

## SOME FEATURES OF AORTIC REGURGITATION IN YOUNG SUBJECTS.

BY

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AORTIC valve disease, causing regurgitation, in young subjects, presents some features of especial interest and importance, runs a course which in many respects is different from the same disease in adults, and gives a clinical picture which has certain distinctive features. Certain of the vascular phenomena which are consequent upon free regurgitation through the valve are seen in most marked and characteristic form in the young, partly, no doubt, from the more yielding nature of their vessels. Compensatory changes in the heart and circulation are rarely satisfactorily established, and the disease is an even more serious one, and runs a shorter course than in adults.

In all the cases mentioned in this paper the patients were so ill as to require admission to hospital, and I have limited myself to in-patient cases, because of the greater opportunity for systematic observation. It follows also from this selection that I deal only with the more serious cases of the disease. Many of them were under my care at intervals for several years, so that the progress of the affection could be followed. In all of them, though in the large majority other valvular lesions co-existed, aortic regurgitation and its results were the predominant feature in the case.

The general incidence of aortic valve disease in hospital in-patient practice is not without interest. Of 3,300 cases admitted to my beds during the time the cases under consideration occurred, there were 96, or nearly 3 per cent., of cases of aortic valve disease; of these 33, or 1 per cent., were under 28 years of age. The age of 28 may seem a somewhat arbitrary upper limit, but it was taken as such in order to include 4 cases in whom the disease began in childhood, and who had been under my observation for many years. Cases in which there was any possibility of congenital heart disease were, of course, excluded. The tables which follow show the sex and age incidence. My colleague, Dr. Carey Coombs, has also kindly given me a

### AORTIC VALVE DISEASE (REGURGITATION). I.—Above 30 Years of Age.

Years of Age.	Total.	Males.	Females.	+ Mitral Disease.	
				Males.	Females.
30 to 35 ... ..	14	13	1	—	—
35 to 40 ... ..	12	12	0	—	—
40 to 45 ... ..	8	4	4	—	3
45 to 50 ... ..	16	13	3	—	3
50 to 60 ... ..	7	4	3	—	1
60 to 70 ... ..	5	4	1	1	—
70 ... ..	1	1	0	—	—
Total ... ..	63	51	12	1	7
		81 per cent.	19 per cent.	12.6 per cent.	

### II.—Under 28 Years of Age.

Years of Age.	Total.	Males.	Females.	+ Mitral Disease.	
				Males.	Females.
12 to 15 ... ..	9	4	5	3	2
15 to 20 ... ..	12	8	4	3	2
20 to 25 ... ..	7	3	4	2	1
25 to 28 ... ..	5	1	4	1	2
Total ... ..	33	16	17	9	7
		48.4 per cent.	51.6 per cent.	48.4 per cent.	

### III.—Out-patients Under 28 Years of Age (Dr. Carey Coombs).

Years of Age.	No. of Patients.
6 to 10 ... ..	4
10 to 15 ... ..	10
15 to 20 ... ..	16
20 to 25 ... ..	3
25 to 28 ... ..	9
Total ... ..	43

Males, 24; females, 18 = Males, 57 per cent.

list of out-patients under 28 suffering from aortic disease. In both in-patients and out-patients the main incidence of the disease lies between 10 and 20 years. The youngest of my own cases was 12 years old, but in Dr. Coombs's series there are four patients between 6 and 10 years. With regard to the adult cases, it may be added that the Bristol Hospital supplies an industrial district containing large docks, and in which the males are mostly engaged in laborious occupations involving heavy strain.

Some points which appear from these tables are that in the in-patient cases under 28 years males and females are about equally affected; in the out-patient cases the proportion of males is a little higher, 57 per cent.; whilst in those above 30 years there are more than four times as many males as females. This difference is connected with the etiology. In the 33 in-patients under 28 acute rheumatism was the cause in 21, chronic rheumatism in 4, and chorea solely in 4, a total of 29, or 87 per cent., in which there was a history or present evidence of some rheumatic manifestation, and in Dr. Coombs's out-patient cases such manifestation was present in 83.3 per cent.; whilst the other causes of aortic disease in adults are, of course, absent. In one patient, however, aged 22, there was a doubtful history of the valvular mischief being caused by a severe strain.

One case, a girl of 14, was an example of the rare form of aortic incompetence produced directly by mechanical dilatation of this orifice. She suffered from a small white kidney, and presented in marked degree—unusually so for a child—the cardio-vascular changes of renal disease. The systolic blood pressure was 190–200 mm. Hg, and during the first part of her stay in hospital there was heard only a very accentuated loud, ringing aortic second sound. After a time, during which she was steadily getting worse, a faint aortic diastolic murmur appeared following the second sound, and this gradually became longer and more distinct; with this, the left ventricle dilated, the lungs became engorged, and she died three or four weeks later of heart failure and uraemia. At the *post-mortem* examination the aorta and aortic orifice were dilated, and the valve did not hold water, but the segments, except for being a little opaque, were quite healthy in appearance, and were not shrunken.

This is the clearest case I have seen in which aortic regurgitation presumably occurred as the direct result of excessive blood pressure, for we were able to watch the process throughout.

Two of the 33 cases were known to develop infective endocarditis. It is interesting to note that of the 4 cases which owned chorea only as the cause, in 2 patients there had been eight, and in 1 seven attacks.

Another point is that, although it is by no means infrequent to find pure or uncomplicated aortic regurgitation, with a diastolic murmur only, in adults, this seems to be decidedly uncommon in younger patients. In 27 out of the 33 patients other murmurs were present, although as regards signs and symptoms the aortic lesion predominated the case. In 9 of the 27 the second murmur was an aortic systolic one, which of course does not by any means imply actual aortic stenosis, nor necessarily any serious complication; in the other 18 there were mitral murmurs. This difference from the adult is probably again connected with the etiological factor of rheumatism.

Professor Clifford Allbutt<sup>1</sup> alludes to extension of pericarditis to the aortic area as a cause of aortic valve disease. In five of my patients acute pericarditis was observed at some time, either preceding the appearance of the diastolic murmur, or both murmur and pericarditis were present on admission. Probably more patients were thus affected, but these were the only attacks actually observed. In those cases in which aortic disease was complicated with mitral regurgitant murmurs, in

at least four cases the aortic disease was certainly primary, and later on, as the heart enlarged, mitral regurgitation ensued. In another a murmur, which was at first mid-diastolic and regarded as an aortic murmur conducted to the apex, became three years later a definite and characteristic presystolic crescendo murmur.

In the majority of these cases of combined lesion both valves were affected, as evidenced by murmurs, at the time they first came under observation. It is generally held that the usual course is for the rheumatic process to spread from the large mitral flap to the adjacent aortic valve segment; but in cases of acute rheumatism, when the heart is first involved, it is certainly not uncommon to hear an aortic systolic murmur accompanying the more obvious signs of mitral valve disease; and I believe that in the cases under consideration the affection of the mitral valve is very often secondary, and the mitral regurgitation due to weakening of the ventricular wall from recurrent rheumatic affection of the heart muscle (carditis), and consequent dilatation of the mitral orifice.

With regard to symptoms, as in cases of aortic disease generally, pain in the precordial region is the most frequent and prominent symptom in young subjects. Many of the patients suffered from attacks of severe angina pectoris. When angina occurs in these young patients it is often remarkable for its frequency; three or four attacks, or even more, may occur in each twenty-four hours, and that at intervals over a long period of time. Although the pain is severe the patient does not, however, look so very ill as an older patient in an attack of angina pectoris, nor is there the same fear of impending death. None of my patients died in an anginal attack. Next in frequency to pain comes palpitation, or rather a violent throbbing of the heart and great vessels. Then comes dyspnoea, either persistent on exertion or in the form of attacks in which the patient gasps for breath and has a most distressing sense of being unable to breathe. Attacks of syncope may occur, and in one case this was the cause of death.

Certain forms of haemorrhage are not infrequent. Epistaxis is by far the most common: in two of my patients it was profuse and often repeated; in two others haemoptysis occurred several times, it was slight in amount but continued two or three days; in one of these there was a mid-diastolic murmur and thrill, so that mitral stenosis may have been present as well as aortic disease; in the other there was pure aortic regurgitation. It is well to bear in mind this possibility of haemoptysis in the aortic disease of the young. In one case associated with mitral regurgitation there was a smart attack of haematemesis, and this also occurred in one of Dr. Coombs's patients; a purpuric rash was observed in another patient, with no evidence of malignant endocarditis. One patient with aortic disease (purely) of eight years' standing, of no great severity and without much enlargement of the heart, had a typical attack of embolism at the age of 20, resulting in right hemiplegia and aphasia. In another patient, aged 18, persistent insomnia and mental symptoms in the form of delusions were the prominent features. Young patients do not, however, suffer from insomnia as older ones do. Oedema, as in cases of heart disease generally in children as compared with adults, is conspicuously absent until the terminal stages of heart failure. A symptom that appears with somewhat unexpected frequency is albuminuria. In 13 out of the 33 cases albuminuria was present on admission to hospital. It did not occur with greater frequency in those patients who had a mitral as well as an aortic lesion. As a rule, the amount of albumen was small, and in 11 patients out of the 13 it disappeared after a few days' rest in bed. Renal casts were either absent altogether, or there were a very few hyaline ones. In the other two patients there was a considerable quantity of albumen, which persisted and was due to coincident renal disease.

With regard to the changes in the heart itself, great enlargement of it is an even more marked feature of aortic disease in children than it is in adults. The bulging of the precordial region, the violent pulsation and shaking of the small chest, are frequent and striking clinical signs. With this goes in a considerable number—36 per cent. of my cases—pronounced dilatation of the aorta, causing extension of dullness to each side of the sternum and upwards, even as high as the sternal notch. Over the

same area, or generally extending considerably beyond it on each side and above, is often to be felt a thrill, and the aorta itself and the thrill may be felt above the sternal notch. These phenomena are rarely seen in older to the same extent as in young patients, and may be attributed partly to the more yielding nature of the aortic wall in the latter and to its stretching more readily under the constantly repeated excessive strain, and partly to the weakening of the wall by recurring attacks of rheumatic infection. The large vessels share to a less extent in the dilatation. In one case, in which these signs of dilatation of the aorta were clinically very conspicuous, although *post mortem* the aortic valve was quite incompetent to tests and the orifice much dilated, the aorta itself was very little, if at all, above the normal size, so that the great dilatation observed clinically was a dynamic and not a permanent feature, and the walls of the large arteries also appeared healthy.

Certain abnormal sounds or murmurs are heard on auscultation of the arteries in cases of aortic regurgitation. As they only occur when the regurgitation is free, they are, apart from their clinical interest, of some importance as indicating severity of lesion. These signs are especially frequent and well marked in young subjects. I allude only to sounds heard when the bell of the stethoscope, or preferably its small end after removal of the wooden or ivory chest piece, is placed as lightly as possible over the vessel, and not to the murmur which is produced when an artery is compressed or narrowed at one point by pressure. The abnormal sound most often heard is an abrupt, sharp, single knock, snap, or "thump"—the latter word sometimes exactly represents the sound—occasionally there is a double "thump, thump." Although this sound can hardly be connected with cardiac murmurs, I find that when the double one occurred there was an aortic systolic as well as a diastolic murmur. These sounds were audible in as many as 15 out of the 33 cases, and in the majority in the most distal arteries, the radial and the dorsalis pedis. In a few instances, the single thump was followed by a diastolic soft murmur, but the latter could only occasionally be traced into the distal arteries; as a rule it was not audible beyond the brachial and femoral arteries—that is to say, the "thump" can be followed far distal to the murmur. In two cases double murmurs, systolic and diastolic, were distinct in the arteries, and in one of them the first element was of an unusual character, being loud and squeaking. Capillary pulsation was not especially marked nor invariably present in the cases with these abnormal arterial sounds. So far as my experience goes, this peculiar "thump" is not met with outside aortic regurgitation, certainly not in arteries so distant as the dorsalis pedis. It is difficult to time, but it seemed generally to follow the arterial wave immediately; if so, it may be due to the sudden relaxation of the over-stretched arterial wall—the murmur which occasionally followed the "thump" was certainly diastolic.

With regard to the heart murmurs, the only points to which I wish to allude in the aortic diastolic murmur of the young subject are that it is often very long, running almost up to the first sound, and to its loudness or harshness, and its variability. The murmur is more frequently heard at the apex than in adults—Flint's murmur; this was the case in 51.5 per cent. of the patients, and in 34 per cent. it was accompanied by a well-marked diastolic thrill. Not infrequently the murmur at the apex is mid-diastolic, and then the diagnosis between a mid-diastolic murmur of aortic origin and one due to mitral stenosis may be extremely difficult. Especially is this the case where the heart affection is rheumatic, and enlargement of the right heart or a systolic apex murmur shows that the mitral valve is involved. I believe that the dictum is correct that in cases where Flint's murmur is present in which there is a history of rheumatism it is impossible to exclude with certainty mitral stenosis. In one case, during the course of about six years' illness, the transformation of a mid-diastolic apex murmur into the typical crescendo murmur of mitral stenosis was watched. On the other hand, a short diastolic murmur limited to the first part of the long silence is not, I believe, of mitral origin.

There is one point with regard to the conduction of an aortic murmur in the child. It comes out, from the analysis of these cases, that in every instance in which the aortic incompetence was concomitant with enlargement

(dilatation) of the right heart (from mitral disease) the aortic diastolic murmur was loudly and plainly conducted down and outside the right edge of the sternum; the conduction might be to the apex as well, but that to the right of the sternum was very marked.

Mention has been made above of the appearance of mitral regurgitant murmurs in the course of heart disease which began as aortic, and in which the mitral affection may fairly be inferred to be secondary to it. No relief appeared to be thereby afforded to the cardiac disorder.

## SUMMARY.

	Males.	Females.	Total.
Aortic regurgitation purely ...	2	4	6
Aortic regurgitation, with aortic systolic murmur ...	4	5	9
Aortic regurgitation, with mitral regurgitation ...	5	4	9
Aortic regurgitation, with mitral stenosis only ...	?1	0	?1
Aortic regurgitation, with aortic systolic murmur, and also mitral disease ...	5	3	8
Cases.			
Great enlargement of heart present in ...			21
Dilatation of aorta ...			12
Flint's murmur ...			17
Flint's murmur, with thrill at apex ...			11
A "knock" audible in peripheral arteries ...			10
A "knock," together with a murmur ...			5
Albuminuria present in ...			13
Albuminuria disappearing rapidly ...			11
History of previous acute rheumatism ...			21
History of rheumatic pains only ...			4
History of chorea only ...			4
History of scarlet fever only ...			1

A few remarks as to treatment may be added. Of course, in cases severe enough to be admitted to hospital rest often over a long time is necessary. Restriction of the amount of fluid taken, so far as can be done without discomfort to the patient, is valuable, by relieving the work of the heart by after a time diminishing the amount of fluid in circulation. In the absence of rheumatic manifestations or of fever, I have not seen much benefit from the continued use of the salicylate group of drugs; short courses may be useful in relieving precordial pains. The iodides, in combination with salicylates or alone, have not seemed to me to influence the course of the disease, and the iodides given for some time have not prevented the occurrence of attacks of angina or acute dyspnoea in those subject to them. Digitalis was given whenever there was signs of heart failure according to the indications in each individual case as they occurred from time to time; it is more often necessary than in the aortic disease of adults, partly because of the far greater frequency of implication of the mitral valve. As to relief of special symptoms, the most urgent are the very distressing and sometimes alarming attacks of angina pectoris, which are not infrequent. In these young patients amyl nitrite is the most effectual drug. In several cases, nitro-glycerine, and the other nitrites, completely failed both to relieve and prevent the attacks, when amyl nitrite gave prompt relief, but of course did not prevent their recurrence. Possibly it owes its good effects to its rapid action, as the attacks, though severe and sometimes very frequent, are as a rule of short duration. The dose inhaled has sometimes to be increased up to  $\text{m} \times$  to  $\text{m} \times \text{i} \text{j}$  at a time to maintain the effect, but seems to entail no unfavourable consequences. One girl, who at times during the last two years of her life suffered from six to ten sharp paroxysms daily, used to inhale as much as  $\text{z} \text{j} \text{ss}$  to  $\text{z} \text{i} \text{j}$  amyl nitrite in the twenty-four hours, and no other drug did her any good. The next most urgent symptom is the attack of dyspnoea or suffocation, which is probably a form of angina. Here again amyl nitrite often relieves, but sometimes caffeine and ammonia is more effectual.

Not so immediately urgent, but distressing from its persistency, is the excessive or violent pulsation of the greatly enlarged heart. This palpitation is often the subject of the patient's chief complaint. I have found a mixture of liq. arsenicalis, ac. hydrocyan. dil., and tinct. veratri viridis, given for some time, most useful in the treatment of this symptom, which is often a most troublesome one, even when the patient is at rest in bed, and when there is no evidence that the heart is not able, in spite of the abnormal conditions, to maintain an efficient circulation.

In other cases, of course, this symptom is one of the expressions of the failure of the heart to do this, and must be treated accordingly.

## REFERENCE.

<sup>1</sup> *System of Medicine*, second edition, 1910, vol. vi, p. 421.

## BLOOD PRESSURE IN MENTAL DISORDERS.

BY

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RECENT investigation tends to disprove that there is any constant correlation, as hitherto more or less generally accepted by alienists, between certain types of mental disorder—namely, states of emotional exaltation and depression—and definite modifications of the arterial pressure. It is chiefly to Dr. Maurice Craig<sup>1</sup> in this country that the view is to be attributed that states of emotional exaltation are usually associated with low pressures and states of emotional depression with high pressures. Of the work recently published which fails to confirm this relationship that of Dr. John Turner<sup>2</sup> is the most important. He concludes from his observations that the blood pressure bears no definite relation to the mental state, but only has a constant relation to the age period.

The observations on which this paper is based were made on cases forming a considerable proportion of the female admissions to the Suffolk District Asylum during a period of two years. The instruments used were Martin's modified Riva-Rocci, and Leonard Hill's latest pattern wrist instrument. In the majority of cases both instruments were used in making the earlier observations, and from time to time the larger one was used to confirm the readings obtained by the wrist instrument. With few exceptions both forms of apparatus were found to give similar results, but Hill's sphygmometer, besides being more portable, has advantages in dealing with the insane; it is less imposing, and in certain types of depressed patients nervous influences appear to cause unduly high readings. In these cases, by spending a quarter of an hour or more taking readings at frequent intervals, I have frequently found that the pressure gradually falls until it reaches a constant point; whereas in cases of mania, more especially those with marked excitement, the same influences do not appear to be at work. The higher readings of earlier observations have been noted by other observers. In the cases here summarized the observations were made about noon with the patient sitting, and resting the forearm on a table so that the artery being examined was approximately at heart level. In the large majority of cases an average was struck from the weekly readings, 3 to 5 in number, immediately following the admission of the patient, and subsequently in many of the cases observations were made at monthly and then longer intervals in patients remaining under treatment. There were some cases in which the number of observations was necessarily limited to one or two, and a few in which single observations were made have been included where I was satisfied with their accuracy. I only attempted to read the manometer scale to the nearest 5 mm., which is accurate enough for ordinary clinical purposes. The pulse-rate was observed at the time, and cases with marked irregularity of rhythm excluded; a note was also made as to the condition of the vessel wall as judged by the fingers. In all cases coming under the headings of mania and melancholia, the observations from which the averages were taken were made while the patients exhibited respectively definite exaltation or depression of the emotional tone.

## MANIACAL STATES.

The observed cases numbered 33. They had an average age of 33 years, and showed an average systolic pressure of 115 mm. The maximum pressures were obtained in three patients aged 56, 56, and 52 years, with readings of 150, 140, and 135 mm. respectively. The minimum pressure was 100 mm., observed in 5 cases aged 19, 21, 30, 30, and 46 years. The radial arteries in these cases were not palpable or barely so. There were included among these 33 cases 10, with an average age of 29 years, of a