Discussion

It has been shown previously in animals (Ead *et al.*, 1952) and in man (Sharpey-Schafer, 1953) that increased pressure transients in the circulation give rise to reflex vasodilatation by a baroreceptor mechanism. The evidence reported here confirms the theory that, with nervous pathways intact, a decreased pulse pressure produces vasoconstriction and an increased pulse pressure produces vasodilatation. The theory has been shown here to apply not only to the normal but also to the failing circulation. Baroreceptor reflexes are active in heart failure provided that the necessary stimulus is applied, as is shown by the cases with increased pulse pressure and subsequent vasodilatation. The lack of baroreceptor response in those cases showing no pulse-pressure change is in accordance with this view. Further evidence of baroreceptor activity in heart failure in response to pressure changes using other stimuli will be presented elsewhere.

The cases of heart failure reported here were all cases with a raised venous pressure and sinus rhythm. Many other cases of heart disease with a normal venous pressure and patients with abnormal cardiac rhythms have also been investigated, and the results lie on the curve shown in Fig. 5; as might be expected, some are in the normal and some in the pathological part of the graph, with others intermediate in position.

Previous workers (Hamilton *et al.*, 1944; Goldberg *et al.*, 1952; Price and Conner, 1953) have recorded "square wave" patterns on arterial-pressure records in cases of mitral stenosis and occasionally other conditions during the Valsalva manœuvre. The explanations offered have been unsatisfactory, probably because it was not recognized that all forms of heart failure show this phenomenon.

It is of interest that the majority of cases of heart failure did not respond to the acute fall in filling pressure produced during the Valsalva manœuvre by an increased stroke volume, as did the majority of all cases of heart failure studied by the Fick method and venesection (Howarth *et al.*, 1946). The difference in duration of the two sets of observations and the heart-rate changes occurring in the latter group may be sufficient to explain the discrepancy.

Clinical Uses

The main effects of the Valsalva manœuvre may be observed by feeling the pulse in the majority of cases. Rate changes are easily detected, but pulse-pressure changes may also be appreciated. It is usually possible to be certain that the response to the manœuvre is normal by feeling the pulse. Occasionally a spurious "heart failure" response may arise from weak blowing, generally seen in young women who lack the competitive spirit of males and children, or from cheating, which appears to be confined to Royal Air Force personnel who have learned to appose tongue and soft palate, thereby maintaining a mercury column indefinitely without any rise in intrathoracic pressure.

The Valsalva manœuvre may be used to elicit the presence of heart failure in difficult cases and to establish the relative innocence of certain noisy hearts. When used, however, to assess the progress of established cases of heart failure, it shows only too often that little real change has been achieved when they are discharged "compensated" from hospital.

It is also possible by auscultation during the Valsalva manœuvre to decide whether a murmur arises on the right or left side of the heart (Zinsser and Kay, 1950). If the murmur is from the right side it will return immediately on release of the intrathoracic pressure rise; if from the left, a delay of a few beats will be observed. This physical sign is present only if the heart is responding in a normal manner. In most cases of heart failure, since there is no change in stroke output there is no differential accentuation of a murmur. The value of the sign is therefore limited.

Summary

The Valsalva manœuvre causes an acute reduction in the effective filling pressure of the heart and has been used to study the circulation in 62 normal subjects and 63 patients with heart failure. Continuous arterial records showed conspicuous differences in the two groups.

In the normal subjects decrease in arterial-pulse pressure was followed by a rise in diastolic pressure (overshoot). In heart failure from any cause there was usually no change in pulse pressure and no overshoot. In the remaining severe cases the pulse pressure was increased and was followed by a decrease in diastolic pressure.

The results in normal subjects and in heart failure are compatible with the general theory of baroreceptor responses to acute pulse-pressure changes. Thus decrease in pulse pressure was followed by vasoconstriction, and increase in pulse pressure by vasodilatation; unchanged pulse-pressure was without effect peripherally.

The Valsalva manœuvre appears to be the simplest repeatable method available at the moment for studying the differences between the normal and failing circulation. It can be used clinically by feeling the pulse.

REFERENCES

- Dornhorst, A. C., and Leathart, G. L. (1952). *Lancet*, 2, 109.
Ead, H. W., Green, J. H., and Neil, E. (1952). *J. Physiol. (Lond.)*, 118, 509.
Goldberg, H., Elisberg, E. I., and Katz, L. N. (1952). *Circulation*, 5, 38.
Hamilton, W. F., Woodbury, R. A., and Harper, H. T. (1944). *Amer. J. Physiol.*, 141, 42.
Hansen, A. T. (1949). *Acta physiol. scand.*, 19, Suppl. 68.
Howard, P., Leathart, G. L., Dornhorst, A. C., and Sharpey-Schafer, E. P. (1951). *British Medical Journal*, 2, 382.
Howarth, S., McMichael, J., and Sharpey-Schafer, E. P. (1946). *Clin. Sci.*, 6, 41.
Lee, G. de J., Matthews, M. B., and Sharpey-Schafer, E. P. (1954). *Brit. Heart J.*, 16, 311.
Price, H. L., and Conner, E. H. (1953). *J. appl. Physiol.*, 5, 449.
Sharpey-Schafer, E. P. (1952). *Brit. Heart J.*, 14, 544.
— (1953). *J. Physiol. (Lond.)*, 122, 351.
Valsalva, A. M. (1707). *De Aure Humana*, p. 84. Utrecht.
Zinsser, H. F., and Kay, C. F. (1950). *Circulation*, 1, 523.

VENOUS PULSE IN ATRIAL SEPTAL DEFECT: A CLINICAL SIGN

BY

JOHN REINHOLD, B.M., M.R.C.P., D.C.H.

From the Hospital for Sick Children, Great Ormond Street, and the Paediatric Unit, St. Mary's Hospital Medical School, London, W.2

The value of observing the venous pulse in the neck is widely recognized, but accurate identification of the waves by timing them against the heart sounds is less commonly practised. This need not be a difficult procedure provided there is no marked tachycardia, particularly where identification of the *a* and *v* waves is concerned, as the former immediately precedes the first heart sound and the latter occurs directly after the second heart sound. Careful observation of the venous pulse in those types of congenital heart disease that predominantly affect the right side of the heart may be of value in establishing the diagnosis. The importance of the large *a* wave in pulmonary stenosis is well recognized (Abrahams and Wood, 1951; Campbell, 1954). It is surprising, therefore, that little attention has been paid to the venous pulse in atrial septal defect. Wood (1950) mentions that it is usually normal, although the venous pressure may be raised in some instances; and Luisada

(1948) states that there may be a very high auricular wave. The purpose of this communication is to describe the pattern of the jugular phlebogram in this condition, and to emphasize that its most important characteristics may be observed by simple inspection of the neck veins.

Material and Methods

Twenty-five patients with atrial septal defect and 16 normal subjects were included in this investigation. Their ages ranged between 3 and 15 years. The diagnosis in the patients with atrial septal defect was made on the accepted clinical and radiographic criteria (Bedford *et al.*, 1941), and all showed incomplete or complete right bundle-branch block on the electrocardiogram (Barber *et al.*, 1950). The diagnosis was confirmed by cardiac catheterization in 19 patients, and in five of these operation for closure was performed and the nature and size of the defect ascertained.

Synchronous jugular phlebograms with phonocardiograms and electrocardiograms as reference tracings have been recorded from each patient. In the majority of cases recordings have been taken on an Elmquist five-channel "klinik" recorder. With this method the venous pulse was usually recorded direct from the internal jugular vein, the pulsations being transmitted to a crystal of Rochelle salt by a fine metal stylus which rests gently on the skin of the neck overlying the vein. In an occasional case the jugular bulb gave a more satisfactory tracing. It was found that certain precautions were necessary to ensure that satisfactory tracings were obtained, and these apply equally to clinical observation of the venous pulse. First, the patient was propped up to about 45 degrees from the horizontal; secondly, the sterno-mastoid was relaxed by keeping his head in the midline; and, lastly, the point of pulsation from which the recording was made was palpated to be sure that there was no superimposed carotid impulse. Tracings were taken with breathing arrested in mid-respiration.

The height of the *v* wave has proved to be of importance in this investigation, and so an attempt has been made to measure this in each individual by comparing it with the height of the *a* wave in the same complex. The height of the *a* wave has been measured from the upstroke denoting the onset of auricular systole to the peak of the wave, and the height of the *v* wave from the point of maximum systolic collapse (*x*) to the peak of the wave. It may be

argued that the *v* wave should be measured from the point of pulmonary valve closure rather than from the maximum systolic collapse, as the two do not usually coincide in patients with atrial septal defect. However, the above measurement has been adopted because the large wave observed in the neck following the second heart sound is due to the combined effect of a deep systolic collapse and the large *v* wave itself, and it is with the clinical recognition of this wave in relation to the *a* wave that this investigation is concerned.

Results

So far as could be estimated by cardiac catheterization and by other data, the septal defect was of moderate or large size in all these patients. Five patients were known to have associated pulmonary hypertension, and these are considered separately. No patient was in congestive cardiac failure when the tracings were taken. The 16 subjects with normal hearts were included to demonstrate the normal phlebogram by the methods used in this investigation and to indicate the normal ratio of the size of the *a* wave to that of the *v* wave.

Normal Subjects.—Phlebograms from three of these subjects are shown in Fig. 1. The height of the *a* and *v* waves has been compared in each individual; in six they were of equal amplitude; in three the *v* wave was larger than the *a* wave, but in none was it more than one and a half times as great; in two the *a* wave was slightly larger than the *v* wave, and in five it was up to twice as large.

Atrial Septal Defect.—The level of venous pulsation above the sternal angle was measured in each patient with the trunk at about 45 degrees to the horizontal; in all it was raised, the measurement varying between 2 and 6 cm. The characteristic pattern in those cases uncomplicated by pulmonary hypertension was of a normal *a* and *c* wave, a deep and steep systolic collapse, followed by a tall sharp-rising *v* wave (Fig. 2). In those with pulmonary hypertension proved by cardiac catheterization the size of the *a* wave was increased, and the *v* wave, although still abnormally large, was not as tall as in the uncomplicated cases.

An analysis of these waves has been made in each individual.

The *a* Wave.—This wave was normal in 17 patients and larger than normal in the five with associated pulmonary hypertension (Fig. 3). The remaining three cases showed either no *a* wave and presystolic collapse or an insignificant *a* wave.

The *c* Wave.—This was normally registered in all cases.

Systolic Collapse (*x*).—In 17 out of the 20 cases uncomplicated by pulmonary hypertension the systolic collapse was deeper and steeper than normal. This is clearly shown in Case 2 of Fig. 2.

The *v* Wave.—In all the 20 cases without pulmonary hypertension the *v* wave was taller than normal and tended to rise sharply to a peak immediately after the abnormally deep systolic collapse; following pulmonary valve closure (Fig. 2) the steepness of this ascending limb was further accentuated in many cases. The height of this wave has been compared with the *a* wave in these patients; it has been termed "normal" if it was slightly greater than, equal to, or smaller than the *a* wave; "large" if it was one and a half to three times as large as the *a* wave; and "giant" if it was more than three times as large as the *a* wave. By these criteria the *v* wave was giant in seven patients and large in thirteen. In three instances it was eight times the height of the *a* wave.

In all but one of the five patients with pulmonary hypertension the *v* wave was also larger than normal, but this change was less

FIG. 1.—Jugular phlebogram in three normal children. 2 L.I.S.=Second left intercostal space. Pcg=Phonocardiogram. L.F.=Low frequency. M.F.=Medium frequency. H.F.=High frequency. 1=1st heart sound. 2=2nd heart sound.

marked than in the uncomplicated cases. Because the *a* wave was also of increased amplitude in these patients, the height of the *v* wave could not be profitably compared with it.

Diastolic Collapse.—The diastolic collapse was well marked in most patients. In two cases in which no *a* wave was recorded it was continuous with the presystolic collapse.

Discussion

A raised venous pressure with an abnormally large *v* wave has been shown to occur in 20 children with atrial septal defect. This wave is usually sharply defined and can be seen on inspection of the neck veins and identified from the *a* wave by its relationship to the second heart sound. Its differentiation from a large *a* wave helps to distinguish this condition from pulmonary stenosis. We have

Ratio of Height of *v* Wave to *a* Wave in Normal Subjects and in Children with Atrial Septal Defect

Ratio of <i>v</i> Wave to <i>a</i> Wave <i>v</i> : <i>a</i>	Classification of <i>v</i> Wave	Normal Subjects	Atrial Septal Defect (Without Pulmonary Hypertension)
1 : 2	..	5	0
1 : <2		2	0
1 : 1		6	0
1.5 : 1		3	0
1.5-3 : 1	..	0	13
>3 : 1	..	0	7
Total	..	16	20

found a large *a* wave in some mild and moderate cases of the latter as well as in more severe cases; and it is these lesser degrees of pulmonary stenosis that may be confused with atrial septal defect because of the occurrence of a widely split pulmonary second sound in both conditions (Leatham, 1954; Bonham-Carter *et al.*, 1954).

The finding of a large *a* wave in a patient suspected of having an atrial septal defect or the development of such a wave in a known case over the course of time suggests the presence of pulmonary hypertension.

The larger wave following the second heart sound in these patients is probably caused by two separate factors. The deep and steep systolic collapse that is often present is thought to be due to a hyperdynamic right ventricle causing an exaggerated descent of the auriculo-ventricular septum. Following maximal systolic ejection there is a rebound of this septum into the cavity of the right auricle, and the height of the sharply rising wave so produced is then accentuated following pulmonary valve closure by overfilling of the right auricle from the left auricle through the septal defect. Immediately the tricuspid valve opens at the end of the second heart sound there is a sharp fall in pressure in the auricle as blood enters the ventricle, and a steep diastolic collapse wave is produced.

FIG. 2.—Jugular phlebogram in three patients with uncomplicated atrial septal defect. X=Systolic collapse.

FIG. 3.—Jugular phlebogram in two patients with atrial septal defect and pulmonary hypertension, proved by cardiac catheterization.

The large *a* wave preceding the first heart sound in those cases complicated by pulmonary hypertension is probably caused in a similar way to that found in pulmonary stenosis (Laubry and Pezzi, 1913). As a result of peripheral pulmonary vascular resistance right ventricular hypertension occurs, and this then constitutes an obstruction to auricular systolic ejection, which in turn produces increased auricular systolic contraction and a large *a* wave in the venous pulse.

Summary

Inspection of the neck veins in children with atrial septal defect reveals a raised venous pressure and exaggeration of one of the waves of the venous pulse. Simultaneous auscultation identifies this large wave, by its time relationship to the second heart sound, as the *v* wave. When pulmonary hypertension is present in these patients the *a* wave also becomes larger than normal.

Recordings of the venous pulse in 25 patients with atrial septal defect compared with those of 16 normal children have confirmed these findings. They have also shown that there is exaggeration of normal systolic collapse in these patients, and this contributes to the prominence of the large *v* wave.

An attempt has been made to explain the haemodynamics of these changes in the venous pulse.

It is believed that the large *v* wave in atrial septal defect is a useful clinical sign, particularly in differentiating this condition from pulmonary stenosis.

I am indebted to Dr. Alexander Nadas for permission to include eight patients in this investigation who were seen at the Children's Hospital, Boston, Mass., and who have been mentioned in a previous communication (Reinhold and Nadas, 1954). I am also indebted to Dr. R. E. Bonham-Carter for his constant help and encouragement during this investigation and to Drs. J. F. Goodwin, C. H. M. Walker, M. G. Thorne, and J. P. M. Tizard for their constructive criticism of this paper. Mr. G. H. Bryant gave valuable assistance in helping to obtain the recordings.

REFERENCES

- Abrahams, D. G., and Wood, P. (1951). *Brit. Heart J.*, **13**, 519.
 Barber, J. M., Magidson, O., and Wood, P. (1950). *Ibid.*, **12**, 277.
 Bedford, D. E., Papp, C., and Parkinson, J. (1941). *Ibid.*, **3**, 37.
 Bonham-Carter, R. E., Graham, G. R., Reinhold, J. D. L., and Walker, C. H. M. (1954). *Lancet*, **2**, 758.
 Campbell, M. (1954). *Brit. Heart J.*, **16**, 273.
 Laubry, C., and Pezzi, C. (1913). *Arch. Mal. Cœur*, **6**, 433.
 Leatham, A. (1954). *Lancet*, **2**, 607.
 Luisada, A. A. (1948). *Heart*, p. 258. Baltimore.
 Reinhold, J. D. L., and Nadas, A. S. (1954). *Amer. Heart J.*, **47**, 405.
 Wood, P. (1950). *Diseases of the Heart and Circulation*, p. 216. London.

Writing in the *Scientific American* (February, 1955) on "A Study of the Anti-scientific Attitude," Bernard and Judith Mausner (respectively a social psychologist and a physician) ask: "Can scientists draw any lessons from the anti-fluoridation outbreaks?" These writers attribute the strength of the opposition to the fluoridation of water supplies to three main factors. "Firstly, the anti-fluoridation arguments are understandable, easy to follow. Their weaknesses are often difficult for a layman to grasp. Secondly, these arguments are grounded in some of the most widely held ideas and emotions of our culture. In their appeal to respect for individual rights, to fear of poison, to a watchful conservation of public funds, they ring a bell for most people. Thirdly, they are clearly related to the *Zeitgeist*, the current suspicion of scientists, the fear of conspiracy, the tendency to perceive the world as menacing. In contrast, the proponents of fluoridation have all too often ignored public psychology in presenting their case. They have relied too heavily on the fiat of organized science, and have tended to dismiss opponents as 'crackpots' and to deride their arguments. The greatest flaw has been the failure to prepare the public adequately. As a result there develops a polarization of attitudes which makes it very difficult to change opinion."

FRACTURES OF NECK OF FEMUR

INCIDENCE AND IMPLICATIONS

BY

I. M. STEWART, M.B., F.R.C.S.Ed.

Registrar, Eastern Region (Scotland) Orthopaedic Service

This investigation is concerned in assessing current and future problems in the organization of treatment for fractures of the neck of the femur.

In June, 1952, a fracture and orthopaedic unit was opened in the Royal Infirmary, Dundee, to which all fractures in the city and environs are referred. This survey has been made in the belief that the 190 cases encountered in the two years completed are the total incidence and unselected. The technical details of treatment and results have not been considered except in so far as they bear on principles involved.

The series is thought to be unselected for the following reasons: (1) The region is small and the fracture and orthopaedic service complete. Fractures are no longer treated in the general surgical wards of the teaching or other hospitals. (2) All such cases are treated in a 66-bed unit in the teaching hospital, which is associated with a 280-bed country orthopaedic hospital carrying out most of the interval orthopaedic treatment for the region. (3) No recent fracture of the neck of the femur from Dundee or the environs was treated in the country hospital. (4) No case was refused admission for any reason whatsoever. (5) The small number of nursing-home cases has been included.

Classification

Study of the age incidence shows a clearly defined distribution, as is apparent in Table I. It seemed significant that two female patients under 50 had had violent falls at work,

TABLE I.—Pattern of Incidence (Female): City of Dundee Census Area

Age (Years)	Cases, 1952-3	Cases, 1953-4
Under 50	0	0
50-54	1	1
55-59	4	1
60-64	3	3
65-69	7	3
70-74	11	13
75-79	8	10
80-84	10	8
85-89	3	7
90-	1	3
Total	48	49

while none over 50 had. In the men, however, major accidents are equally scattered over the normal life-span. The average age of the male patients is lower, as shown in Table II. The average for the female patients of 75 years is higher than in previous series (Stebbing, 1927, 69 years; Watson-Jones, 1935, 60 years; Murray and Frew, 1949, 62 years). This bears out the unselected nature of the series and, presumably, the significance of the ageing trend of population distribution. The source of the classical fractures of the upper end of the femur is thus determined by age.

TABLE II.—Average Age (190 Cases)

Sex	Fracture Type	Average Age	Cases in 2 Years
Male	Cervical	66.8	14
	Trochanteric	65.5	36
Female	Cervical	75.8	67
"	Trochanteric	75.4	73