Selective Cine-fluoroscopic Studies of Pulmonary Circulatory Disorders

M. J. RAPHAEL AND R. E. STEINER

Department of Diagnostic Radiology, Hammersmith Hospital and Postgraduate Medical School of London, Duane Road, London W.12

This paper describes the results of applying the cine-radiographic technique of Chrispin and Steiner (1964) to the study of various types of pathological pulmonary circulation, secondary to heart disease.

SUBJECTS AND METHODS

The injection and cine-radiographic technique used by Chrispin and Steiner was followed with minor modifications in this investigation. Up to 5 ml. Hypaque 65 per cent were injected at a steady rate through a Courmand end-hole cardiac catheter, usually a No. 7, by the gravity pump of Jacobson (1963). In children we used a lesser quantity, usually 2-3 ml. The period of injection usually lasted about 3 seconds, so as to cover several heart beats, and was timed by an electric clock attached to the pump. The electrocardiogram was recorded during and after the injection period when the contrast was flowing through the pulmonary circulation. Filming was by an Arriflex camera at 48 frames per second through a Philips 9 in. image intensifier. Most of our patients were examined with the breath held in inspiration.

We confined ourselves to injections made with the catheter tip in the "pre-wedge" position (Chrispin and Steiner, 1964). To obtain this position the catheter was usually withdrawn from the wedge position until a pulmonary artery pressure trace could be seen on the monitor. A small test quantity of contrast medium was then injected through the catheter by a three-way tap interposed between pump and catheter. The catheter was considered to be correctly placed in normal patients, and in those with shunts, anticipating a normal or large blood flow, when the test dose was completely washed out of the arteries with each systole. In patients with hypertensive pulmonary vascular disease this criterion was not applicable, as diminished blood flow was to be expected. In these cases the vessel in which the catheter lay was visualized by a test injection to ensure that its diameter was appreciably larger than that of the catheter and that there was no question of

the catheter obstructing flow. The size of the catheter in relation to the enclosing vessel, and the related degree of obstruction to flow, were of crucial importance to the success of the examination. If the catheter tip was "too-wedged", i.e. placed too distally in too small an artery, it caused an appreciable obstruction to blood flow. This produced an artefact, a dense non-fluctuating capillary blush. This "too-wedged" position predisposed to coughing during the examination, a reaction which was prone to occur when relatively large doses of contrast (up to 5 ml.) were injected, particularly in children, even with reduced amounts of contrast. If the catheter was too proximally placed, i.e. in too large a vessel, the contrast medium was excessively diluted. Further difficulties were caused by catheter movement with heart action, so that a good position could never be held, and catheter "whip" occurred during the injection, even with the low injection rate. The right lower lobe was the most common site of injection, the catheter entering it by following its natural curve. Injections in the left lower lobe were abandoned, as the heart shadow obscured the necessary detail. It has not yet been possible to examine upper and lower lobes in the same patient, though this should prove of great interest in patients with mitral valve disease. All the films obtained were analysed frame by frame on a Tage Arno projector and the events were plotted on a time-scale and related, where possible, to the electrocardiogram.

The examinations were carried out in collaboration with the Unit of Clinical Cardiology during routine diagnostic cardiac catheterization. Over 60 patients with either congenital heart disease or acquired valvular lesions were examined, but only 16 films were adjudged suitable for full analysis. Reasons for failure or rejection were varied, but incorrect catheter position and coughing were the commonest. Many earlier examinations of patients with hypertensive pulmonary vascular disease failed because the camera was not run long enough to see the veins opacify, as the extent of slowing of the circulation was not appreciated.

Thus, examinations were rejected for the following reasons: when there was obvious artefact due to the

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catheter being in too small a vessel; when dilution of contrast due to too proximal a catheter position made interpretation impossible; when breathing, coughing, or other movement was so great as to blur the picture; or when the veins were not visualized.

INTERPRETATION OF THE FILM

Only those films in which arterial and venous phases were clearly demonstrable were accepted for analysis. The injection had to cover at least one complete cardiac cycle and preferably more.

Arterial Phase. In normal patients Chrispin and Steiner (1964) described pulsatile onward movement of contrast medium in systole with a filling up of the arterial tree at the catheter tip in diastole. We wished to discover whether this type of pulsatile flow was maintained in abnormal circulatory states. In patients in whom the systolic blood flow was sufficient to wash all the contrast out of the injected vessel, it was possible to see the contrast re-collecting in diastole at the catheter tip. It was possible to count the number of frames during which the contrast medium remained before a further systole washed it away, thus assessing the time scale for this event. It was also possible to note whether the contrast merely collected in diastole or actually continued to move distally. The period during which contrast collected at the catheter tip we have called the period of "diastolic stasis". The completeness with which contrast was cleared from the larger arteries with each systole we have called "pulsation", and this we have estimated as good, impaired, or absent. The number of beats after the end of the injection, before contrast was driven from the smallest arterioles individually identi-

fiable on the film, has been termed the "arterial clearance".

Capillary Phase. We did not find the analysis of the capillary blush of any value as it seemed to depend more on the technique of the examination, as discussed before, than the underlying pathology.

Venous Phase. The interval from the start of the injection to the first appearance of contrast in the veins has been designated the "venous opacification time" recorded in seconds and estimated from the electrocardiogram. The density of the venous opacification is described merely as gross (3), obvious as in the normal patients (2), or just perceptible (1). Fluctuation in density of the veins is classified as follows; definite (2), that is, individual frames can be designated as showing a vein opaque or clear; probable (1), that is, contrast density appearing to wax and wane, as the film is run through. However, it was not easy to designate individual frames as showing opaque or non-opaque veins. The grading of (0) is used when the density does not vary even with the film running.

RESULTS

The results of the analysis in the 16 patients are presented in tabular form (Table I) so that the clinical and catheter findings can be related to the angiographic appearances. The first 3 patients show what we feel are the normal findings, and these are similar to the original description of Chrispin and Steiner (1964). Fig. 1 illustrates the normal appearances.

TABLE I
CLINICAL DATA AND ANGIOGRAPHIC FINDINGS IN 16 PATIENTS

	Clinical data								Angiographic findings					
	Case No.	Diagnosis	Pulse interval (sec.)	Rhythm	Mean pul-mon. artery pressure	Mean pul-mon. wedge pressure	Pul-mon. art-eriol ar resist.	Pul-mon. to sys-tem. shunt ratio	Arterial phase			Venous phase		
									Arterial clearance (beats)	Diastol. stasis (sec.)	Pulsation	Opacification time (sec.) (beats)	Density	Pul-sation
Aortic valve disease	1	AS/AI	0.8	SR	27	18	2	0	1	0.4	Good	2.6	3	2
	2	AS/AI	0.8	SR	38	27	2.4	0	3	0.4	Good	2.7	3	2
	3	AS	1.0	SR	20	10	2	0	2	0.4	Good	2.0	2	2
Moderate mitral valve disease	4	MI	0.8	AF	25	16	3.9	0	2	0.5	Good	4.0	5	1
	5	MI	0.8	AF	37	20	1.5	0	1		Good	2.1	3	2
	6	MI	0.6	SR	45	23	8	0	2	0.25	Good	3.5	6	2
Severe mitral valve disease	7	MS/AI	0.8	AF	22	13	1	0	2	0.4	Good	6.2	8	0
	8	MI	0.4	AF	80	28	8.2	0	3		Good	6.2	10	2
	9	MI	0.8	AF	42	30	5.2	0	5	0.5	Impaired	11.2	17	1
	10	AI/MI	1.0	AF	43	23	5.2	0	7		Absent	22	22	1
	11	MI	0.6	AF	73	28	22	0	4		Impaired	6.5	9	0
Intracardiac shunts	12	VSD	0.4	SR	72		1.7:1	1	0.25	Good	0.9	2	3	0
	13	VSD	0.6	SR	70	14	1.3:1	1		Good	1.1	2	3	0
	14	VSD	1.0	SR	19	11	2.7:1	1	0.25	Good	2.1	2	3	0
	15	ASD	0.8	SR	18		4:1	1	0.25	Good	1.4	2	3	0
	16	ASD	0.6	SR	48	20	2.5:1	1	0.6	Good	1.6	2	3	0

AS, aortic stenosis; AI, aortic incompetence; MI, mitral incompetence; MS, mitral stenosis; VSD, ventricular septal defect; ASD, atrial septal defect; Pulse interval, average interval between heart beats; SR, sinus rhythm; AF, atrial fibrillation.

FIG. 1 (Case 1).—Normal selective angiogram. Injection made into a right lower lobe segmental artery. (A–D) arterial phase; (E–H) venous phase. The accompanying time-scale illustrates the time relationships of the Figs. (A and C) contrast collecting in diastole; (B and D) onward movement in systole; (E and G) venous opacity present (arrow point); (F and H) venous opacity absent (arrow point).

Mitral Valve Disease. Included are 8 patients with acquired valvular lesions in whom the common factor was chronic raised left atrial pressure. Several constant deviations from the normal were noted. Contrast was delayed in the arterial tree. This effect varied from trivial when arterial clearance was hardly delayed from 1–2 systoles, to severe when the delay might reach 7 or more systoles. Fig. 2 illustrates the appearances seen in moderately severe disease. In severe cases the main arterial pulsation was so grossly impaired that contrast was not cleared from the injected artery in systole, giving the impression that pulsatile flow was lost. However, careful analysis shows an advance of the blood contrast border with systole and a re-filling during diastole, and this is shown in Fig. 3.

The venous opacification time was increased, the increase varying from trivial in some cases to over 20 seconds in our most severe case. The veins

when seen were small and usually faintly opacified. Fluctuation in density of the veins, once opaque, might or might not be detectable. The small faint veins seen in severe disease are illustrated in Fig. 2 and 3, as are also the gross pruning of the arterial tree seen in severe pulmonary vascular disease.

Intracardiac Shunts. Five patients with large shunts yielded films that were suitable for analysis. Pulsatile arterial flow was maintained, emptying of the major vessel occurring with great vigour during each systole and the arterial clearance time being only one systole. However, diastolic stasis was noted in 3 patients (one could not be analysed), and in one (Case 15) the diastolic contrast collection was seen to be moving slowly, peripherally, during diastole, but was flushed away vigorously in systole. The venous opacification time was short, the veins

FIG. 2 (Case 9).—Mitral valve disease of moderate severity. Injection made into a right lower lobe segmental artery. (A–D) arterial phase; (E–H) venous phase. The accompanying time-scale illustrates the time relationships of the Figs. (A and C) contrast collecting in diastole; (B and D) onward movement in systole; (E and G) small opaque vein (arrow point); showing minimal fluctuation as compared to F and H. Frames marked with an asterisk are counted from the restart of the camera during the interval.

appearing after 2 or 3 systoles, and the veins were large and dense. The rhythmic density fluctuations of the veins were strikingly absent. Fig. 4 and 5 illustrate the typical appearances in this type of case. The results of the 16 films analysed are summarized in Table II.

DISCUSSION

We feel that the normal onward blood flow with each systole is sufficient to expel contrast from the major segmental and sub-segmental arteries, and that the onward blood flow from the first and second systole following the end of the injection

TABLE II
SUMMARY OF ANALYSES

Diagnosis	No. of cases	Arterial pulsation	Arterial clearance in pulse beats	Venous opacification time in pulse beats	Venous pulsation
Normals	3	Good	2–3	2–3	Good
Mitral disease {	Mild 4	Good	2	3–8	Impaired
	Severe 4	Impaired or absent	3–7	9–22	Impaired, absent
Intracardiac shunts	5	Good	1	2	Absent

FIG. 3 (Case 10).—Severe mitral valve disease. Injection made into the right lower lobe segmental artery. (A–D) arterial phase; (E–H) venous phase. The accompanying time-scale illustrates the time relationships of the Figs. (A and C) diastolic filling phase; (B and D) systolic injection phase. Note the up and down movement of the contrast meniscus (arrowed) with each diastole and the very inadequate movement of contrast in systole. Marked “pruning” of the arterial tree is clearly shown. (E–H) very thin poorly opacified pulmonary veins (one of which is arrowed) show no convincing evidence of density fluctuation. Frames marked with an asterisk are counted from the restart of the camera during the interval.

should be sufficient to empty contrast from even the smaller arteries, i.e. those large enough to be identified on our films. In addition in the normal subject, contrast in the veins should be identifiable within 2 to 3 systoles of the start of the injection. Any lengthening of these time intervals must represent a reduction in systolic flow, and we feel

that the extent of prolongation is related to the reduction in flow. Conversely increased flow will be reflected by more vigorous arterial pulsation and more rapid opacification of the veins.

Mitral Valve Disease. Patients with chronic raised left atrial pressure have reduced calibre of

FIG. 4 (Case 14).—Ventricular septal defect. Injection into a right lower lobe segmental artery. (A-D) arterial phase; (E-H) venous phase. The accompanying time-scale illustrates the time relationships of the Figs. (A and C) contrast collecting in diastole; (B and D) onward movement in systole; (E-H) enlarged dense, non-fluctuating vein (arrowed). Frames selected to cover the cardiac cycle.

the lower lobe pulmonary veins (Doyle *et al.*, 1957). This change is associated with a redistribution of lung blood flow seen typically in patients with mitral valve disease (Dollery and West, 1960). The phenomena we have observed during selective angiography of the lower lobe vessels of patients with chronic raised left atrial pressure are a reflection of the alterations in blood flow. Minor, but appreciable, deviations from the normal are

detectable in patients with relatively mild valvular lesions. This suggests that the redistribution of blood flow occurs at an early stage of left heart disease, long before changes in measured pulmonary arterial resistance occur. Our selective studies do show that the perfusion of the upper lobes at the expense of the lower lobes, which is such a feature of chronic raised left atrial pressure, can progress to such an extent that the lower lobe blood flow can

FIG. 5 (Case 16).—Atrial septal defect. Injection made into the right lower lobe segmental artery. (A–D) arterial phase; (E–H) venous phase. The accompanying time-scale illustrates the time relationships of the Figs. (A and B) contrast collecting in diastole; (C and D) onward movement in systole; (E–H) dense non-fluctuating veins, one of which is arrowed. Frames selected to cover the cardiac cycle. The arterial injection covers only one cycle.

make no appreciable contribution to pulmonary blood flow. In Fig. 3 the catheter lies in a relatively large artery, probably a segmental artery: it delivers approximately 1.2 ml. of contrast per second, which is washed away at approximately the rate that it is delivered, as the blood contrast border does not advance appreciably during the injection. Assuming that there are four such arteries on the right and three on the left, all having equal blood flow, the perfusion to the basal segments of the lungs will be about 10 ml. or just about one-sixth of the stroke volume of 63 ml.

Venous opacification was very poor in severe cases, and this was probably due to the fact that the veins were very small. In these poorly opacified veins it was very difficult to be certain of definite rhythmic fluctuation of density, which is typical of the pulsatile flow dilution effect of Chrispin and Steiner (1964). It is likely that only the low blood

flow, which produced much less over-all dilution of the contrast medium in its passage through the lungs, enabled us to detect the venous phase at all. We were unable to relate the presence or absence of venous pulsation to such factors as the severity or precise nature of the heart disease, or the presence or absence of atrial fibrillation, or congestive cardiac failure.

Intracardiac Shunts. In patients with shunts, the persistence of the period of complete diastolic stasis suggests that there is no forward blood flow in diastole even in the smaller arteries and even with the relatively large shunts. In one patient with a large shunt (Case 15), the contrast could be seen to move slowly peripherally in diastole, with the usual spurt forward in systole. We feel that this is fair evidence that in patients with fairly large shunts pulmonary artery flow remains com-

pletely pulsatile in nature with no continuous diastolic component. Only when the shunt is very large does forward flow occur in diastole.

In the venous phase the rapid appearance of contrast in the veins is what might be expected in patients with high blood flow in the lungs. The intense opacification of the veins, which is a feature of patients with left-to-right shunts, is rather unexpected as the high flow should produce dilution of the contrast medium. The dense opacification is probably related to the disproportionate increase in the size of the veins, which increases their radio-density even if the contrast within them is diluted.

The loss of perceptible venous opacification is even more paradoxical and is also probably related to the increase in size of the veins. Our studies on the arteries confirm that flow remains purely pulsatile without a continuous component in shunts, unless they are very large. This suggests that venous pulsation, if related in any way to right ventricular systole, should become more obvious. However, it seems likely that the enlarged veins with their increased capacity act as a volume damping system for the blood pulsating in from the capillaries, and by the time contrast carrying blood reaches the segmental veins pulsatile flow is entirely damped out. The flow pattern in the pulmonary veins can be studied by direct counter-current injections into the veins themselves by catheters passed through an atrial septal defect into them. We have as yet insufficient data for analysis.

SUMMARY

Studies were made on over 60 patients by selective pulmonary cine-angiography, and of these 16 were selected for detailed analysis. Three patients with normal right heart pressures and no intracardiac shunts were considered to have normal appearances;

8 patients with varying degrees of pulmonary hypertension secondary to acquired valvular heart disease, and 5 patients with intracardiac shunts of varying sizes, were considered to have abnormal appearances. In patients with raised left atrial pressures and varying degrees of pulmonary hypertension, the reduced lower lobe blood flow was manifested by impaired onward movement of the contrast medium with each heart beat and delayed appearance of contrast in the pulmonary veins which showed very impaired pulsation.

In patients with a large left-to-right shunt the onward movement of contrast medium into the pulmonary arteries with each heart beat was accentuated. Unless the shunt was very large a period of diastolic stasis of contrast medium was maintained as in normal patients. This suggested that purely pulsatile arterial flow was still maintained, even in the presence of large shunts. Contrast medium rapidly reached the enlarged veins which failed to show pulsatile fluctuations in density, thus suggesting absence of pulsatile venous flow. This might be explained by the associated venous enlargement producing a volume damping effect.

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