Probably pulmonary regurgitation from this cause would be more frequent than it is were it not for the safety-valve action of the right ventricle, and its occasional occurrence was pointed out by Dr. Fothergill.¹ I have little doubt that it was present in the following case.

CASE I. Mitral Regurgitation; Obstructive and Regurgitant Murmurs in Pulmonary Area.—M. M., aged 36, housewife, admitted into the Royal Infirmary under my care in the absence of Dr. Davidson, May 15th, 1893, with dropsy in legs and abdomen. Her illness began eight months before with palpitation and dyspnoea on exertion, followed by swelling in the legs. She denied having ever had rheumatism. When first admitted there was great abdominal distension, which was relieved by successive tapplings, and the pulse was quick; but the general condition improved greatly in two or three weeks, and I was able to note carefully and repeatedly the exact conditions of the heart. The pulse was regular in time and force and of moderate rapidity, and was not in the least suggestive of aortic disease. The impulse was in the sixth left intercostal space, $5\frac{1}{2}$ inches from the middle line and $1\frac{1}{2}$ outside the mammillary line. It was regular and strong and no thrill was felt. The second sound was reduplicated, and was loudly accentuated at the pulmonary cartilage. In the mitral area there was a loud systolic murmur, conducted towards the left axilla, and heard at the back. There was also a loud systolic murmur heard at the right border of the sternum at the base of the ensiform cartilage, apparently due to tricuspid regurgitation. In the pulmonary region two murmurs were heard, the one systolic, the other diastolic. The systolic murmur was loud, had the rough quality of a direct murmur, and was conducted towards the left clavicle; the diastolic murmur was soft; it began with or directly after the second sound, was heard best in the second left intercostal space close to the sternum, and was also audible in the third space and faintly in the first. In the aortic area a much fainter systolic murmur was heard, probably radiated from the left side; but no diastolic murmur was present, nor was one heard at the lower end of the sternum nor in the carotid artery.

Primary endocarditis is as rare on the right side of the heart as it is common on the left, judging from the extreme infrequency with which signs of inflammation, either old or recent, are found in *post-mortem* examinations in this situation. I once found some vegetations, probably recent, about the size of a split pea, on the margin of the tricuspid valve, when I was making a *post-mortem* examination on the body of a patient who died in the Stanley Hospital some years ago; but I have unfortunately not been able to find the notes of the case among the records at the hospital. The following case, treated in the Royal Infirmary, seems to have been one of rheumatic endocarditis affecting the tricuspid valve.

CASE II. Antecedent Rheumatism; Loud Systolic Murmur in Tricuspid Area.—W. J. R., aged 17, errand boy, admitted under my care, in the absence of Dr. Davidson, June 15th, 1893, suffering from some functional nervous disorder, apparently independent of the condition I am about to describe. He stated that he suffered from shortness of breath and swelling of the legs, and that when 11 years old he was treated in the Children's Infirmary for rheumatism and heart disease. While he was under my care a systolic murmur was audible all over the precordium, and was very loud in the tricuspid area. Its point of maximum intensity was at the left border of the sternum at the level of the fourth intercostal space, and it could be heard to the right of the sternum. In passing from the tricuspid to the mitral area it diminished in intensity, and although it was loud in the former situation it was quite inaudible at the back.

In the foregoing instances the murmurs pointed with tolerable clearness to organic disease on the right side of the heart; but it need hardly be said that murmurs in the pulmonary region generally admit of a different interpretation. Not to speak of hæmic murmurs which are always systolic and are hardly likely to be misinterpreted, we sometimes hear a diastolic murmur in this situation, while yet the associated conditions make us hesitate to affirm that pulmonary regurgitation is present. In the following case the interpretation of such a murmur presented no small difficulty.

CASE III. Mitral Stenosis and Regurgitation; Diastolic Murmur in Pulmonary Area probably Aortic in Origin.—F. J., aged 16, confectioner, was admitted under Dr. Caton on June 3rd, 1893. She had had rheumatic fever two years, and a second attack seven months, before admission. The heart was enlarged, its action tumultuous, pulse 96; there was a presystolic thrill at the apex, and a diastolic shock at the second left cartilage. At the apex we heard a long rough presystolic, and a systolic murmur, which together occupied nearly the whole cardiac cycle. In the second left interspace a *brui* was heard with the second sound, having the peculiarity that it lessened in intensity, or even disappeared at the end of expiration. There was no systolic murmur in the same region, but one was heard at the aortic cartilage. The tumultuous action and the presence of loud mitral murmurs throughout nearly the whole cardiac cycle made the exact differentiation of the diastolic murmur very difficult, but the absence of a pulmonary systolic murmur made it unlikely that there was regurgitation, since, according to Dr. Balfour, in all cases of pulmonary regurgitation the murmur has been double. Further, the pulse was somewhat collapsing, and there was visible capillary pulse; the conclusion arrived at was that there was aortic regurgitation, and that the murmur was really aortic in origin. The indications of the sphygmograph are not often of practical value, but the tracing in this case had a percussion stroke of great amplitude, and so far supported the diagnosis of aortic disease. It is not easy to say why the murmur was not heard in the usual situation.

A CASE OF PEMPHIGUS FOLIACEUS.

By C. FIRMIN CUTHBERT, M.R.C.S.,
Surgeon to the Children's Hospital, Gloucester.

THIS case (the accompanying photographs of which were kindly taken for me by my friend Mr. Hodges, of this city) was that of a young woman, aged 27, first seen by me on June 1st, 1892.

She complained of aphthæ on and under the tongue, and on the mucous membrane of the mouth and pharynx. There was considerable swelling of the lips and tongue, salivation, and fœtor. Her condition had been getting gradually worse since March 27th. She was very constipated. She stated that for the previous three summers she had large blisters on the soles of her feet, but walked about on them all the time, with the exception of resting a day or two now and then. The blisters had gradually dried up and the skin peeled off. She had always had tender, sweating, and fœtid feet, and moist, clammy hands. She had had amenorrhœa at 20 for nine months, but dysmenorrhœa was usual. The catamenia commenced at 14, and were regular up to 20. She had been always well as a child, and no medicine had been taken likely to produce her illness. The family history was good. She had used the ordinary drying powders for the blistered feet. She was prescribed boro-glyceride to wash out the mouth and throat frequently, and a chlorate of potash mixture.

Her condition gradually got worse up to June 15th, when bullæ made their appearance over the extensor surfaces of each elbow. A few days later the conjunctivæ became swollen and suffused, and small vesicles were to be seen on them. Bullæ also appeared on the ears, and the lips became much more swollen and painful, so that the taking of nourishment was distressing to her. She was ordered to remain in bed about June 11th.

Her treatment now consisted of charcoal dusted into the mouth at frequent intervals, with the occasional local use of iodoform. For the bullæ lead lotion was kept constantly applied. Internally arsenic and general tonic remedies were employed. Six weeks later the mouth began to clean, and the patient herself improved considerably, and no fresh bullæ made their appearance, but after a few days the mouth became as bad as ever, and large bullæ made their appearance upon various parts—on the axillæ, neck, back, chest, abdomen and legs, and around the anus and vulva; they were intensely irritable and painful. The eyelids, face, and scalp were affected next; the bullæ became confluent, and the masses of excoriated surfaces were covered over with pigmented crusts of a dark brown colour, some being almost black. She had no bullæ on her feet during this time. Her condition from the beginning of August became worse and worse, and during the last three weeks of her life she was scarcely able to be moved at all, and the dressings were extremely painful to her. She had constant diarrhœa, which was followed by exhaustion and death on August 26th. At no time was there any albuminuria or blood in the urine. The fluid in the bullæ was invariably turbid from the first.

The cause of this condition could not be attributed to cold or damp, or bad and insufficient food, or any mental worry; neither was there any suspicion of a syphilitic taint; neither did the scabs leave any but superficial excoriation under them and no ulcer, as under the crusts of rupia.

In his very able work on diseases of the skin, Dr. Jamieson says, "Pemphigus in any form is rare," and classes it under the head of general exfoliative dermatitis; and at page 35 of book his account of the foliaceous variety corresponds with this case. Hebra is quoted as finding that pemphigus foliaceus is more common in women. It is peculiarly malignant, extremely rare, and always fatal.

Erasmus Wilson tells us that amenorrhœa predisposes to pemphigus, and that it occurs usually in people with habitually dry skins, and may be preceded by some poisonous injury. In this case there appeared to be no predisposing cause existing except the amenorrhœa, which, however, was years previous to the commencement of the pemphigus.

Although pemphigus is said sometimes to be accompanied by aphthæ and vesicles on various mucous membrane, this

¹ *The Heart and its Diseases*, 2nd edition, p. 180.

case seems to have made its first appearance (excluding the bullæ she previously had on her feet) on these surfaces. Among other plans of treatment, some parts affected were

treated perfectly dry, with starch or flour powdered on, but with no better effect than those treated with soothing lotions. Curiously enough, another case of pemphigus was under my

care at the same time as the one I have previously described. This was in a married lady, about 35 years of age. Two or three bullæ made their appearance on the shin, coming on

with cold, and febrile symptoms. They were irritable and painful. The bullæ dried up, and the skin peeled off after about three weeks, and no others have since appeared. The patient was suffering at the time from neuralgia, and was in a generally weakly condition.

MULTIPLE INSULAR NECROSIS OF SKIN AND SUBJACENT TISSUES.

By HERBERT SMITH RENSHAW, M.D.

A PECULIAR necrosis of small patches of skin subjacent connective tissue and small portions of bone, has been under my care for two years. In many respects the case has been similar to those observed and classified by Dr. Maurice Raynaud.

This disease occurred in a stout, cheerful, healthy, well-nourished child, aged 4 years, living in a new well-ventilated and well-drained house. Previous to this illness she had croup, followed by whooping-cough and measles, from which she recovered perfectly. Her father died of diphtheria, one of his brothers died of diabetes, another of fever. Her father's father is living, and her mother's parents are healthy people.

In May, 1891, she complained of cold extremities, and her feet swelled; there was also a mottled bluish appearance of the surface of both legs, and a dusky coloration extending across the lower part of the abdomen. Shortly afterwards the fingers of both hands became very painful. During this attack the second finger end of the right hand dried up; a portion of bone necrosed and separated, the part healing with bulbous enlargement of the bone stump. At the point of separation a new nail formed, and grew over and round the end of the cicatrix. The skin of the tip of the little finger and of the index finger of the left hand dried up without further injury, except that the nail of the little finger fell off and was renewed. A slough formed in the middle of the right arm, and healed up slowly after throwing off a tough dried up patch of skin.

The patient became gradually worse, the general symptoms being extreme irritability of temper, pain, restlessness, increasing emaciation, and loss of appetite. She often preferred to sit near an open outside door, and when in severe pain pressed her hands firmly together, with her fingers downwards between her knees, and in this position often rocked herself, or caused her attendant to do so, for many hours. When the line of separation formed marking off the dead from the living parts, pain rapidly abated, appetite returned, and with this a great desire for fat food. This was freely indulged. Cod-liver oil and iron were given, and the former plump and cheerful state returned. This attack commenced in May, 1891, and did not terminate before March, 1892.

The winter of 1892 passed without further disaster, but in the spring of 1893 the old symptoms returned; appetite failed; the body emaciated; pain became intense, destroying the child's rest and making her extremely irritable, excitable, and passionate. During this relapse she was not sensitive to cold, and often preferred to sit with the outside door open, and exposed to a current of cold air. Her feet were often damp and cold, but were not affected by the same intense changes that destroyed patches of the upper extremities and parts of the fingers. On March 11th, 1893, the first indications of relapse were ushered in with great restlessness.

The child became almost sleepless, and her rest was broken by startling dreams. Nausea became a prominent symptom attended by epigastric pain, and vomiting, with partial perspirations on the head and neck. The pulse varied between 100 and 140. The temperature, which rose to 100° on March 11th, subsided to normal on March 19th, rose again to 100° on March 22nd, was down again on March 28th, and never rose again to 100°. Though occasionally subnormal during the relapse, after this date it was sometimes 99.2°. In the period of increased temperature the skin was flushed. At the same time the right side of the neck became stiff, and some pain was also noticed in the right knee. The voice became