right atrium and

the inferior vena cava; however, cases have been reported in which pressure gradients have existed or where decortication around the inferior vena cava has led to appreciable postoperative relief.¹⁰ Finally, many of these patients have gross ascites, which would make both the hepatomegaly and the hepatic pulsations difficult to palpate.

In the present study, in the 28 patients who underwent pericardiectomy the abnormal clinical findings had disappeared or were appreciably less prominent postoperatively. In 20 of the 21 patients with hepatic pulsations these pulsations had disappeared completely after pericardiectomy. One patient who had an incomplete pericardiectomy continued to have hepatic pulsations as well as other signs of constriction. This persistence of hepatic pulsations after inadequate postoperative relief has been noted before and has been proposed as a useful index of the adequacy of pericardiectomy.⁶

In conclusion, pulsatile hepatomegaly is present in a high proportion of patients with constrictive pericarditis. The hepatic pulsations conform very closely to and have the same importance as the jugular venous pulse. An awareness and effort are necessary to elicit the sign.

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