LEFT VENTRICULAR FAILURE.

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Congestive failure is brought about by inability from various causes to maintain a satisfactory circulation, resulting in vascular stasis.

Myocardial weakness often occurs long before failure is noted. The condition may come on suddenly or gradually, the strain being usually unilateral—one ventricle (commonly the left) failing before the right. Normally the left ventricle has to bear a considerably greater strain than does the right, especially in those past middle age.

Left ventricular failure occurs as a primary manifestation of myocardial inefficiency at least three times more often than does failure of the right ventricle. The factors causing it are commonly hyperpiesia, aortic valve incompetence, and myocardial infarction, whereas those commonly causing right ventricular failure are mitral stenosis, pulmonary fibrosis, and certain congenital heart lesions. It is, however, an interesting point that by far the commonest cause of right ventricular failure is the result of back pressure from a failing left ventricle. (Thompson & White, 1936.)⁽¹⁾

Hope,⁽²⁾ in 1833, was the first to point out the effect on the lungs of left ventricular failure.

The theory of back pressure, which held sway for many years as the cause of heart failure and was later discarded for diminished *vis a tergo* (force from behind), has now come into favour again.

Ætiology.

As already stated the chief factors of left ventricular failure are chronic hypertension, aortic valve disease and myocardial infarction. Less common factors are severe rheumatic carditis, thyrotoxicosis, congenital defects, anæmia, and abnormally rapid heart rates. Rarely arteriovenous aneurysm, cardiac trauma, thoracic deformities, pericardial adhesions, and tumours may be the causal factors.

Acute failure is relatively rare, but may be met with in certain febrile illnesses such as diphtheria, acute rheumatism or pneumonia, in some cases of coronary thrombosis, and in certain cases of prolonged paroxysmal tachycardia.

Usually heart failure is of gradual onset and it is therefore of the greatest importance to recognise the early signs so that suitable measures of treatment may be started without delay. Obviously the earlier such a patient comes under treatment the better will be his chance of avoiding, or at any rate retarding, the more advanced stages of failure.

Heart failure occurs more frequently and at an earlier age in males. The condition, however, is commonly met with in old age. Seventy-five per cent. of cases occur after 50 years of age.

In cases with heart disease failure may be precipitated by relatively trivial circumstances such as slight respiratory infection, abdominal distension, excitement or slight over-exertion. The onset is, however, usually insidious and only when failure is moderately advanced does the patient come under medical care.

Pathology.

There are few pathological changes characteristic of congestive failure. The condition is a functional one, almost invariably associated with organic heart disease. A normal heart may fail if the strain is sufficient, but this is extremely rare.

Failure is usually the result of mechanical strain, as in marked hypertension. It may be brought about by inflammation of the myocardium, as in severe rheumatic infection, or by severe anæmia. Commonly there is cardiac enlargement consisting usually of both hypertrophy and dilatation, and rarely of dilatation alone. The latter only occurs if the failure is acute and rapid.

If left ventricular failure results from chronic hypertension, dilatation takes place in an already hypertrophied left ventricle, the mitral valve may become incompetent, the left auricle over-filled, the pulmonary arterial pressure increased with engorged pulmonary vessels, and finally strain on the right heart and great veins may occur.

Aortic disease and coronary disease may be found together with generalised arteriosclerosis, and atheroma of the aorta may be present.

Clawson,⁽³⁾ in 1924, from post-mortem study of 102 cases of myocardial failure, noted severe coronary sclerosis in 22.5 per cent.

Syphilitic myocarditis is extremely rare, and the myocardial changes met with in cardiovascular syphilis are the result of ischæmia from narrowing of the coronary orifices.

About half the deaths from myocardial failure show no pathological changes in the heart muscle.

Symptoms.

The earliest symptom is dyspnœa, which at first may be only apparent on moderate exertion. Its significance may be completely missed by the patient, who attributes it to over-work, want of a holiday, or increasing years. Later, even slight exertion such as walking up a mild incline or hurrying, especially when the weather is cold or soon after a meal, will bring on shortness of breath, and the patient finds it expedient to walk more slowly and perhaps stop for short rests on hills. Excitement may also cause dyspnœa. Finally dyspnœa is present even when at rest.

In the recumbent posture the heart work is often greater than in the upright position owing to removal of the effect of gravity, whereby the stomach and abdominal organs tend to rise up and press on the heart. This is frequently the case in those who are over-weight and have large abdomens, and is therefore most commonly found amongst those of sedentary habits. It is also for this reason that dyspnœa is often most marked during the night.

A typical form of paroxysmal dyspnœa of sudden onset, occurring during the night and sometimes awaking the patient from sleep, is known as cardiac asthma. The dyspnœa is sometimes extreme, the chest is tight, the lungs full of rhonchi, and the patient deeply cyanosed and fighting for breath. The paroxysm may last from a few minutes to several hours, and ends by the expectoration of a small amount of viscid tenacious sputum. The patient is extremely exhausted by the attack, which may prove fatal. Cardiac asthma is a sign of advanced left ventricular failure. Its presence is often associated with an impaired renal function.

A failing left ventricle causes increased pressure of the pulmonary circulation with consequent congestion. Thus cough and expectoration are almost inevitable occurrences. At first there is an increased tendency to attacks of bronchitis, which later on becomes chronic with cough and muco-purulent expectoration. As the pulmonary circulation deteriorates the lungs become more congested and there is an increasing oedema, at first at the bases and later involving more and more of the lung tissues.

With the incidence of oedema the general condition of the patient has further deteriorated, and general weakness tends to prevent adequate elimination of sputum. There is usually a useless cough which still further exhausts the patient and prevents sleep. The breathing becomes more difficult and laboured and the patient can only breathe sitting upright in bed.

By this time signs of right heart failure have become evident, namely a large, tender, congested liver, increasing flatulence and constipation due to portal obstruction, a diminished urinary output, and soon after oedema of the lower extremities gradually rising upwards to involve the abdominal wall and lumbar region.

Ascites is usually present. It generally occurs as a late manifestation of right heart failure, but in cases of mitral stenosis tends to precede oedema of the lower limbs.

Acute pulmonary oedema is a condition somewhat similar to cardiac asthma. This condition may come on acutely and in a few moments the patient is in a hazardous state, intensely cyanosed, bringing up large quantities of watery, blood-stained sputum, with profuse sweating and rapid and imperceptible pulse. The condition is often fatal but sometimes prompt measures may pull the patient through.

Disturbances of respiratory rhythm may occur, the most important of which is Cheyne Stokes' breathing. In this there are alternate phases of over-breathing and apnoea—the respirations after a period of apnoea gradually increasing up to a summit and then gradually fading away up to the next apnoeic period. The condition is usually noted during sleep, especially if morphia has been given. It may, however, occur during the daytime, when it is a very grave prognostic sign.

Pain is not common in left ventricular failure in the substernal and præcordial regions, but in nervous types there may be an almost constant præcordial ache.

Other symptoms are weakness, which is usually complained of, insomnia, which is especially common in hypertension cases, and headache, which is of frequent occurrence.

The majority of patients are nervous about their condition and are easily worried and inclined to irritability.

Mental disturbances are occasionally met with. They may be merely impaired memory, especially for recent events, or may culminate in mental hebetude with hallucinations and dementia.

Palpitations may be a symptom where there are complicating disturbances of rhythm.

Signs.

There is usually evidence of cardiac enlargement. If left ventricular hypertrophy is marked there is a heaving impulse, but with dilatation the impulse is either absent, feeble or diffuse.

Organic or functional murmurs are often found in the mitral area. The quality of the heart sounds is altered. There may be a tic tac rhythm or a proto-diastolic rhythm. This latter is often found in association with a bundle branch lesion. Arrhythmia due to multiple premature contractions or to auricular fibrillation is present in 50 per cent. of cases. Bradycardia is occasionally found, either of physiological type or due to auriculo-ventricular block.

The blood pressure shows great variations. Thus the pressure may be extremely high, as in cases with hypertension and in aortic disease, or it may be very low as in most cases with recent cardiac infarction. A sudden big fall in the blood pressure is especially significant of cardiac weakness and is a common occurrence in cases of acute coronary occlusion, especially of the main descending branch of the left coronary artery.

Pulsus alternans is another important sign of left ventricular failure. It may occur relatively early, but its presence may be missed. It is usually discovered in the course of taking the blood pressure. The condition may occur for a few beats only, usually following a premature beat, or may be constant as long as the state of failure lasts.

The earliest signs of failure are found in the lungs. There is diminished vital capacity as shown by the spirometer, and this instrument should be used in all cases whose condition does not prohibit the test, and it is an extremely valuable aid in assessing the progress of the case under treatment.

Increased pulmonary work results in emphysema and bronchitis. At first there are transient râles at the bases, which later become constant, and then râles throughout. With the advent of oedema of the bases weak breath sounds with fine râles are heard. Later hydrothorax occurs. This usually is noted first on the right side, but later both sides may become involved. By this stage there is usually evidence of right ventricular failure, as shown by jugular engorgement, enlarged congested liver, ascites, or oedema of legs. The excretion of the kidneys is lessened in the later stages and albumen and casts are present. Biochemical tests show evidence of impaired kidney function.

Investigations.

X-ray examination is of definite help in certain cases of left ventricular failure. There can be noted the size and shape of the heart and great vessels, the strength or weakness of the cardiac impulse, the presence or absence of increased hilar shadows due to congested pulmonary vessels, and the condition of the lung bases.

Electrocardiography has proved of great value in elucidating the type of arrhythmia and the state of the myocardium. The electrocardiogram has proved of the greatest importance in the diagnosis of acute coronary occlusion, as not only does the record reveal its occurrence but also denotes accurately the site and extent of the infarct.

The basal metabolic rate is usually raised, sometimes as much as plus 50 per cent.

The blood flow is often decreased and the blood thick and sticky.

Exercise tests are of limited value. A more accurate estimation of the heart's function can be gauged from the history of limited effort.

Prognosis.

Left ventricular failure is usually of gradual progress and much can be done by restricting the heart's activities in the early stages.

Dyspnoea at rest, especially the occurrence of attacks of cardiac asthma, is of serious import.

Acute pulmonary oedema is one of the common causes of death in left ventricular failure, and the majority of patients succumb to an attack.

Marked electrocardiographic abnormalities, especially those showing damage to the conductive tissues, are serious to life, and it is unlikely that such a case will survive more than two or three years, if so long. The advent of right ventricular failure is the beginning of the end, though adequate treatment may still bring about a partial recovery.

Syphilitic aortitis with failure rarely lasts more than a year or two.

Severe renal disease is a grave complication, as many of the methods of relieving the circulation depend on the integrity of the kidney function.

The presence of retinal papilloedema in cases of hyperpiesia with impaired renal function is an extremely grave prognostic sign and death occurs in a few months or less.

An important prognostic point is the ability and willingness to carry out the prescribed treatment. This, however, may be impossible upon financial grounds.

Treatment.

Treatment of left ventricular failure depends on the gravity of the condition. By far the most important factor is rest. This may mean simply some curtailment of activities to absolute rest in bed. In early cases restricting the more strenuous forms of exercise and taking up easier forms such as walking, mild golf, croquet, etc., may be sufficient. Later on avoidance of all physical effort such as hurrying, going up stairs or hills, or lifting, may be necessary.

There is a definite association between physical and mental fatigue, and for this reason curtailment of business hours may be indicated. Often for those patients whose big financial interests have entailed over-work and constant worry, a three months' holiday (such as a sea voyage) has a remarkably beneficial effect, and in my experience has to some given a new lease of life.

For those cases whose failure entails confinement to bed, the most comfortable posture is allowed. Most patients prefer being propped up in the semi-upright position, and in this connection a special heart bed or an arm chair may be preferred.

In severe cases of left ventricular failure, as after an acute coronary occlusion, absolute rest for six weeks is necessary, and during this time the patient is not allowed to make any movement whatever—not even to move himself in bed or to feed himself. Excitement and emotion increase the work of the heart and these must be avoided as far as possible.

Dieting is an important adjunct in the treatment of these cases. Flatulence is usually present and is sometimes a very disturbing factor, and therefore small, dry meals are given and fried foods, cooked fats, potatoes and all rich foods are forbidden. In patients with hypertension red meat and meat extracts are not

allowed. In all forms of left ventricular failure it has been my practice to cut out salt entirely from the diet, allowing in lieu Heudebert's synthetic salt, "Selarom" (Bayer), or "Ruthmol". On the other hand I encourage the consumption of plenty of bland fluid—either lemonade, barley water sweetened with glucose or one of the alkaline spa waters such as Vichy, Contrexeville, etc., even in patients with oedema.

In all patients with impaired left ventricular function a short period of rest after meals, especially after lunch, is an essential part of the ritual. In those patients who cannot give up work on financial grounds I advise one rest day in bed each week.

Drugs. The digitalis group of drugs is indicated in all cases of left ventricular failure, whether the rhythm is regular or not and whether or not there is oedema.

In auricular fibrillation the amount of the drug required is that adequate to control the ventricular rate. The optimum rate is that in which all beats reach the radial pulse and the patient feels at his best. It is usually between 60 and 80.

In patients with regular tachycardia and signs of failure digitalis is indicated, though its efficacy is much less than in auricular fibrillation—the equivalent of 20 to 30 minims of the tincture daily usually suffices.

Occasionally in cases of acute failure digitalisation has to be rapid, as for instance acute pulmonary oedema or the incidence of auricular fibrillation with severe failure. In these cases oral digitalis is often unsatisfactory and may cause nausea, and in my experience the best preparation is strophanthin, gr. 1/100 intravenously, followed by strophanthin, gr. 1/200, twice daily until the ventricular rate has dropped to 90, when oral digitalis is substituted.

The use of diuretics is advisable in all cases where there is fluid hold-up in the tissues. The mercury compounds are the most efficacious and I have used Salyrgan, 2 c.c. intramuscularly every third day, for years. I usually give at the same time urea, 15 grammes tds. The use of diuretics largely depends on the functional integrity of the kidneys.

Sedative drugs are most valuable since these patients are usually excitable. Bromides and luminal are those most used, but where a strong sedative is necessary morphia should be given in adequate dosage. Heart cases stand morphia extremely well and it is the only drug of real value in acute coronary occlusion and in acute pulmonary oedema, where it can be given in large doses, gr. 1/4 to gr. 1/2.

Aperients are given as required, and unless contra-indicated I usually allow the patient to take the aperient he is used to.

Venesection and leeching are in my experience two of the most valuable measures in the treatment of serious cases of left ventricular failure. Venesection I do as a routine in the following cases:—acute pulmonary oedema, severe cases of acute coronary occlusion, cardiac asthma, extreme hypertension, and Cheyne Stokes' breathing when prolonged and causing distress, in conjunction with oral Euphyllin. Certainly some of the most dramatic recoveries in my experience have followed removal of 15 to 20 ozs. of blood.

I use leeches in cases of congestive failure with an enlarged congested liver and ascites—applying six to twelve leeches over the liver area. In my experience, after leeching, oral digitalis can be well tolerated in almost every case. Leeches are also of value for the relief of pain in acute pericarditis, whether due to acute rheumatism or to cardiac infarction.

Oxygen should be given intranasally by Marriott's inhaler in all cases with impaired respiratory exchange.

Eradication of septic foci where possible should be done—notably of the teeth.

Specific treatment of the causative diseases will of course be given—namely anti-syphilitic measures for cardio-vascular syphilis, and thyroid medication for myxoedema.

Inhalation treatment for bronchitis with pulmonary congestion is sometimes of the greatest value in combination with expectorants. I have had extremely good results from Collison's Apneu inhaler.

Physiotherapy has played an important rôle in the treatment of the earlier cases of left ventricular failure. Bad Nauheim, Royat, and more recently Leamington, have gained a well deserved reputation in the treatment of these cases. Carbon dioxide baths in conjunction with massage and exercises are main essentials of the treatment at these spas. Recently short wave diathermy has been tried with promising results.

Surgical Treatment. Surgical treatment of cardiovascular disease is of relatively recent date, but already evidence is accumulating of its increasing value.

Complete thyroidectomy has been performed for patients with angina and for those with congestive heart failure, on the grounds that reduction of the basal metabolism would ease the work of the heart. The results on the whole have been disappointing, since the improvement has been only temporary.

Removal of the stellate ganglion with the pressor nerves to the heart has been performed for several years for cases of angina, but the results have been uncertain and the operation is rarely performed nowadays.

Cardiomentopexy has been performed by Sauerbrunch in Berlin and by O'Shaughnessy in London in the last two years in cases of left ventricular failure, and the results are extremely promising. This operation certainly seems to offer a real hope that many cases of cardiovascular disease with serious functional disability will be able to resume their former activities in greater or less degree, whereas formerly they had little hope of any marked amelioration of their condition.

In the last few years certain cases of left ventricular failure have been noted as being due to hyperthyroidism. The signs of hyperthyroidism are often difficult to recognise, and the condition has come to be known as masked hyperthyroidism. The signs usually found are tachycardia, arrhythmias—either multiple premature contractions or auricular fibrillation—moist skin, loss of weight and mental excitability. The usual methods of treating this type of left ventricular failure are often disappointing and the only treatment likely to prove efficacious is partial thyroidectomy.

The following cases demonstrate some of the types of left ventricular failure mentioned:—

Case 1. I was called about midnight on October 18th, 1936, to see an elderly lady aged 74 who was having a severe attack of cardiac asthma. She was very cyanosed and distressed and her respirations were shallow and forty per minute. She was sitting

propped up in bed and perspiring freely. The heart action was 100 regular and the blood pressure 170/100. The attack had then continued for an hour and she had been given an injection of Coramine, 1 c.c. The heart sounds were inaudible owing to the presence of rhonchi all over.

She was given an intravenous injection of Perphyllon (Homburg) 2 c.c. slowly, and within five minutes the breathing became easier. Later she was given another hypodermic injection of morphia, gr. $\frac{1}{4}$, intravenously, and she had a good night's rest. She was put on a dry, anti-flatulent diet. She made a satisfactory recovery.

I was called in again on November 9th, 1937. She had had a severe hæmatemesis in August, 1937, from which she gradually recovered—the cause of this was not investigated. On the morning of my visit she had had another attack of cardiac asthma which gradually passed off after an injection of Cardiazol-Ephedrine.

On examination the heart apex was 5 in. from the midline. There was a mitral systolic murmur. The lungs were full of râles and rhonchi with crepitations at the right base. The abdomen was large and flatulent with a palpable liver but no ascites. The blood pressure was 160/100 and the apical rate 146 with a radial deficit of 14. The electrocardiogram showed auricular fibrillation with uncontrolled ventricular rate.

Six leeches were put over the liver and she was given strophanthin, gr. 1/200 intravenously twice daily for three days, then the drug was stopped and the ventricular rate controlled by Nativelle's digitalin granules, gr. 1/600, three times daily, which she is still continuing. She was also given inhalations through an Apneu apparatus. Since that time she has kept fairly well though she has to lead a very quiet existence.

Case 2. A man of 64 had cardiovascular sclerosis with raised blood pressure for several years. He came to me in October, 1931, with uncontrolled auricular fibrillation, a blood pressure of 190/130, and an apical rate of 136. He did fairly well on the treatment (rest, digitalis, diet, etc.) up to the beginning of 1933 when he had an influenzal attack followed by broncho-pneumonia with swinging temperature between 100 and 102. Though he was extremely ill at the time he gradually recovered.

In June, 1933, he came to me after three weeks' holiday at Torquay. He made a good recovery. The blood pressure was 200/130 and the ventricular rate 84. He continued in good health until March 6th, 1935, when I was called to him urgently one night and found him in extremis in an attack of acute pulmonary oedema. He was given straight away strophanthin, gr. 1/100 intravenously, and 20 ounces of blood was drawn off from a vein. During this time he was kept warm with numerous hot bottles in the bed, and given continuous intranasal oxygen. At the end of the venesection he was given morphia, gr. $\frac{1}{4}$. By the time I left his pulse was countable though feeble and irregular—100 per minute. He felt very much better and gradually regained his normal level. He kept in fair health for eight months and then had another attack in which the same treatment was again successful. He came to see me recently and was keeping well for him and had had no further attacks. His electrocardiogram showed auricular fibrillation with intra-ventricular block.

Case 3. A timber merchant, aged 63, was brought to see me on February 24th, 1937, suffering from marked dyspnoea and exhaustion. He was having attacks of paroxysmal dyspnoea at night which prevented sleep.

On examination he was sparely built with a pale complexion and cyanosed lips. He was dyspnoeic at rest. The heart was considerably enlarged and there was a marked gallop rhythm over the mitral area. On screening the heart was considerably enlarged, the heart shadow reaching as far as the thoracic border; the aorta

was wide and there was a considerable hydrothorax on the left side. The urine contained albumen. The blood pressure was 190/150, the pulse 116 regular and small, and the retinal vessels showed marked arterio-sclerosis. The electrocardiogram showed a left axis deviation, notched QRS complexes, inversion of Tii, and Tiii of hump-backed character. The record suggested a recent posterior infarct.

He was put to bed, given an anti-flatulent, salt-free diet, though fluids were not curtailed, and given digitalis, Salyrgan and sedatives. He improved steadily. The gallop rhythm disappeared and the left hydrothorax diminished.

On March 5th the blood pressure was 195/145 and the pulse 82 regular. On March 8th he complained of a sharp pain over the right lower ribs posteriorly. No friction was heard but the right base gradually became dull and the breath sounds weaker. The blood count done at this time was normal as was also the blood urea, which was 34 mgm. per cent. His general condition improved during his seven weeks in bed.

On April 14th the blood pressure was 175/120 and the pulse 96 regular. The right base was dull and screening showed a large right hydrothorax. He was not breathless at rest. He was sent to Torquay where he stayed for ten weeks—resting most of the day for the first few weeks and then gradually getting up and about.

I saw him on his return on June 22nd, when I found his general condition considerably improved. He stated he could walk uphill gently without symptoms. The blood pressure was 222/130 and the pulse 85 regular. The heart was still very large, but there was no longer a gallop rhythm. On screening both lung fields lit up normally.

The electrocardiogram shows no axis deviation, a regular rhythm of 66, T1 positive and of low voltage beginning below the iso-electric level, and T2 and T3 hump-backed and beginning well above the iso-electric level. R2 has apical notching and Q2 and Q3 are large and notched. The record indicates gross myocardial damage and is suggestive of a posterior infarct.

Various electrocardiograms were taken between 24/2/37 and 21/3/37 and remained unaltered in spite of various changes in his general health. No record was taken during his terminal illness.

I saw him again in September when his condition was quite satisfactory.

In October he noticed slight weakness of the left leg and left arm. The oscillometric readings of the lower limbs were satisfactory. The left side paresis gradually disappeared and he kept well, except for shortness of breath on slight exertion, in spite of going to business five times a week.

On February 2nd this year he had a sudden severe pain across the chest. I saw him at his home about three hours after, when he had already been given morphia, gr. $\frac{1}{4}$. His colour was very pale, the blood pressure 140/85, and the radial pulse 27—the apical rate being 54, due to alternating premature beats. Morphia gr. $\frac{1}{4}$ was repeated, and in half an hour the rate was 60 regular with no pulse deficit.

He had a bad night with frequent vomiting, and the following morning ten ounces of blood was drawn off and he was put on rectal salines and fluids *ad lib*. At that time he was having Cheyne Stokes' breathing which was relieved by Euphyllin tablets. He was also put on continuous intranasal oxygen and given inhalations of oxygen with 5 per cent. carbon dioxide for the Cheyne Stokes' dyspnoea. The blood pressure was 116/86 and the pulse was 72 regular. The following day his condition had improved. His colour was much more normal and the heart impulse stronger.

On the 5th there were the following complications: The lungs contained numerous coarse râles all over and there were signs of congestion at the left base. The respiration rose to 36. The blood pressure was 118/86.

On the 7th the lungs had improved and he was more comfortable. The blood pressure, however, was lower—108/70, the respirations 26, and the pulse 64 regular. He had not been sleeping well with various mild hypnotics and it was decided to give him morphia, gr. $\frac{1}{4}$, which gave him a good night.

On the 9th the lungs were quite free from adventitious sounds and his breathing was normal. The heart, by then, however, was irregular due to premature contractions, and the apical rate had risen to 116. The blood pressure was 130/80. He was given strophanthin, gr. 1/200, and later was put on Nativelle's digitalin, gr. 1/600, three times daily, and Coramine injections six-hourly.

On the 10th he was much better and particularly cheerful, but died suddenly the same evening from a pulmonary embolism.

This case shows several points of interest, namely marked left ventricular failure complicated by hydrothorax on two separate occasions; what must have been a small thrombosis causing temporary paresis of the left side; and finally a large thrombosis of the left coronary artery causing even graver signs of ventricular failure—Cheyne Stokes' breathing—and ultimately proving fatal through a pulmonary embolus.

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